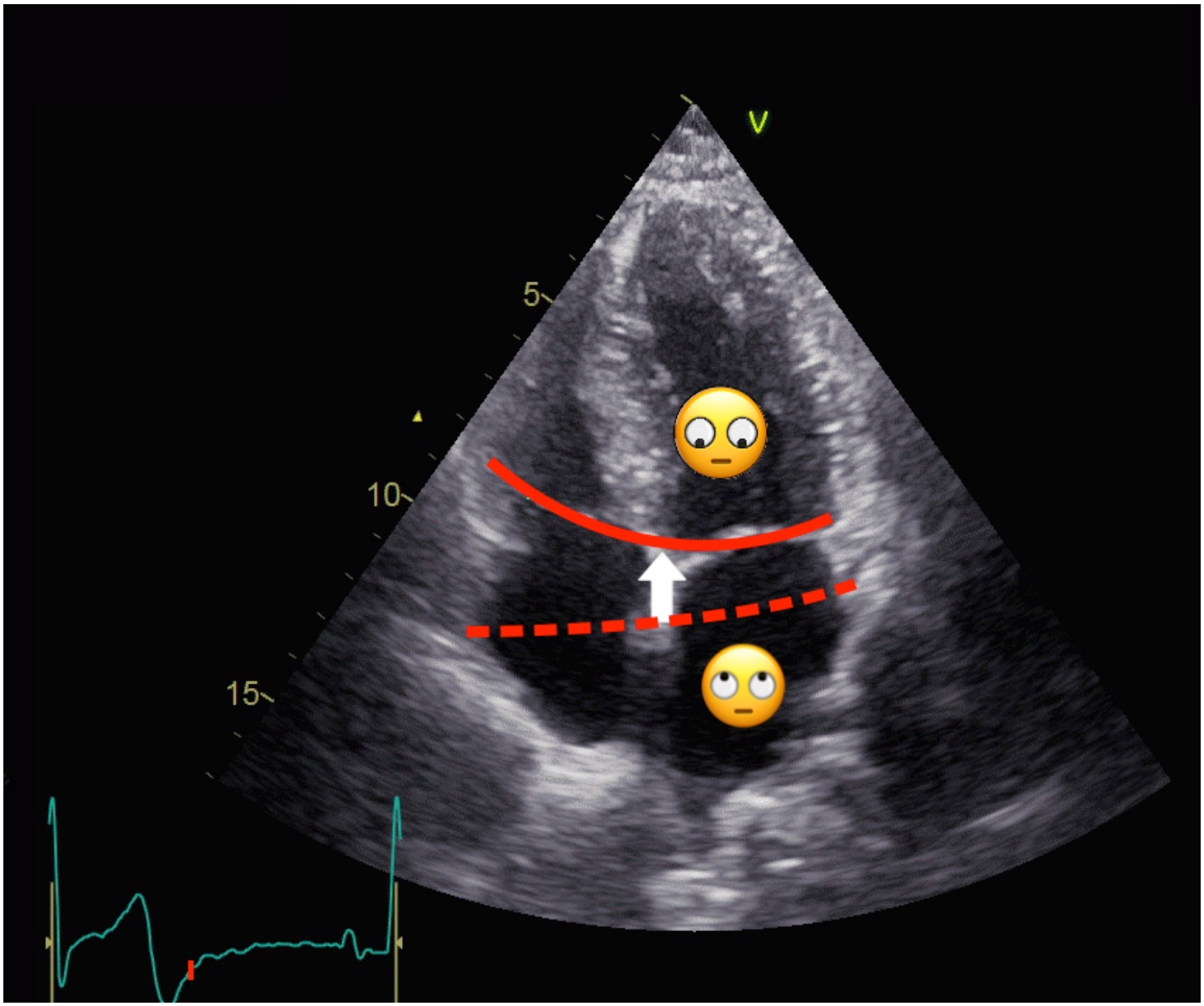
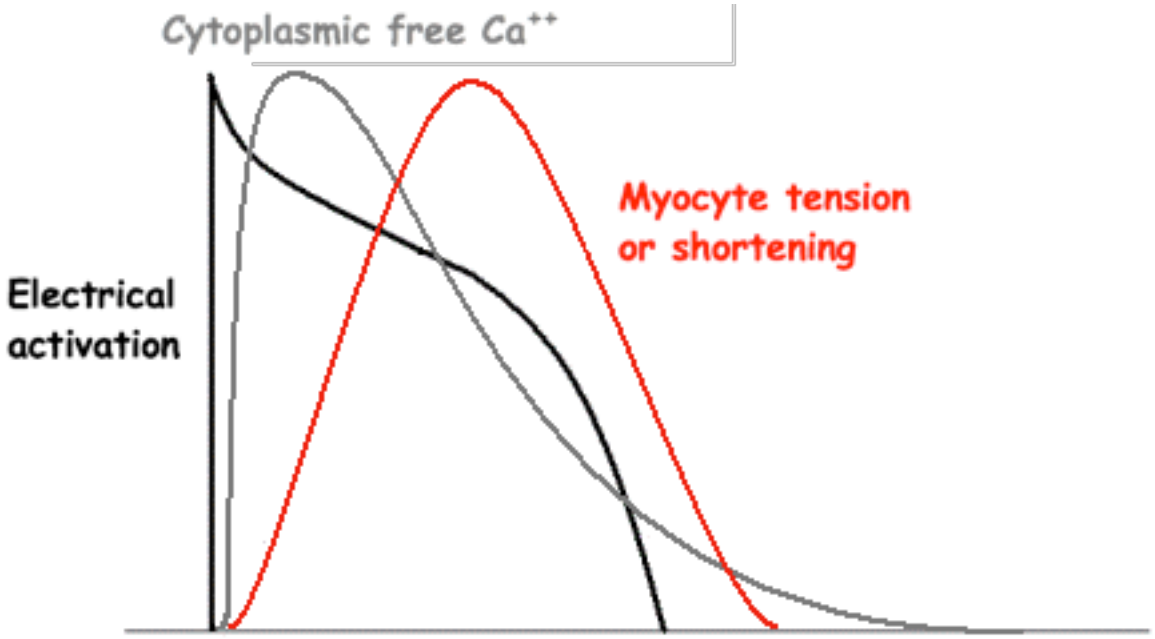
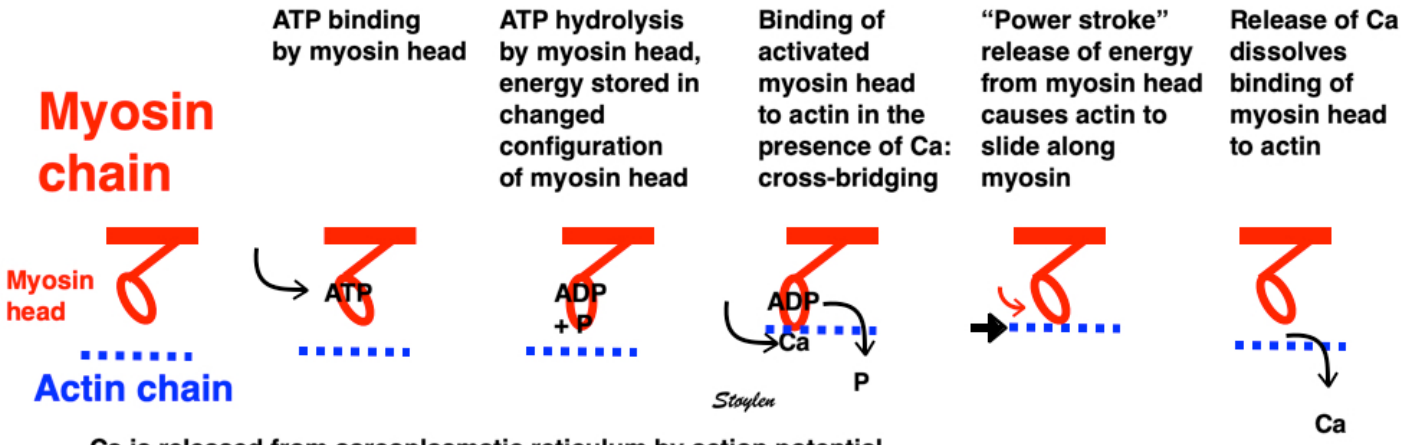
Compendium on left ventricular mechanics



Asbjørn Støylen
Professor Emeritus, Dr. Med.
https://stoylen.folk.ntnu.no/strainrate/

Contraction



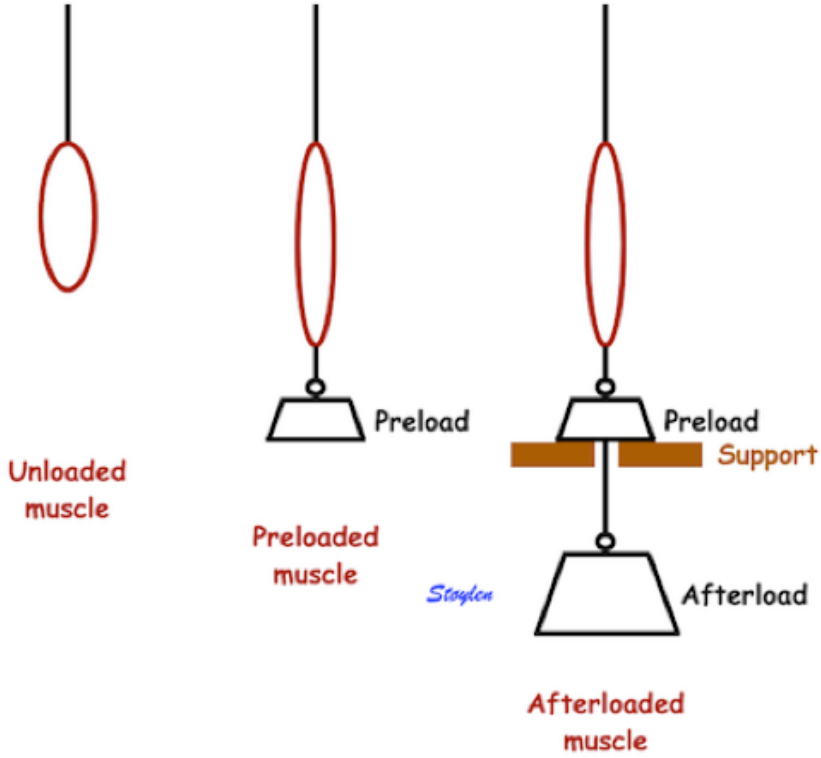


Ca is released from sarcoplasmatic reticulum by action potential
- causing binding of activated myosin heads to actin; cross bridging, enabling fliament sliding
- in the continuing presence of Ca, the cycle will repeat, and contraction continues

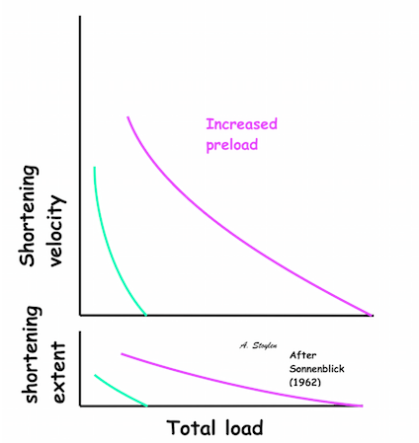
If Ca is removed from cytoplasm into sarcoplasmatic reticulum, new cross bridges will not form, contraction declines.

Ca removal by SERCA requires energy (ATP) for pumping against concentration grtadient

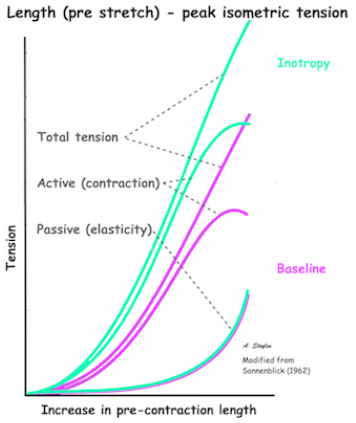
Load



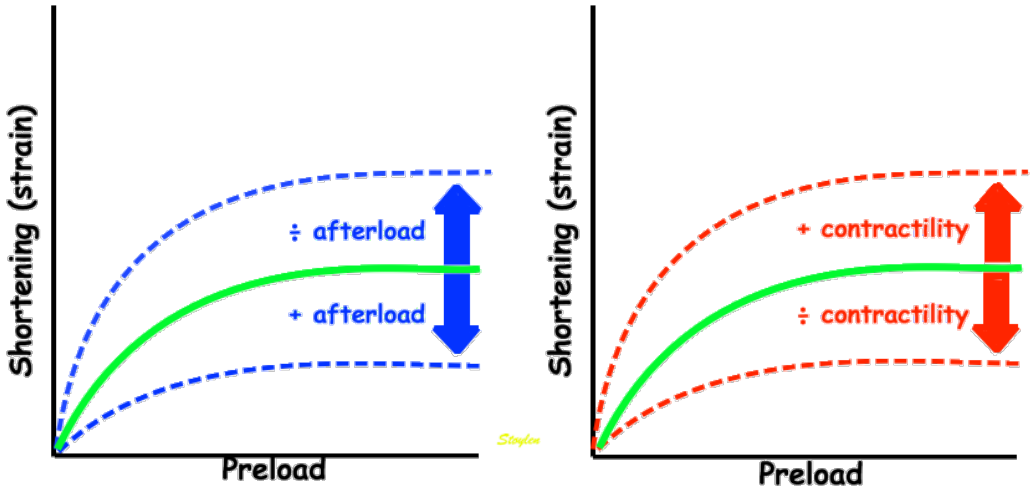
The difference between pre- and afterload. After preload is added, a support is placed, preventing further stretch of the muscle when another weight is added. This second weight is the afterload. When the muscle contracts, it has to develop a tension that is equal to the total load, before it can shorten. If the peak force is higher than the total load, the muscle will then shorten without generating more tension, in an isotoniccontraction.



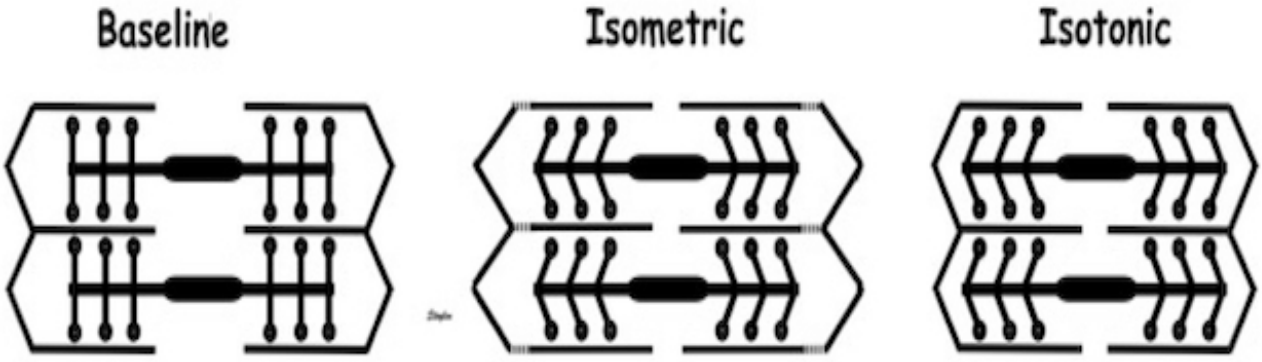
Shortening velocity and total shortening, Relation to preload and total load. Both shortening and velocity can be seen to decrease with increasing afterload (total load), but increase with preload.



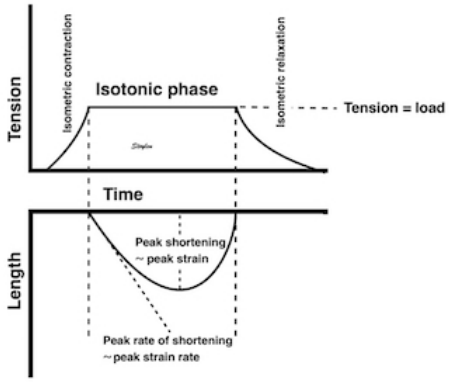
Stretching the muscle before stimulation, increases tension. The increase in passive tension will be present at rest, before twitch. During twitch, there is an increase in total tension with increasing pre twitch length. The increase in contractile tension is then the difference between the passive and the total curve. At a certain length, active tension starts to decline, even if passive and total tension still increases.



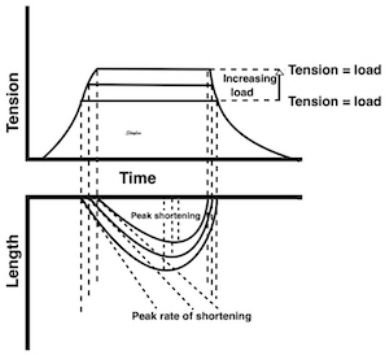
Myocardial shortening vs pre-, afterload and contractility. shortening increases with preload, as shown in both panels, although to a certain extent, until the preload insensitive zone (where active tension starts to decline, but passive tension still increases). Shortening is the resultant of force vs. afterload, the higher the afterload, the less the shortening, for a given contractility. Contractility is the load independent part of force development. The higher the contractility, the more the shortening for a given afterload.



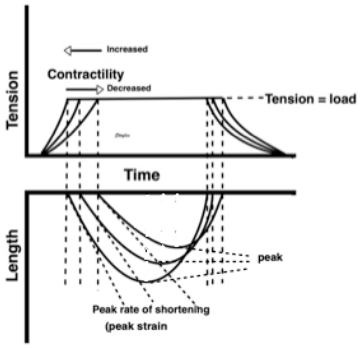
Shortening is the result of tension versus load. If the load is higher than the maximal tension the muscle can develop, the muscle will not shorten at all. Actin is still moved along the myosin , but the energy is stored as deformation within the sarcomere without shortening aof the sarcomere. This means that the force generated by contraction is stored as elastic tension in the muscle, and the contraction is isometric The middle figure, retaining the length of the baseline - left). If the load is less than the maximal tension, the muscle will start to shorten when tension equals load, and from there the contraction is isotonic - shortening at constant load. The right sarcomere is shorter than the baseline.



Length tension diagram of a muscle twitch in an isolated muscle preparation. The muscle takes some time to develop the tension that equals the load, and during that period the contraction is isometric, with no shortening. Shortening starts when tension equals load. When the muscle relaxes, relaxation induces shortening until tension again equals load, after that relaxation is isometric.

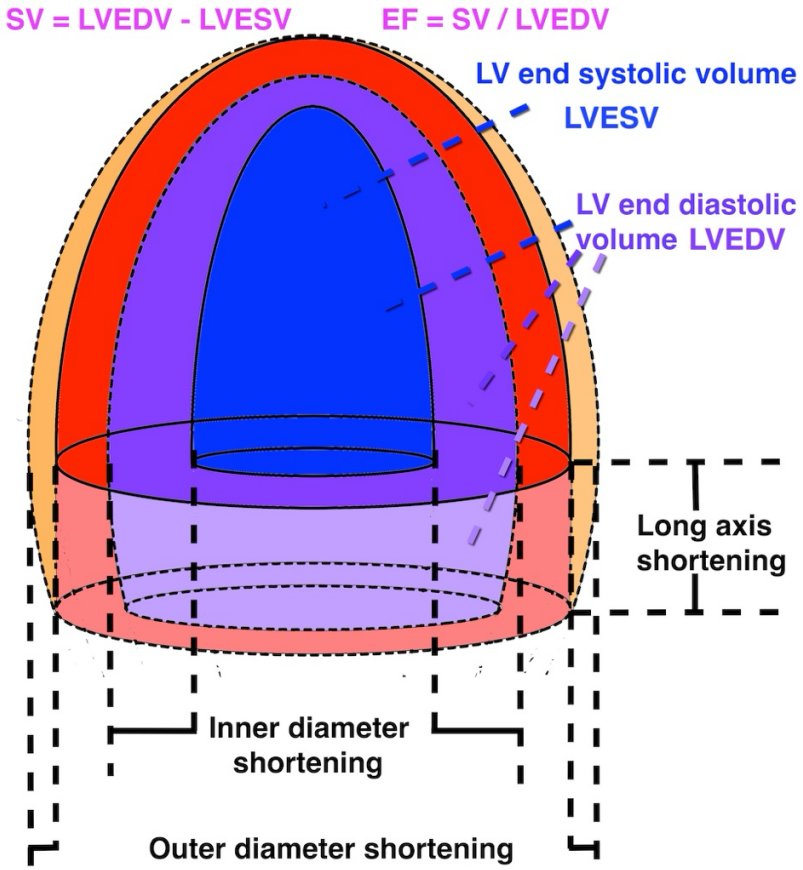


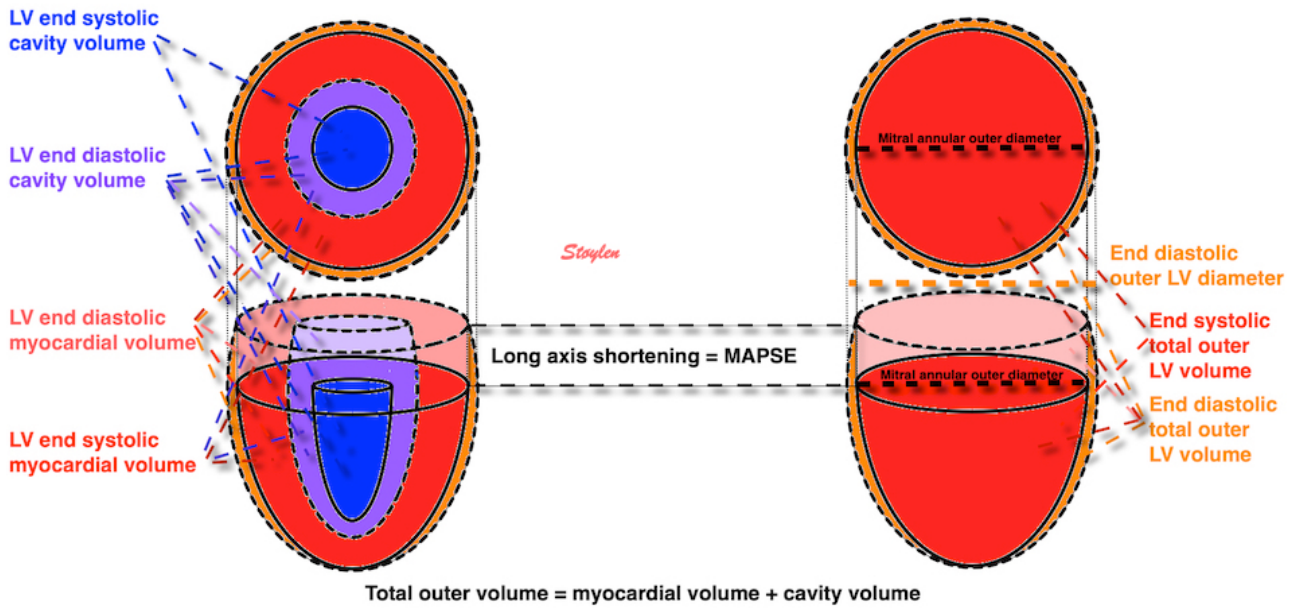
Series of twitches with different loads. All twitches follow the same tension curve, i.e. shows the same contractility, but as load increases, shortening starts at later time points, and the shortening time as well as the extent and rate of shortening decrease.



Series of twitches with the same load, but with different contractility (ability to develop tension). With decreasing contractility, it takes longer to develop tension = load, the period of shortening as well as the extent and rate of shortening decrease.

Volumes





Outer volume decrease has two components:

Longitudinal component = MAPSE × Mitral annular outer area

= MAPSE × outer mitral annular diameter / 2

Transverse component which is SV - longitudinal component.

Given (roughly) incompressible myocardium, myocardial volume is the same in end diastole and end systole, and the total (outer) volume reduction must be equal to the stroke volume:

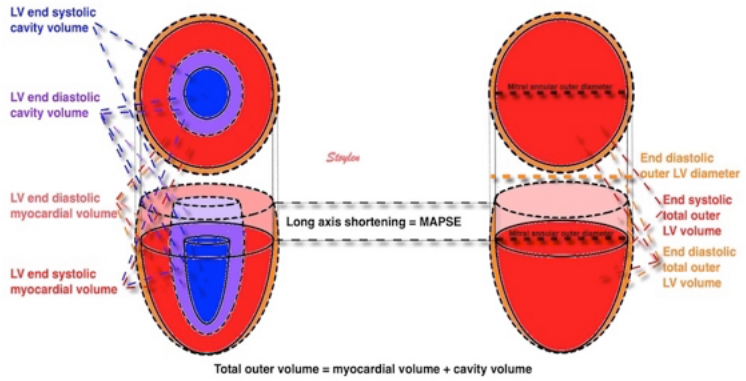
SV = LVEDV - LVESV
= (total EDV - myocardial EDV)
- (total ESV - myocardial ESV)

Given myocardial incompressibility,
Myocardial EDV = Myocardial ESV:
SV = Total EDV - total ESV

Long axis outer shortening = 60 - 70% of SV

Cross sectional shortening = 30 - 40% of SV

Systolic LV long axis shortening



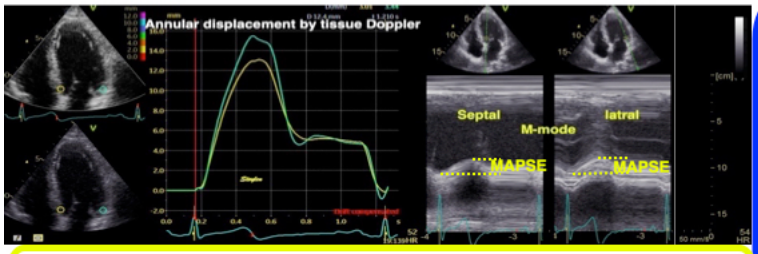
Given (roughly) incompressible myocardium, myocardial volume is the same in end diastole and end systole, and the total (outer) volume reduction must be equal to the stroke volume:

SV = LVEDV - LVESV
= (total EDV - myocardial EDV)
- (total ESV - myocardial ESV)

Given myocardial incompressibility,
Myocardial EDV = Myocardial ESV:
SV = Total EDV - total ESV

Long axis outer shortening = 60 - 70% of SV [3, 4]
Cross sectional shortening = 30 - 40% of SV

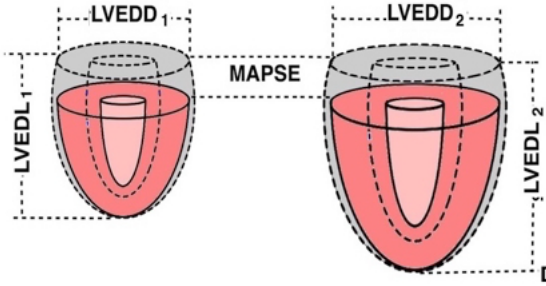
Systolic LV long axis shortening = Mitral Annular systolic displacement[1] = Mitral Annular Plane Systolic Excursion (MAPSE)



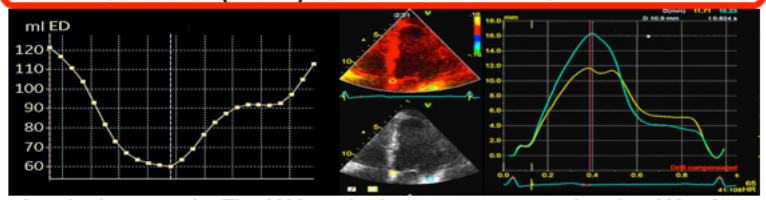
MAPSE can be measured by annular TDI or by M-mode.[2] The average of lateral and septal MAPSE is representative for global MAPSE.[4]

The apex is stationary, so MAPSE
= total absolute LV systolic shortening.
- MAPSE correlates weakly positively with BSA (R=0.12) but not with sex, and negatively with age (R=-0.50).
- MAPSE correlates weakly with SV (R=0.25) and EF (R= 0.16)[4] in a healthy population
- MAPSE contribution to SV in % is constant across both body size and age,[4] and as SV:
- decreases with age,
MAPSE changes in ageing are proportional to SV changes.[5]

Both EDV and SV correlate positively with BSA, negatively with age. The concomitant drop in EDV and SV is the reason for the preserved EF, being SV/EDV, which thus is independent of age. MAPSE is proportional to SV, and the ratio of MAPSE to SV is constant across age, and there is no compensatory increased short axis function, FS is unchanged with age. Both EDV and SV increases with BSA, so EF is independent of BSA. MAPSE correlates only weakly with BSA,

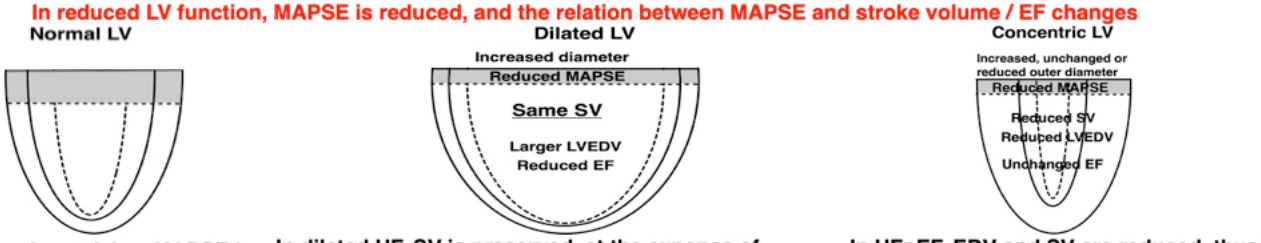


A larger ventricle due to larger BSA, is proportionally both longer and wider, so the same MAPSE generates a proportionally larger SV, explaining the weak correlation (R=0.12) between BSA and MAPSE.[4]



During the heart cycle, The LV length changes are proportional to LV volume changes, and the annulus motion is similar to an inverted volume curve

In reduced LV function, MAPSE is reduced, and the relation between MAPSE and stroke volume / EF changes



In normal ventricles, MAPSE is proportional to SV and EF.

In dilated HF, SV is preserved, at the expense of increased EDV. Thus, EF decreases. As LV widens, the same SV is generated by a smaller MAPSE, which thus is reduced in line with EF.[1, 2]

In HFpEF, EDV and SV are reduced, thus EF is unchanged (which is the shortcoming of EF). With reduced SV, MAPSE is reduced in line with SV.[6]

Thus MAPSE is reduced both in HFrEF and HFpEF, and is a better measure of reduced function and prognosticator than EF.
As MAPSE is near BSA independent, it is thus also a better prognosticator than GLS as documented [7]

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1 Simonson JS, Schiller NB. Descent of the base of the left ventricle: an echocardiographic index of left ventricular function. J Am Soc Echocardiogr 1989;2:25-35
2 Höglund C, Alam M, Thorstrand C. Effects of acute myocardial infarction on the displacement of the atrioventricular plane: an echocardiographic study. J Intern Med 1989 Oct;226(4):251-6
3Carlsson M, Ugander M, Mosén H, Buhre T, Arheden H.Atrioventricular plane displacement is the major contributor to left ventricular pumping in healthy adults, athletes, and patients with dilated cardiomyopathy. Am J Physiol Heart Circ Physiol. 2007 Mar;292(3):H1452-9.
4 Støylen A, Mølmen HE, Dalen H. Relation between Mitral Annular Plane Systolic Excursion and Global longitudinal strain in normal subjects: The HUNT study. Echocardiography. 2018;35:603–610
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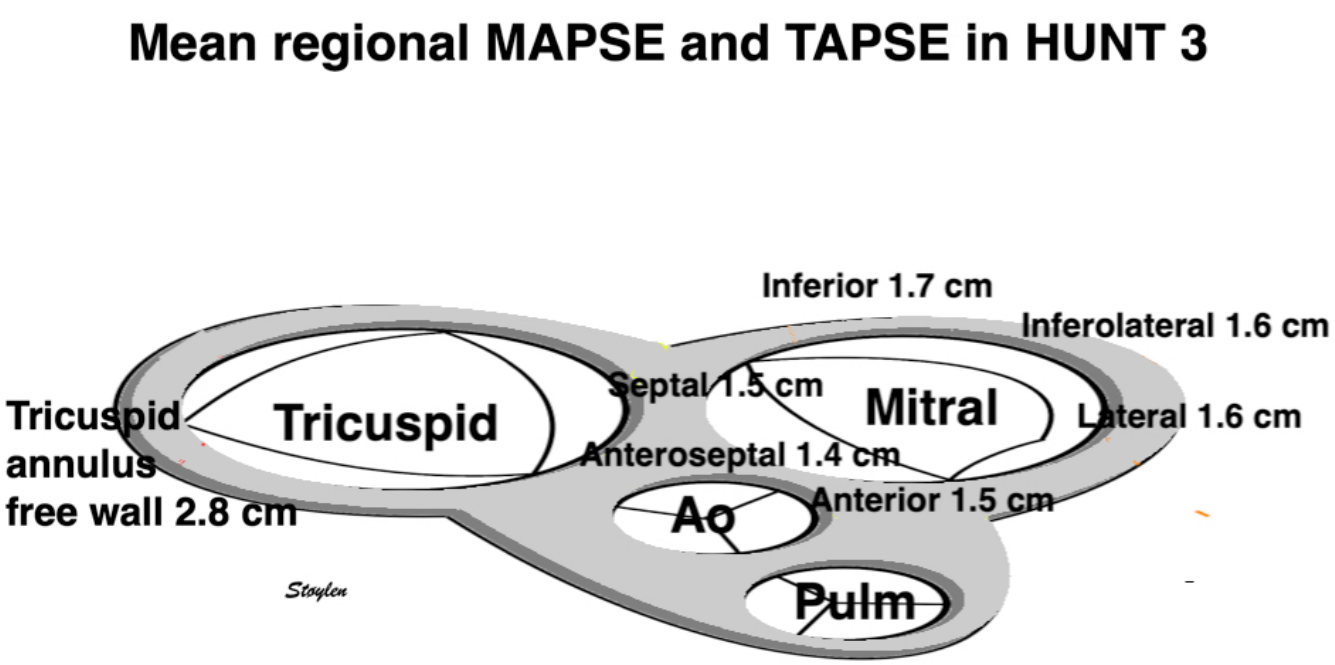
6Vinereanu D, Nicolaides E, Tweddel AC, Fraser AG. "Pure" diastolic dysfunction is associated with long-axis systolic dysfunction. Implications for the diagnosis and classification of heart failure. Eur J Heart Fail. 2005 Aug;7(5):820-8.
7Automated In-Line Artificial Intelligence Measured Global Longitudinal Shortening and Mitral Annular Plane Systolic Excursion: Reproducibility and Prognostic Significance.
Xue H, Artico J, Davies RH, Adam R et al.J Am Heart Assoc. 2022 Feb 15;11(4)

AV-plane bending:



Mean AV-plane systolic displacement at different sites, showing the variability in the Mitral annulus, and TAPSE in the tricuspid annulius which is higher than any site in the mitral annulus.

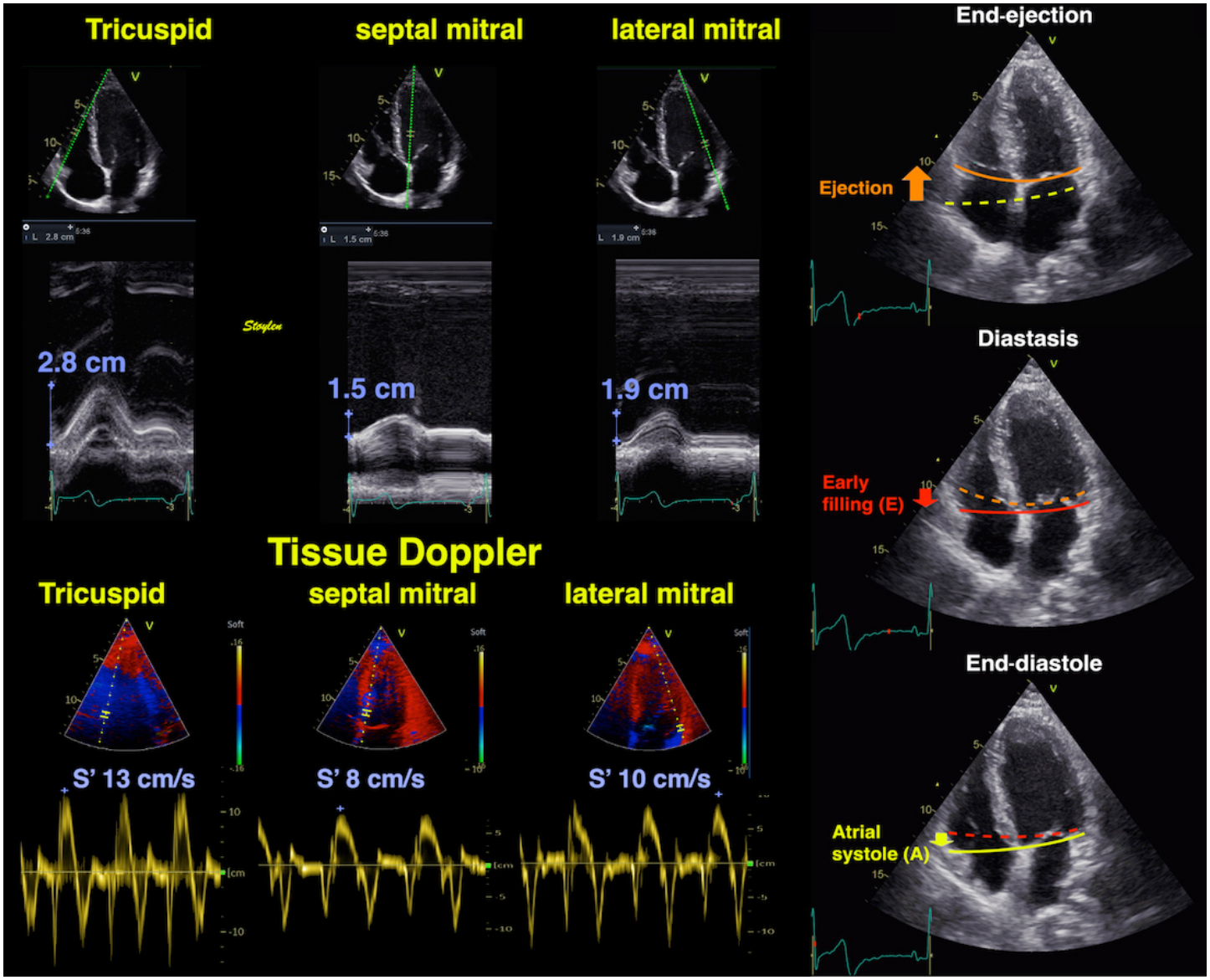
Age (years)	N	MAPSE (2 walls)	MAPSE (4 walls)	MAPSE (6 walls)
<40	330	1.69 (0.21)	1.73 (0.20)	1.67 (0.33)
40–60	656	1.55 (0.24)	1.58 (0.23)	1.51 (0.34)
>60	264	1.39 (0.25)	1.40 (0.22)	1.32 (0.32)
All	1250	1.56 (0.26)	1.58 (0.25)	1.51 (0.35)

All motion values in cm. Standard deviations in parentheses. All means (of two, four, and six walls) were significantly different ($P < .001$) from each other. Differences between age groups were all significant $P < .001$ in post hoc. There were no gender differences.

Normal annular peak S', e' and a' per wall in the HUNT study by tissue Doppler.

	Right ventricle	Anterior	septal	Inferior	lateral
PwTDI S' (cm/s)	12.6 (2.1)	8.3 (1.9)	8.0 (1.2)	8.6 (1.4)	8.8 (1.8)
PwTDI e' (cm/s)	12.9 (3.2)	11.6 (3.7)	9.9 (2.9)	11.2 (3.5)	12.5 (3.7)
PWTDI a' (cm/s)	14.3 (3.8)	9.4 (2.3)	10.2 (2.2)	11.0 (2.3)	9.4 (2.4)

Støylen A, Mølmen HE, Dalen H.Regional motion of the AV-plane is related to the cardiac anatomy and deformation of the AV-plane. Data from the HUNT study. Clin Physiol Funct Imaging. 2023 Jul 3. doi: 10.1111/cpf.12845.



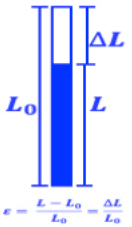
Example from a single subject. As seen both AV-plane systolic motion and AV-plane peak systolic velocity are highest in the rght lateral part, and lowest in the central part. This means that the differential motion will correspond to a systolic bending of the AV-plane, and a tilting towards the left.

What are the functional significance of the systolic AV-plane bending?

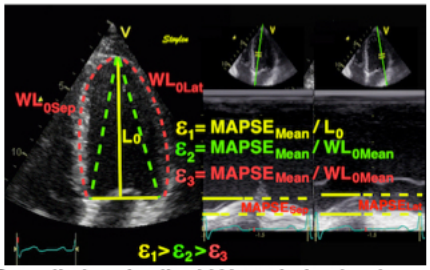
- **Firstly, Bending of the AV-plane reduces the cross sectional area.**
  - **As the tension (load) then is distributed across a smaller area, this reduces the load.**
  - **As the heart tapers from the AV-plane towards the apex, bending to a narrower diameter fits the AV plane into the narrower apical space.**
  - **The AV-plane bending take part in the transverse narrowing of the ventricles, also due to the circumferential fibre shortening, so the two mechanisms work together.**
- **Secondly, the bending of the AV-plane may be a mechanism for storing force from systole, to recoil in early diastole, meaning that the unbending is part of the restoring forces.**

The systolic bending is evident also from the systolic velocities, but from this table it is also evident that most of the unbending happens in early diastole (highest e' in the RV, lowest in the septum), while the av-plane remains straighter, but with a tilting towards the right during atrial systole (highest in the RV, lowest in the left lateral)

Relative systolic LV long axis shortening; GLS



Strain is dimension change relative to original length (Lagrangian definition) shortening is negative, lengthening is positive



Systolic longitudinal LV strain is absolute systolic shortening (MAPSE - negative value), divided by end diastolic length. In linear strain, value depends on denominator

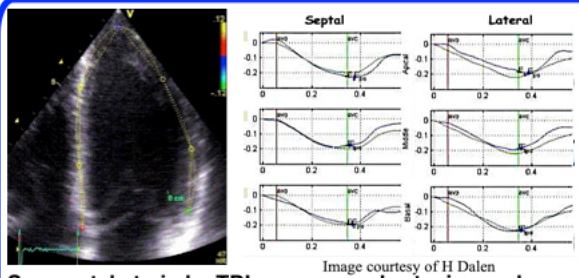
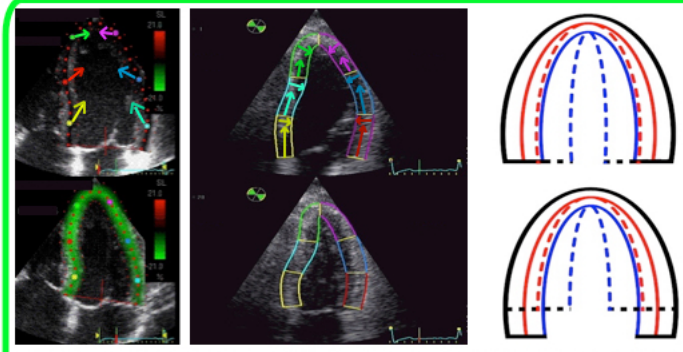
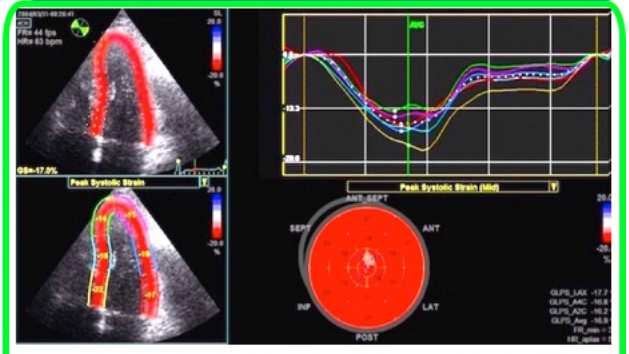


Image courtesy of H Dalen

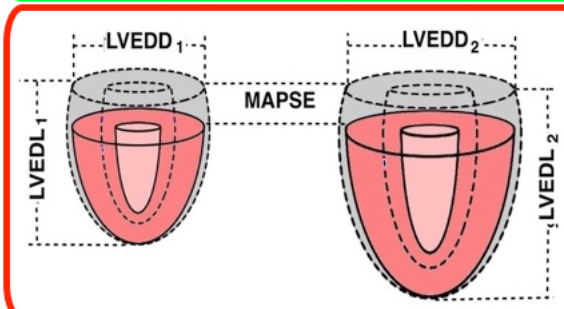
Segmental strain by TDI, measures shortening and segment length along the same straight line, so measures are fairly similar to linear strain along straight lines apex - annulus [1]



Speckle tracking follows stable acoustic markers through the heart cycle. However, as the wall shortens, it will also thicken. This means not only longitudinal, but also inwards tracking, increasing towards the endocardium. Inwards motion of the midwall and endocardial lines will give shortening even without longitudinal shortening, and this adds to the longitudinal. Thus speckle tracking strain is numerically higher than linear strain.

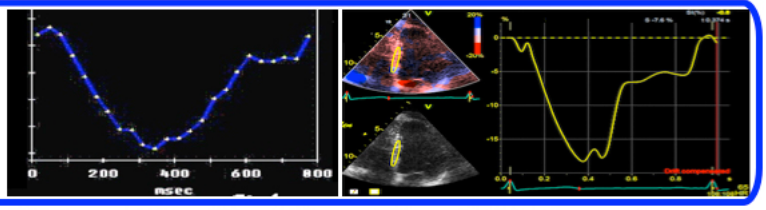


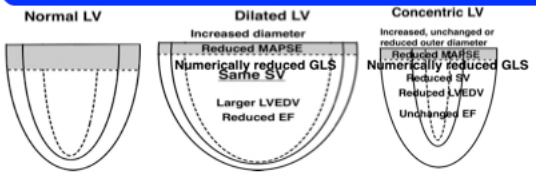
Different vendors with different hard- and software may differ in number and weighting of markers, smoothing algorithms and other functions. These are often industrial secrets, but will also contribute to inter vendor differences and method dependency.



EDV and SV correlate positively with BSA, negatively with age. EF, being SV/EDV, is thus independent of BSA and age.
MAPSE is the absolute, GLS the relative LV systolic shortening. GLS correlates with EF across a healthy population (R = 0.24). Both LV length and diameter increases with BSA, so a larger ventricle generates a higher SV, with little change in MAPSE. LV length is the denominator of GLS, so GLS decreases with increasing LV size and BSA (R = - 0.25). Thus there is no correlation of GLS with SV across a helthy population, and GLS is numerically higher in females, but this is an effect of BSA. GLS decreases with increasing age (R= - 0.40), so the decrease in MAPSE is greater than the decrease in LV length, but still, as EF is unchanged across BSA and age, GLS correlate negatively with both.[2]

During the heart cycle, the volume curve (here MUGA) and the relative wall length (strain) curve follow each other, showing the close relation between LV volume and length.





Relations of GLS to EF and SV are similar to MAPSE. In normal ventricles, MAPSE is proportional to SV and EF. In dilated HF, SV is preserved, at the expense of increased EDV. Thus, EF decreases. As LV widens, the same SV is generated by a smaller MAPSE, also giving a numerically reduced GLS. In HFpEF, EDV and SV are reduced, thus EF is unchanged . With reduced SV, MAPSE is reduced in line with SV, giving numerically reduced GLS.

Thus GLS is reduced both in HFrEF and HFpEF, and is a better prognosticator than EF. [3]

However, BSA is a factor reducing GLS, and hence a confounder, so GLS is not as good as MAPSE. [4]

References

1 Støylen A, Mølmen HE, Dalen H. Relation between Mitral Annular Plane Systolic Excursion and Global longitudinal strain in normal subjects: The HUNT study. Echocardiography. 2018;35:603–610

2 Støylen A, Dalen H, Molmen HE.Left ventricular longitudinal shortening: relation to stroke volume and ejection fraction in ageing, blood pressure, body size and gender in the HUNT3 study. Open Heart. 2020 Sep;7(2):e001243. doi: 10.1136/openhrt-2020-001243

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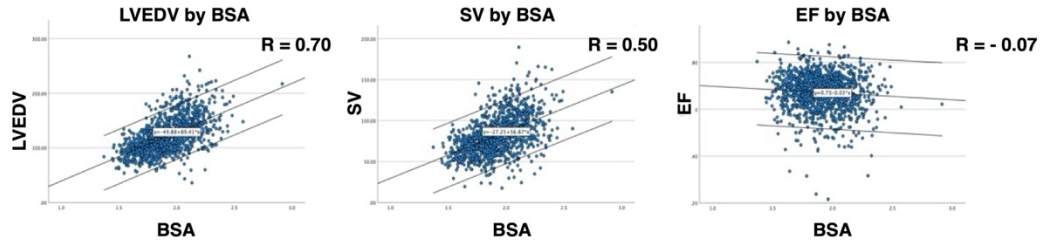
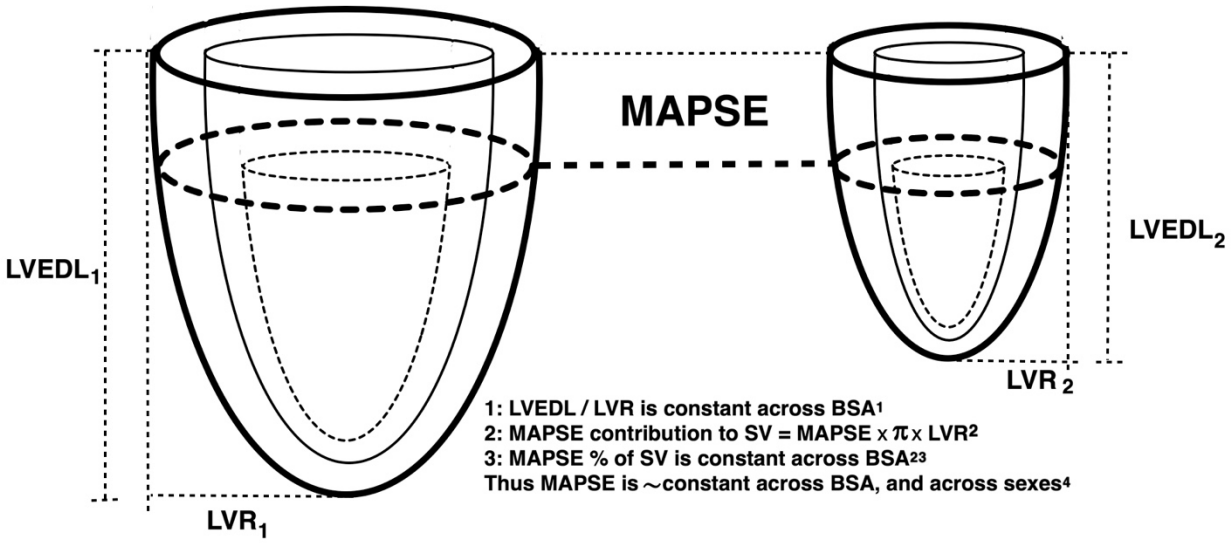
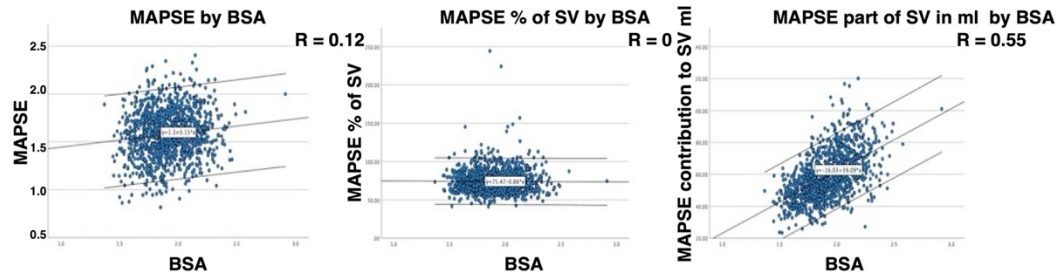
SV ~ BSA, EDV ~ BSA, EF is near constant across BSA

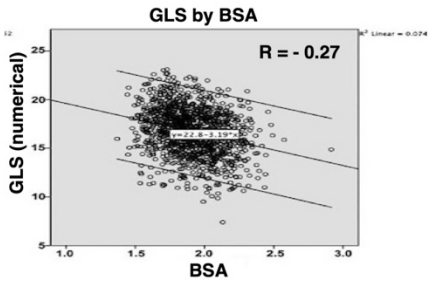




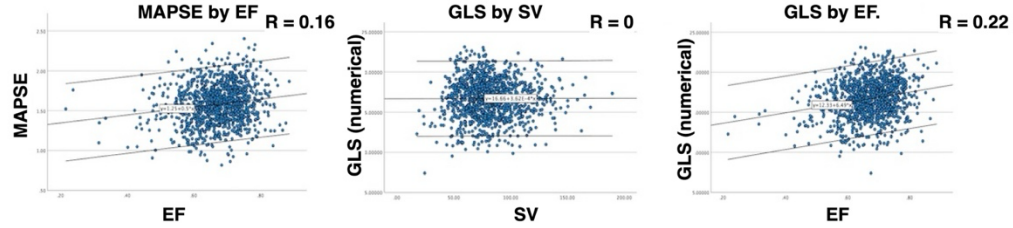
1: LVEDL / LVR is constant across BSA[1]
2: MAPSE contribution to SV = MAPSE x π x LVR²
3: MAPSE % of SV is constant across BSA[2,3]
Thus MAPSE is ~constant across BSA, and across sexes[4]



4: As LVEDL / LVR is constant over BSA,
$LVEDL_1 / LVR_1 = LVEDL_2 / LVR_2$,
but $LVEDL_1 > LVEDL_2$
5: As MAPSE is constant over BSA,
$MAPSE_1 = MAPSE_2$
6: GLS (in numerical values) = MAPSE / LVEDL.
Thus $GLS_1 < GLS_2$; GLS (in absolute values) is inversely related to BSA[4]

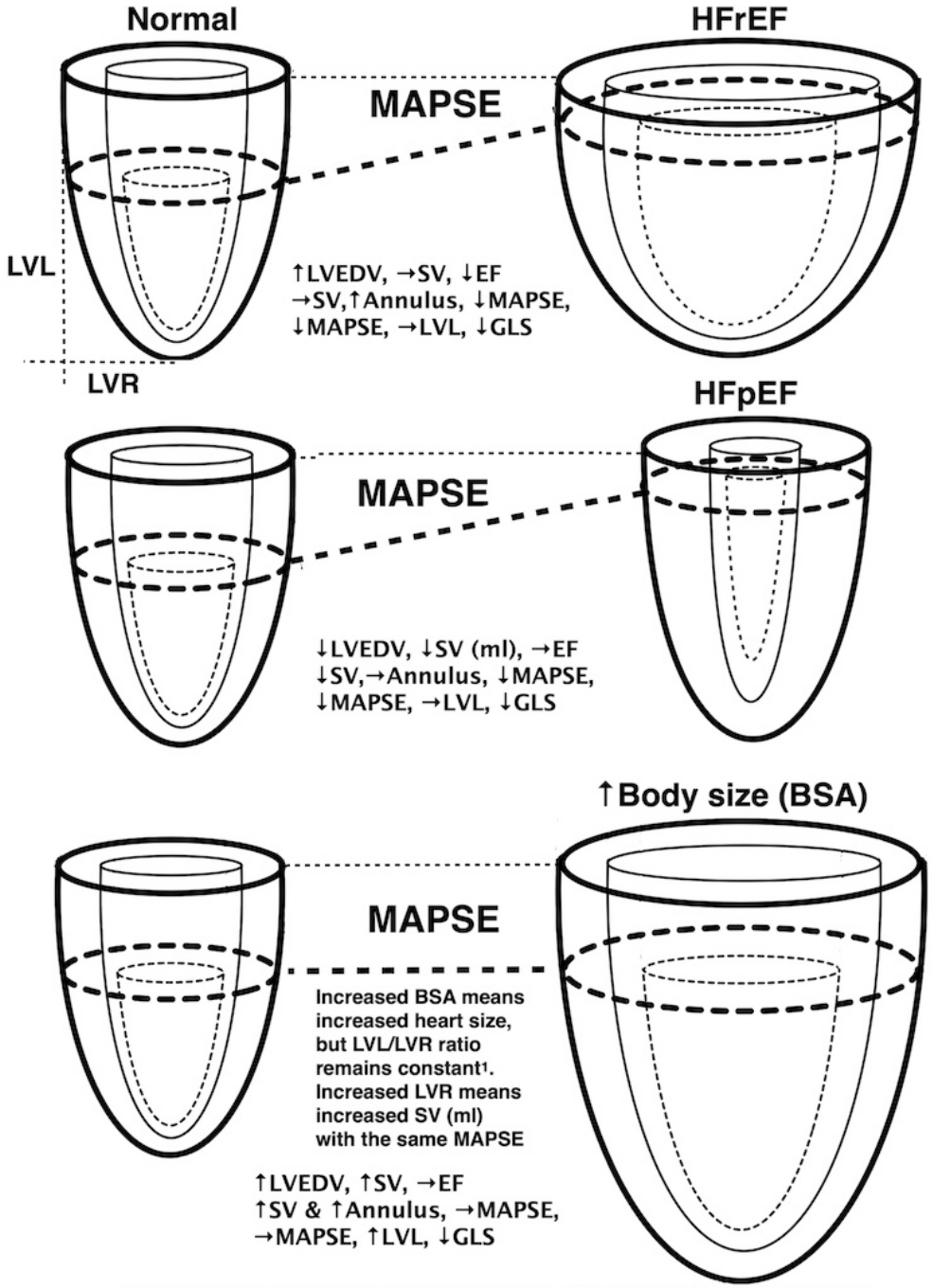


7: MAPSE is ~independent of BSA, EF is independent of BSA, MAPSE is weakly related to EF.[3]
8: GLS (numerical) is negatively related to BSA, SV is positively related to BSA, GLS do not correlate with SV[3]
9: GLS (numerical) is negatively related to BSA, EF is independent of BSA, GLS (numerical) is weakly related to EF[3]



1 Støylen A, Mølmen HE, Dalen H. Importance of length and external diameter in left ventricular geometry. Normal values from the HUNT Study. Open Heart. 2016 Sep 21;3(2):e000465.
2 Carlsson M, Ugander M, Mosen H, Buhre T and Arheden H. Atrioventricular plane displacement is the major contributor to left ventricular pumping in healthy adults, athletes, and patients with dilated cardiomyopathy. American journal of physiology Heart and circulatory physiology (2007); 292: H1452-1459.
3 Støylen A, Dalen H, Molmen HE.Left ventricular longitudinal shortening: relation to stroke volume and ejection fraction in ageing, blood pressure, body size and gender in the HUNT3 study. Open Heart. 2020 Sep;7(2):e001243.
4 Støylen A, Mølmen HE, Dalen H. Relation between Mitral Annular Plane Systolic Excursion and Global longitudinal strain in normal subjects: The HUNT study. Echocardiography. 2018;35:603–610

MAPSE % of SV is constant across BSA
GLS (numerical) = MAPSE / LVEDL
MAPSE contribution to SV (ml) = MAPSE x π x LVR



Thus: Unchanged MAPSE and increased LVL, means reduced (numerical) GLS

Thus: **EF is insensitive for HFpEF, but is not confounded by BSA, and is a prognosticator for HFrEF only, but not overall for HF[2].**
GLS is sensitive to both HFrEF and HFpEF, and is an over all prognosticator for HF[3], but is confounded by BSA[4]
MAPSE is sensitive to both HFrEF and HFpEF[2], is not confounded by BSA[4], and is thus a better over all prognosticator for HF than GLS[5]

However: Both GLS and MAPSE decrease by increasing age, while EF do not[6].
GLS, MAPSE and EF are all load dependent

1 Støylen A, Mølmen HE, Dalen H. Importance of length and external diameter in left ventricular geometry. Normal values from the HUNT Study. Open Heart. 2016 Sep 21;3(2):e000465.
2 Willenheimer R, Cline C, Erhardt L, Israelsson B. Left ventricular atrioventricular plane displacement: an echocardiographic technique for rapid assessment of prognosis in heart failure. Heart 1997;78:230-36.
3 Cho GY, Marwick TH, Kim HS, Kim MK, Hong KS, Oh DJ. Global 2-dimensional strain as a new prognosticator in patients with heart failure. J Am Coll Cardiol. 2009 Aug 11;54(7):618-24.
4 Støylen A, Mølmen HE, Dalen H. Relation between Mitral Annular Plane Systolic Excursion and Global longitudinal strain in normal subjects: The HUNT study. Echocardiography. 2018;35:603–610
5 Automated In-Line Artificial Intelligence Measured Global Longitudinal Shortening and Mitral Annular Plane Systolic Excursion: Reproducibility and Prognostic Significance.
Xue H, Artico J, Davies RH, Adam R et al.J Am Heart Assoc. 2022 Feb 15;11(4)
6 Støylen A, Dalen H, Mølmen HE.Left ventricular longitudinal shortening: relation to stroke volume and ejection fraction in ageing, blood pressure, body size and gender in the HUNT3 study. Open Heart. 2020 Sep;7(2):e001243.

Why do we need strain, if MAPSE is better?

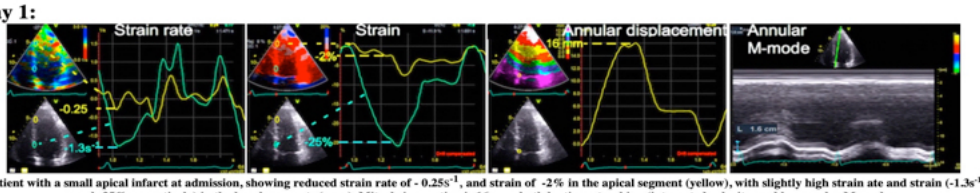
Regional function

MAPSE is a better prognosticator than GLS, as it is not confounded by body size, and thus, possibly a better diagnostic too, for reduced global systolic performance. Why then do we need strain / strain rate?

In ischemia, both MAPSE, S' and GLS is reduced, reflecting the total loss of contractile function[1]. But S' and regional displacement of the mitral annulus, do not identify the location of ischemia[2 3]. Only segmental strain / SR identifies infarct site[2].

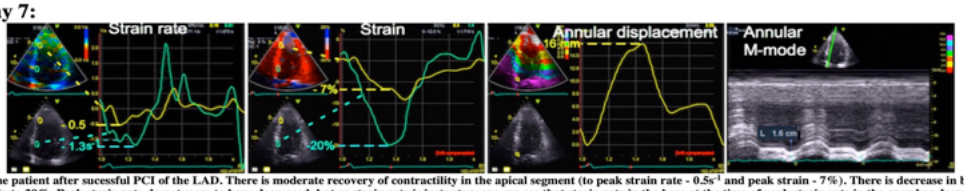
The infarct recovery cycle shows initial hypokinesis in the infarcted apical segment, and hyperkinesia in the normal basal segment. In recovery, there is partial recovery of shortening in the infarct, regress of hyperkinesia in the healthy segments[4]. The hypo- and hyperkinesis cancel out, giving the same displacement in the acute as the recovery phase, in the adjacent part of the mitral annulus.

Day 1:



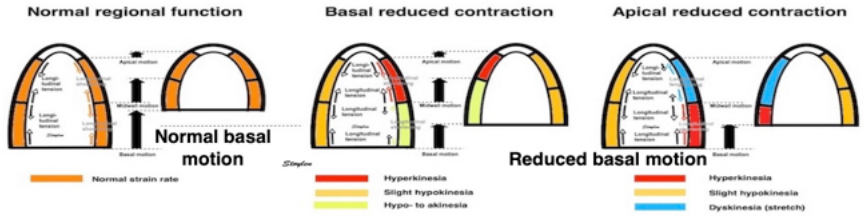
Patient with a small apical infarct at admission, showing reduced strain rate of - 0.25s^{-1}, and strain of -2% in the apical segment (yellow), with slightly high strain ate and strain (-1.3s^{-1} and -25%, repectively) in the basal segments (cyan). Mitral ring motion is 16 mm, both by tissue tracking (integrated velocity, and by annular M-mode.

Day 7:



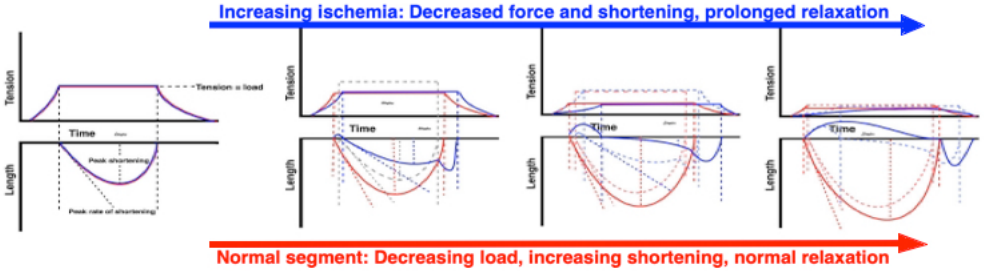
Same patient after sucessful PCI of the LAD. There is moderate recovery of contractility in the apical segment (to peak strain rate - 0.5s^{-1} and peak strain - 7%). There is decrease in basal strain to 20%. Peak strain rate do not seem to have decreased, but as strain rate is instantaneous, we see that strain rate in the base at the time of peak strain rate in the apex has decreased to - 1s^{-1}. The reciprocal changes in strain in the two segments results in no change in the regional annulus motion which still is 16 mm by both methods.

Systolic regional deformation and segment interaction

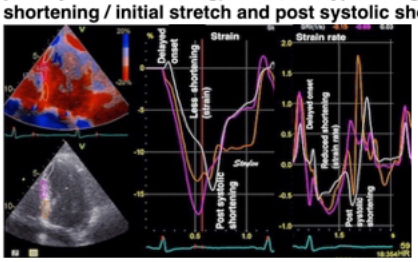


Thus global MAPSE is reduced in relation to the total infarct size but cannot resolve the infarct locatien, due to the compensatory interaction of hyper- and hypokinesia. Only strain / SR can identify the infarcted segments[2].

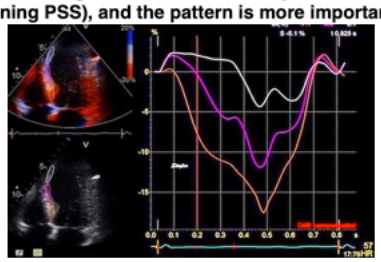
The reason is the afterload sensitivity of strain /strain rate. The afterload of a segment is both intraventricular pressure, but also the tension from neighboring segments. In regional dysfunction, the load becomes asymmetric, and so does the shortening.

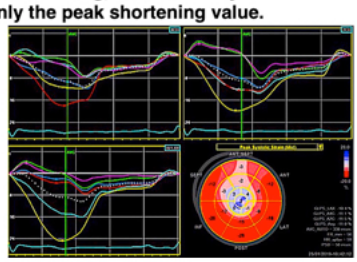


Decreasing contractility (force) in an ischemic segment (blue) will reduce shortening and rate of force development with initial stretch. The non ischemic segment (red) will experience decreasing afterload (force from dysfunctional segment), and shorten more. In ischemia, relaxation is slowed by energy depletion, and (relatively) increased afterload, prolonging tension and shorten when normal segment is relaxing (ischemic post systolic shortening). Thus, the typical segmentl finding in ischemia is not only decreased systolic shortening, but also delayed onset of shortening / initial stretch and post systolic shortening PSS), and the pattern is more important than only the peak shortening value.



Small apical infarct, showing delayed onset, systolic hypokinesia and post systolic shortening (PSS) in the apical segment (white).

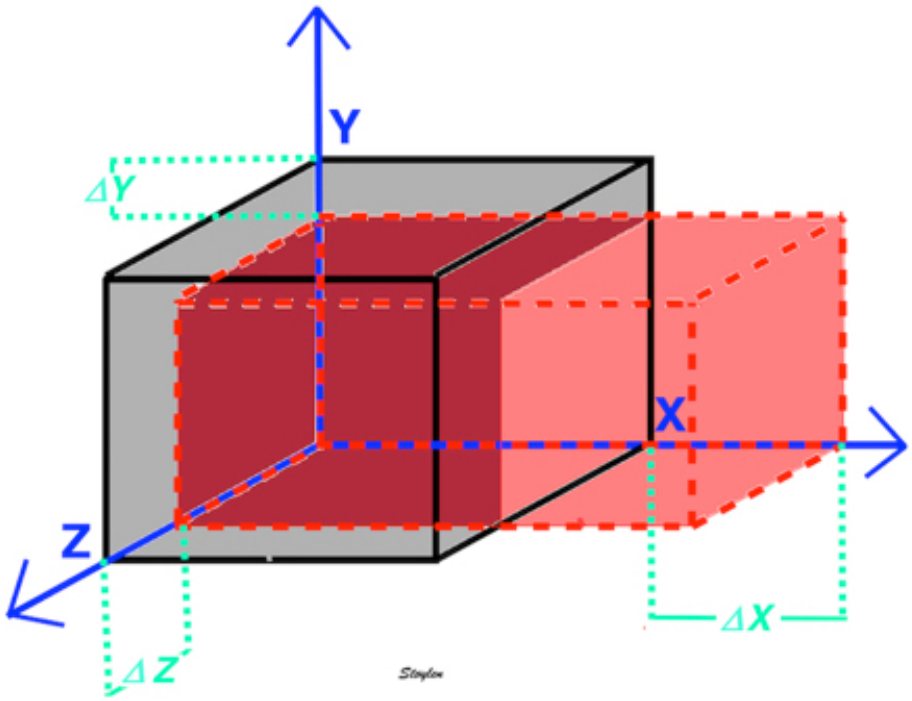




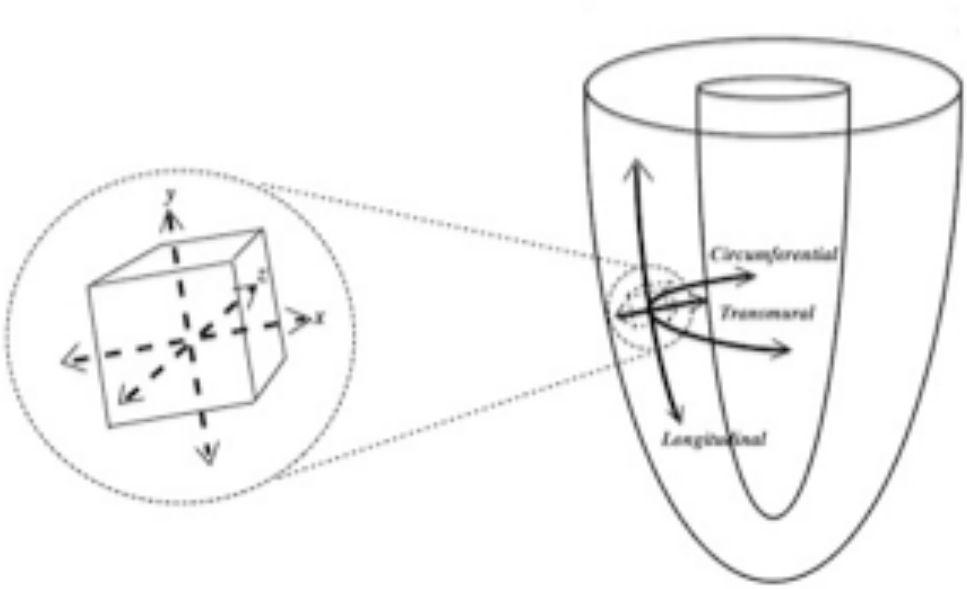
Large apical infarct, showing apical dyskinesia with PSS (white), midwall initial stretch and systolic hypokinesia with PSS (magenta) and basal delayed onset with near normokinesia with PSS (orange). Left speckle tracking strain from all walls of the same patient, showing the same pattern, and bull's eye of the syst. shortening.

1 Höglund C, Alam M, Thorstrand C. Effects of acute myocardial infarction on the displacement of the atrioventricular plane: an echocardiographic study. J Intern Med 1989 Oct;226(4):251-6
2 Stoylen A, Skjaerpe T. Systolic long axis function of the left ventricle. Global and regional information. Scand Cardiovasc J. 2003 Sep;37(5):253-8.
3 Pahlm U, Seemann F, Engblom H, Gyllenhammar T, Halvorsen S, Hansen HS, Erlinge D, Atar D, Heiberg E, Arheden H, Carlsson M. Longitudinal left ventricular function is globally depressed within a week of STEMI. Clin Physiol Funct Imaging. 2018 Apr 27
4 Ingul CB, Stoylen A, Slordahl SA. Recovery of stunned myocardium in acute myocardial infarction quantified by strain rate imaging: a clinical study. J Am Soc Echocardiogr. 2005 May;18(5):401-10.

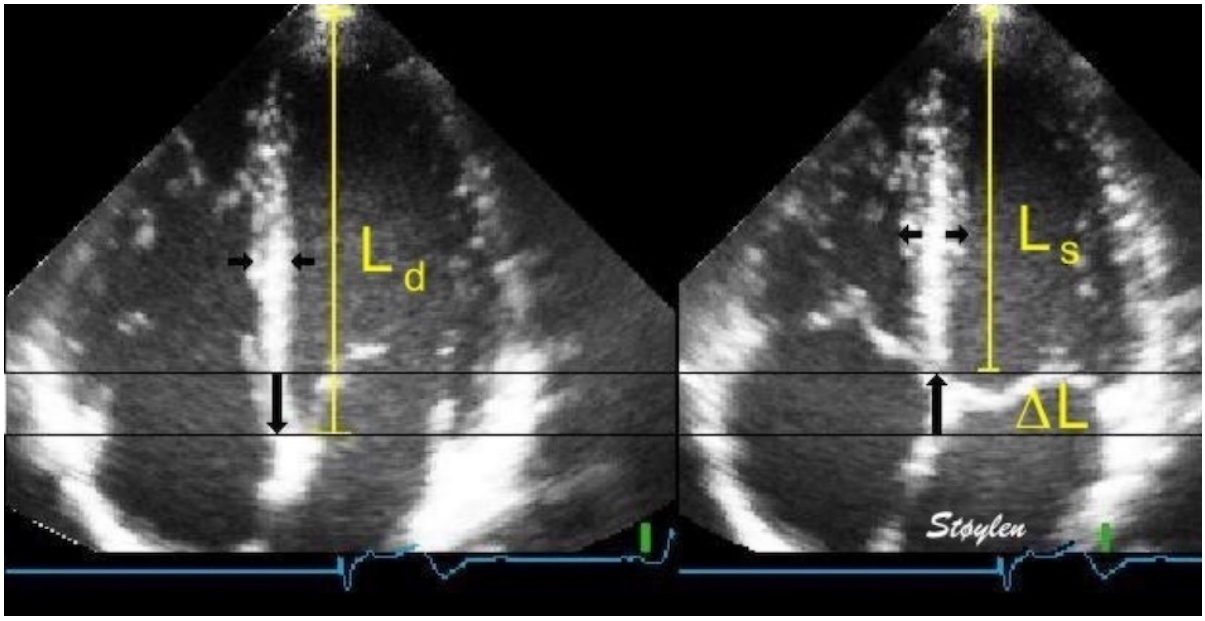
Strain in three dimensions



$\varepsilon_x = \Delta X / X_0 \quad \varepsilon_y = \Delta Y / Y_0 \quad \varepsilon_z = \Delta Z / Z_0$



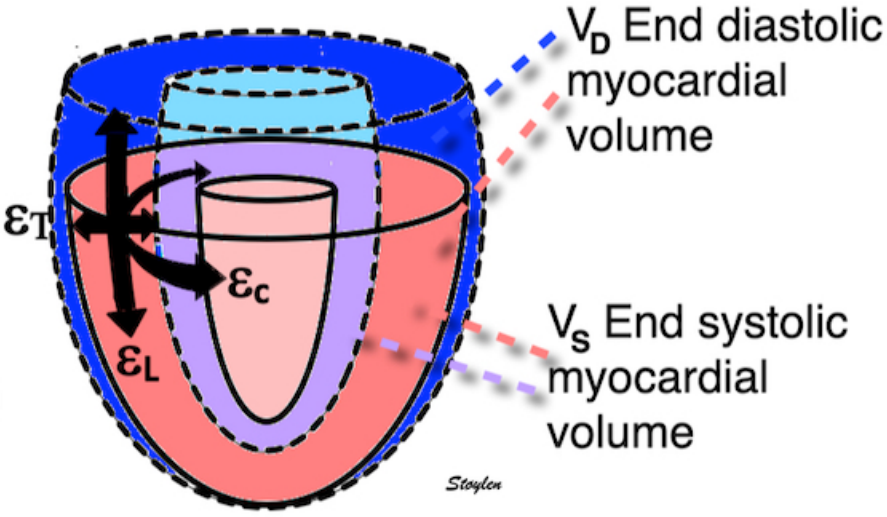
- **The cardiac coordinates are also cartesian. Strain directions are spatial coordinates of deformation, not fibre function**



As long as the myocardium is partly incompressible, longitudinal shortening must result in transmural thickening.

Volume before deformation (V_0) = $X_0 * Y_0 * Z_0$
Volume after deformation $V = (X_0 + \Delta X)(Y_0 + \Delta Y)(Z_0+\Delta Z)$
$\varepsilon_x = \Delta X / X_0$ $\varepsilon_y = \Delta Y / Y_0$ $\varepsilon_Z = \Delta Z / Z_0$
$V / V_0 = (X_0 + \Delta X) / X_0 * (Y_0 + \Delta Y)/Y_0 * (Z_0 + \Delta Z)/Z_0 = (1 + \Delta X/X_0) (1+ \Delta Y/Y_0) (1+ \Delta Z/Z_0)$

$$V / V_0 = (1 + \varepsilon_x) (1 + \varepsilon_y) (1+\varepsilon_Z)$$

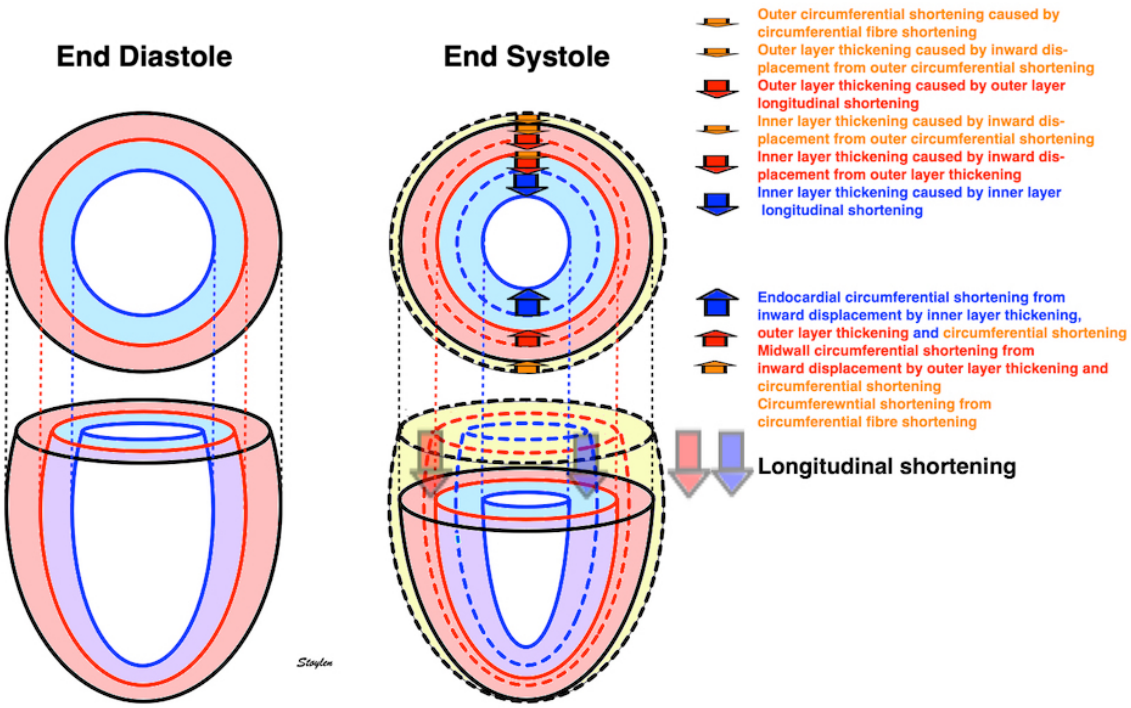


ε_L Longitudinal strain
ε_T Transmural (radial) strain
ε_C Circumferential strain

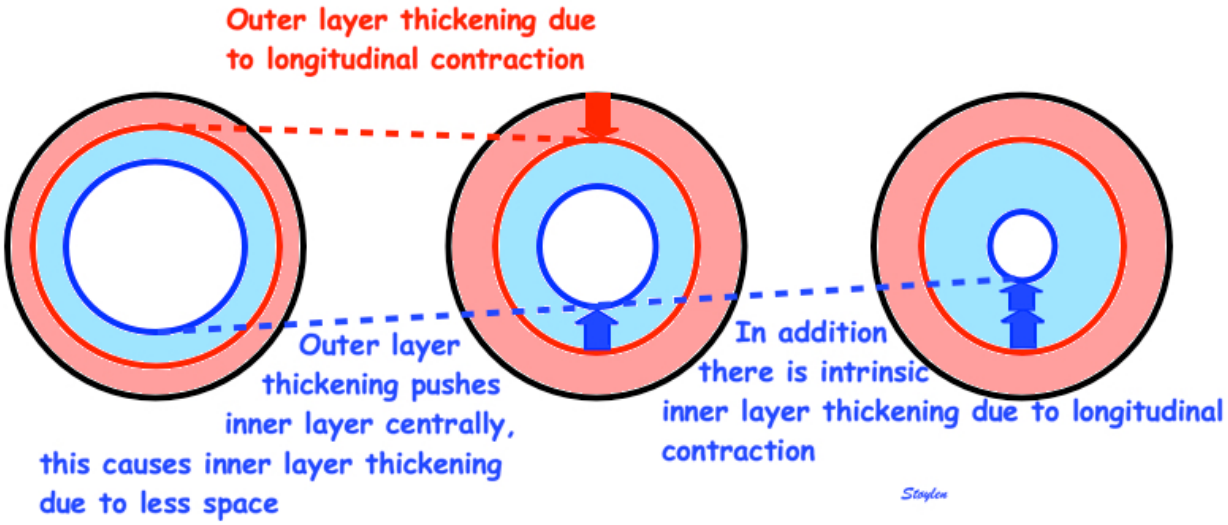
$$V_S / V_D = (\varepsilon_L +1) * (\varepsilon_T +1) * (\varepsilon_C +1)$$

If the myocardium is incompressible, $V_S / V_D = 1$
If the myocardium in somewhat incompressible, $V_S / V_D < 1$, less the nmore compressible.

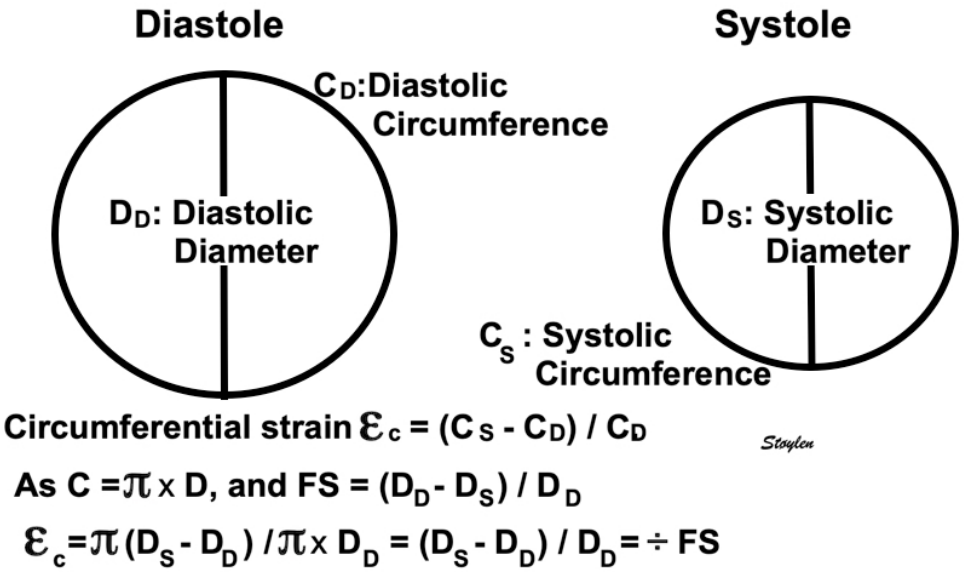
The three strains are inter related:



Transmural strain – wall thickening:

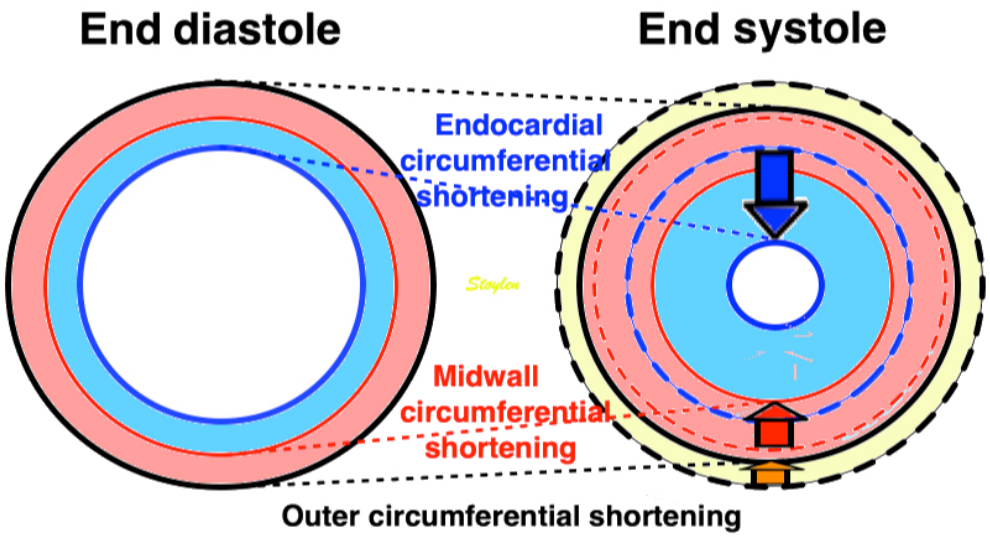


Gradient of thickening illustrated by two layers: Left, end diastole. Disregarding outer contour decrease, the outer layer thickens due to longitudinal shortening (middle). That means the outer layer pushes the inner layer towards the centre, where there is less room. This effect alone will cause the inner layer to thicken due to the reduced diameter. In addition, the inner layer also shortens due to longitudinal shortening. This means that the inner layer thickens due to both shortening and inward displacement, and thus thickening more than the outer layer.



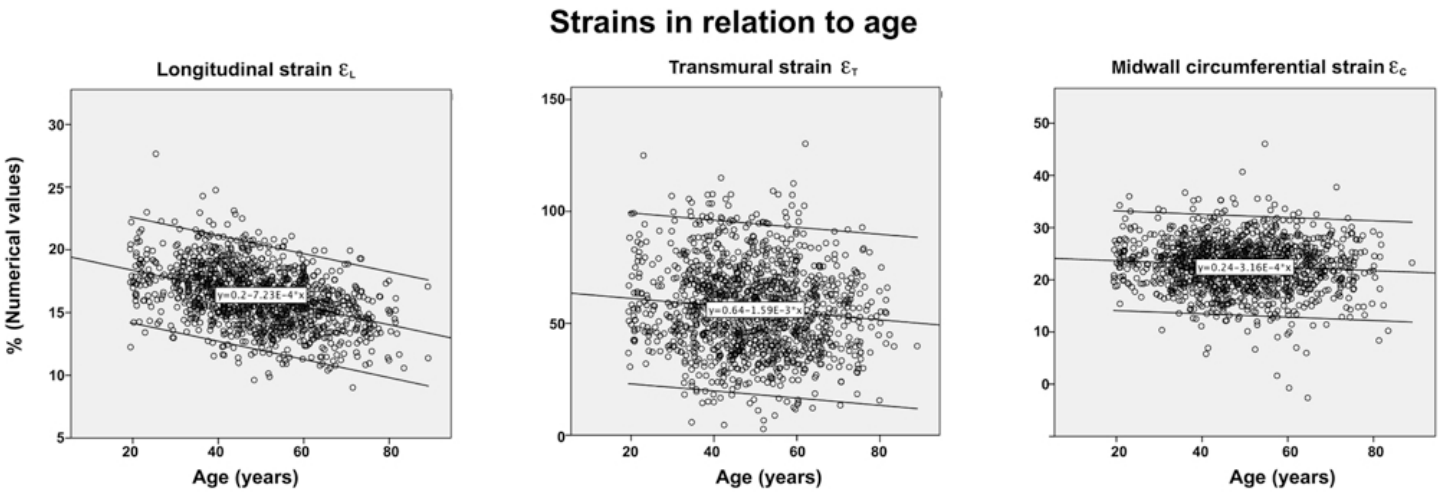
As diameter and circumference are proportional, fractional circumferential shortening and fractional diameter shortening are equal.

Wall thickening means displacement of midwall contour



1. **Outer circumferential strain reflects circumferential fibre shortening**
2. **Midwall circumferential strain is the most representative, and the one demonstrating the interaction between strains, but is partly a function of wall thickening, which again is a function of**

    1. **longitudinal shortening and**
    2. **circumferential outer shortening and wall thickening**
3. **Endocardial circumferential strain equals fractional diameter shortening, but is mainly wall thickening**

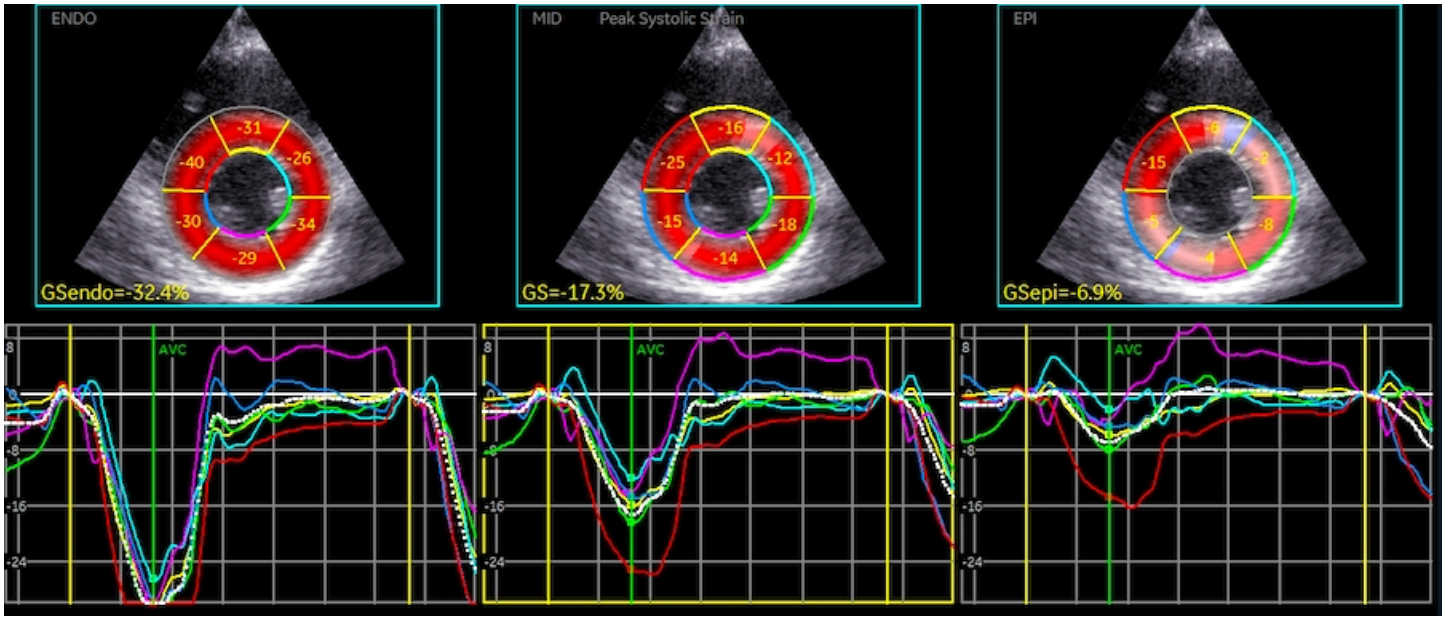


All three strains decline numerically by age.

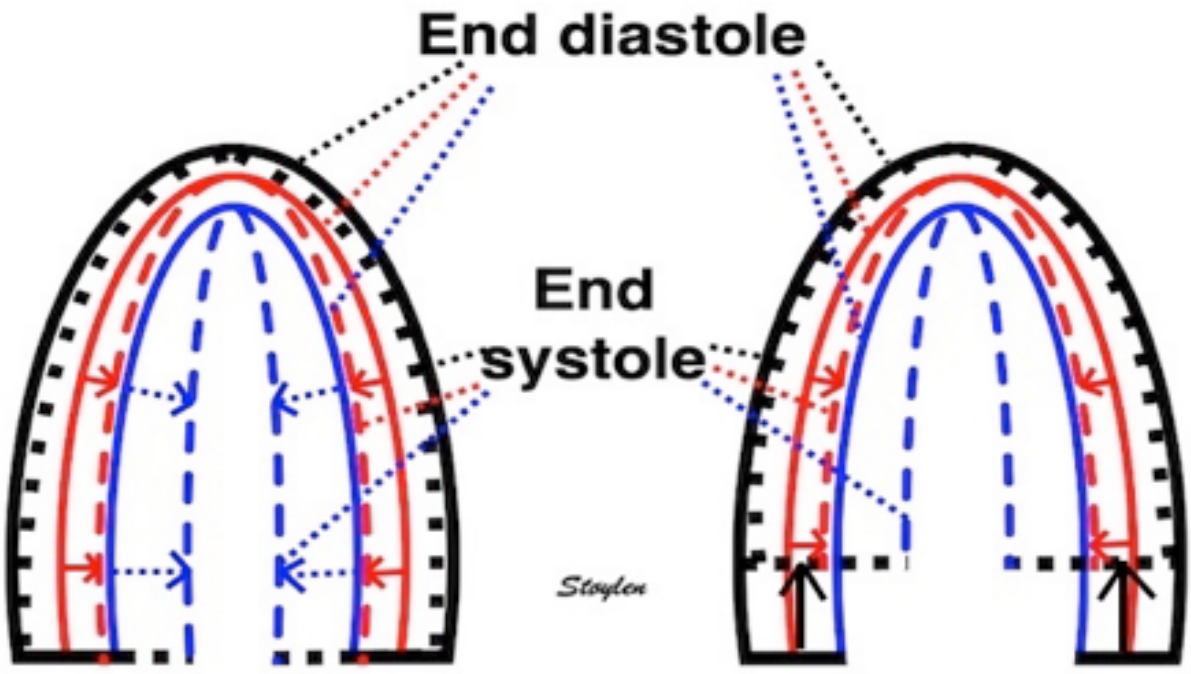
Age (years)	ε_L (%)	Wall thickness (mm)	ε_T (= Wall thickening) (%)	LVIDD (mm)	Endo-card ε_C (=_FS) (%)	Midwall ε_C (%)	External ε_C (%)
Women							
<40	-18.1 (2.0)	7.6 (1.3)	45.8 (25,7)	49.3 (4.2)	-36.6 (6.1)	-23.9 (4.1)	-14.1 (3.3)
40 – 60	-17.0 (2.2)	8.2 (1.3)	44.6 (23.7)	48.8 (4.5)	-36.5 (6.9)	-23.2 (4.8)	-13.2 (4.2)
> 60	-14.8 (2.1)	8.8 (1.4)	43.7 (22.6)	47.8 (4.8)	-36.0 (9.1)	-22.3 (5.6)	-12.1 (4,2)
Total	-17.0 (2.4)	8.2 (1.4)	44.8 (24.1)	48.8 (4.5)	-36.4 (7.1)	-23.2 (4.8)	-13.3 (4.0)
Men							
<40	-16.5 (2.0)	9.0 (1.3)	44.5 (19.9)	53.5 (4.9)	-35.5 (6.9)	-22.4 (4.6)	-12.6 (3.7)
40 – 60	-15.4 (1.9)	9.6 (1.4)	44.1 (22.6)	53.0 (5.5)	-35.8 (7.4)	-22.2 (4.9)	-12.2 (3.8)
> 60	- 14.9 (1.9)	10.1 (1.5)	41.3 (18.8)	52.1 (6.4)	-36.0 (8.0)	-21.9 (5.2)	-11.8 (4.4)
Total	- 15.5 (2.0)	9.6 (1.5)	43.5 (21.1)	52.9 (5.6)	-35.8 (7.5)	-22.2 (4.9)	-12.2 (3.9)
All	**- 16.3 (2.4)**	**8.8 (1.6)**	**44.2 (22.7)**	**50.8 (5.4)**	**-36.1 (7.3)**	**-22.7 (4.9)**	**-12.8 (4.0)**

Wall thickness, cavity diameter, Longitudinal, transmural and endocardial, midwall and outer circumferential strains by linear measurements from the HUNT3 study. Mean and standard deviations are given. LVIDD do not change with age. WT incrrases with age, Longitudinal transmural and external and midwall circumferential strains decline with increasing age, endocardial strain (AKA FS) do not.

Støylen A, Mølmen HE, Dalen H. Left ventricular global strains by linear measurements in three dimensions: interrelations and relations to age, gender and body size in the HUNT Study. Open Heart 2019;6:e001050.



Transmural gradient of circumferential strain, Endo > midwall > epi (in numerical values)

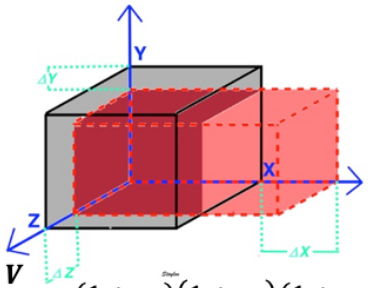


Speckle tracking tracks both inward and longitudinally. Inward motion will decrease length, even without true longitudinal shortening. This virtual shortening adds to the true longitudinal shortening, increasingly towards the endocardium, and the observed gradient is a geometric artefact due to the tracking.

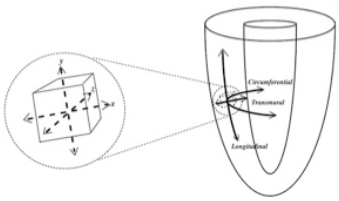
Cheat sheet on strain geometry

Strain (ε) is relative deformation (dimension change)

$$\varepsilon = \frac{L - L_0}{L_0} = \frac{\Delta L}{L_0}$$



Tissue is three- dimensional, and strain is simultaneous in all three dimensions, ε_x, ε_y and ε_z. In the left ventricle the three main strain directions are longitudinal (ε_L), transmural (ε_T) and circumferential (ε_C), which in reality also are cartesian.

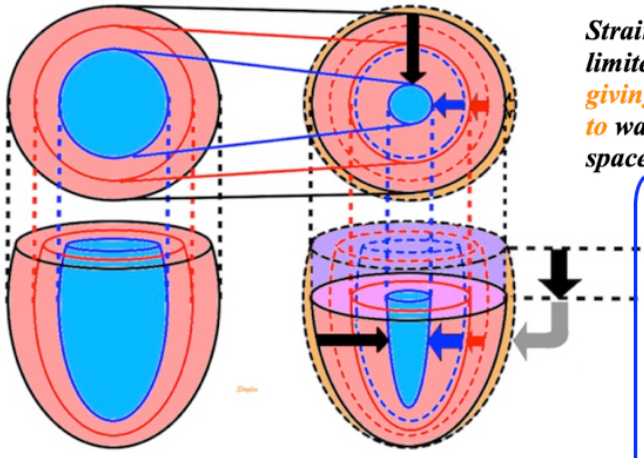


Strains are related to tissue volume changes :

https://stoylen.folk.ntnu.no/strainrate/Motion%20and%20deformation.html#incomp

$$\frac{V}{V_0} = (1+\varepsilon_x)(1+\varepsilon_y)(1+\varepsilon_Z) \qquad \frac{V}{V_0} = (1+\varepsilon_L)(1+\varepsilon_T)(1+\varepsilon_C)$$

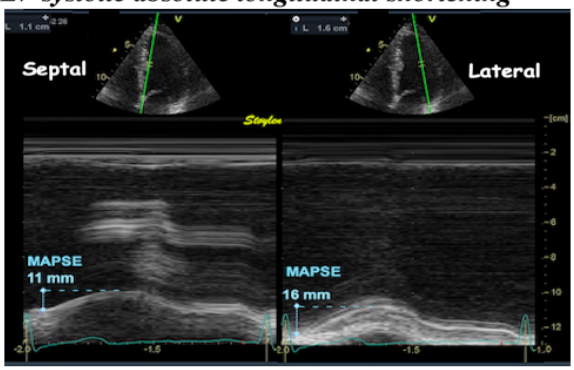
In an incompressible object the deformations cancel out, and $\frac{V}{V_0} = 1$ *(volume conservation).*



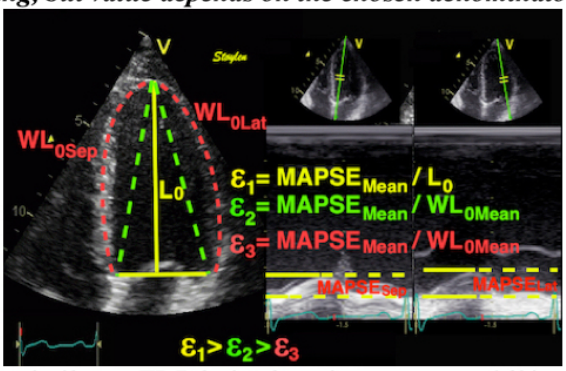
Strains are interdependent as the myocardium has limited compressibility. There is circumferential shortening, giving ca 10% outer diameter decrease, but this also contributes to wall thickening as the wall is pushed inwards into more limited space.

Longitudinal shortening contributes to wall thickening through volume conservation, both of outer and inner layer. Inner layer thickens most, as there is less space towards the center. Wall thickening pushes both midwall and endocardial circumference inwards (circumferential shortening); endocardial most. This is added to outer circumferential shortening. As diameter is proportional with circumference ($C = \pi D$) circumferential shortening = freactional shortening, midwall or endocardial, respectively. Thus, longitudinal strain carries most of the information

Mitral Annular Plane Systolic Excursion (MAPSE) = LV systolic absolute longitudinal shortening

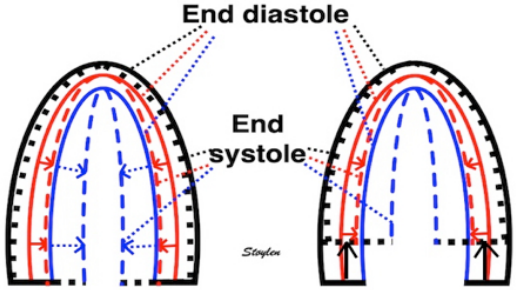


Global longitudinal strain (GLS) = LV systolic relative shortening, but value depends on the chosen denominator



Linear strain, using straight lines from annulus to apex (ε_2) is similar to TDI derived strain, mean ca - 16%.

Speckle tracking tracks both inward and longitudinally. Inward motion will decrease length, even without true longitudinal shortening. This virtual shortening adds to the true longitudinal shortening. Speckle tracking GLS (midwall) is thus normally around -19% increases towards the endocardium.



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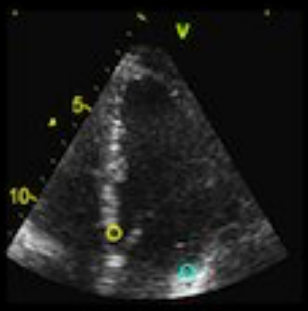
Støylen A, Mølmen HE, Dalen H. Relation between Mitral Annular Plane Systolic Excursion and Global longitudinal strain in normal subjects: The HUNT study. Echocardiography. 2018;35:603–610

Støylen A, Mølmen HE, Dalen H. Left ventricular global strains by linear measurements in three dimensions: interrelations and relations to age, gender and body size in the HUNT Study. Open Heart 2019;6:e001050.

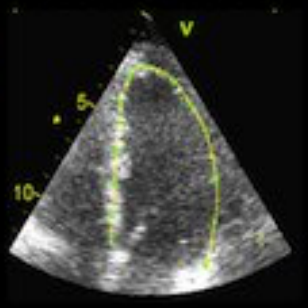
The heart cycle in motion and deformation

Basal velocities

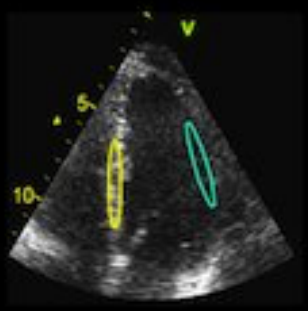


Basal displacements - MAPSE

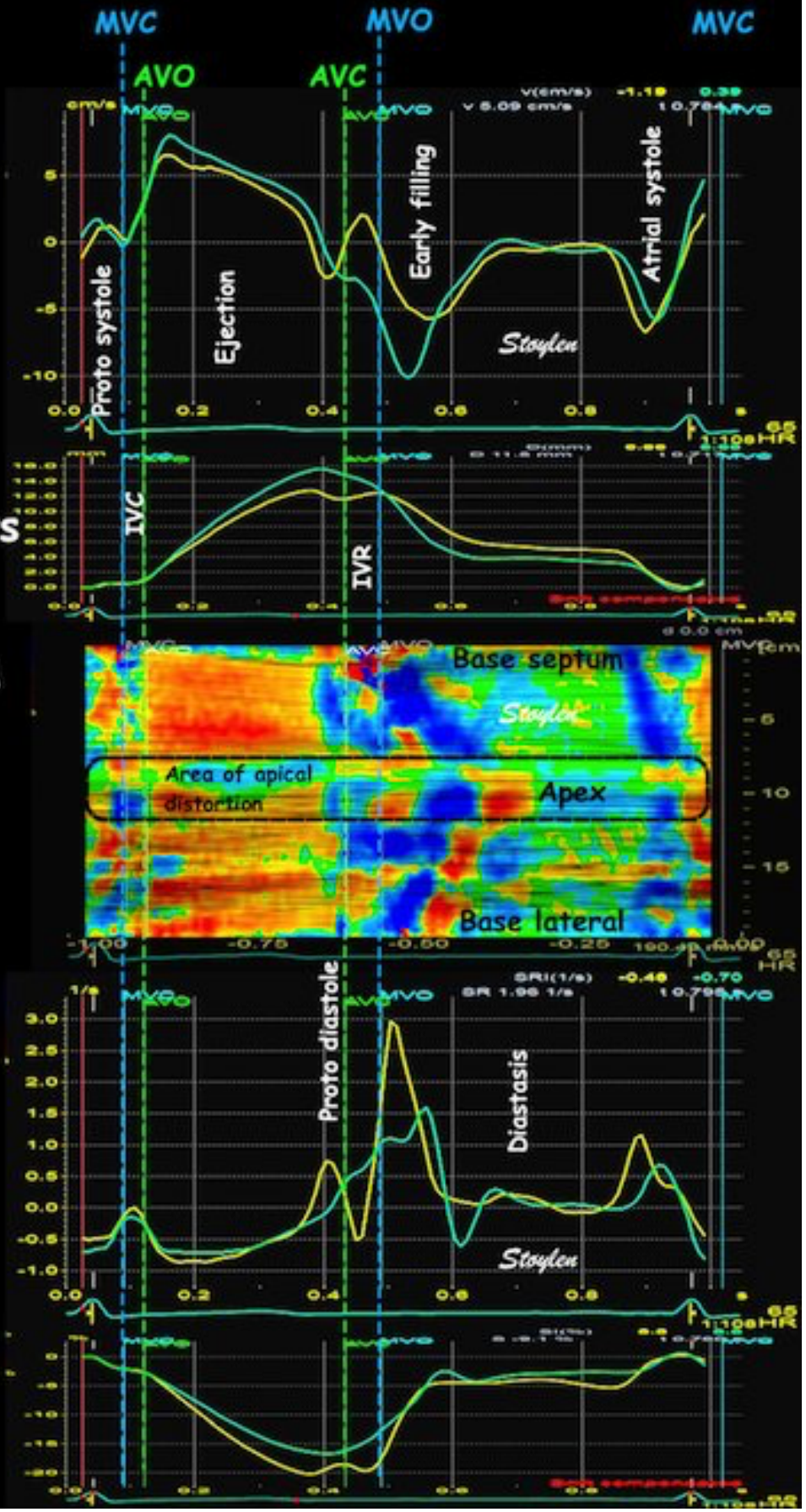
Strain rate CAMM



Strain rate

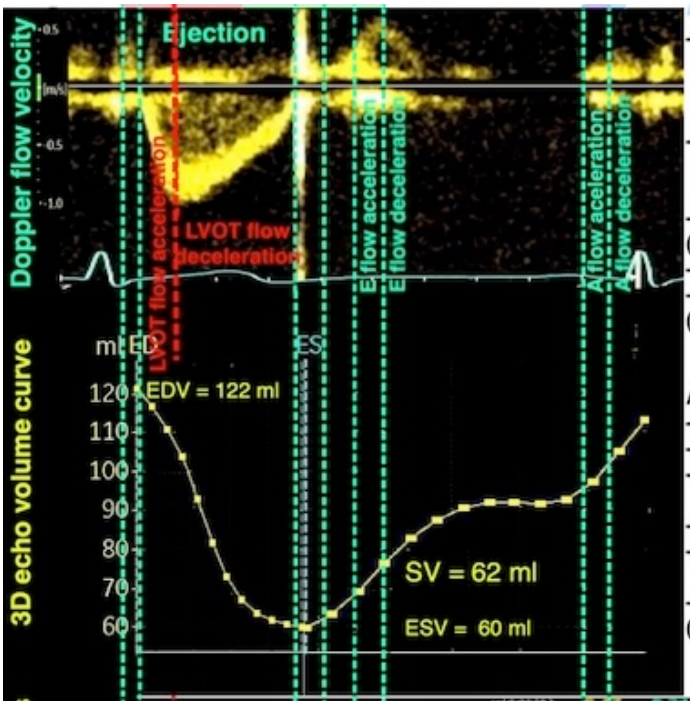


Strain



Flow

Vs longitudinal motion and velocity



Atrial relaxation

- Blood is accelerated by pressure gradients
  accelerates along a gradient
  decelerates against a gradient

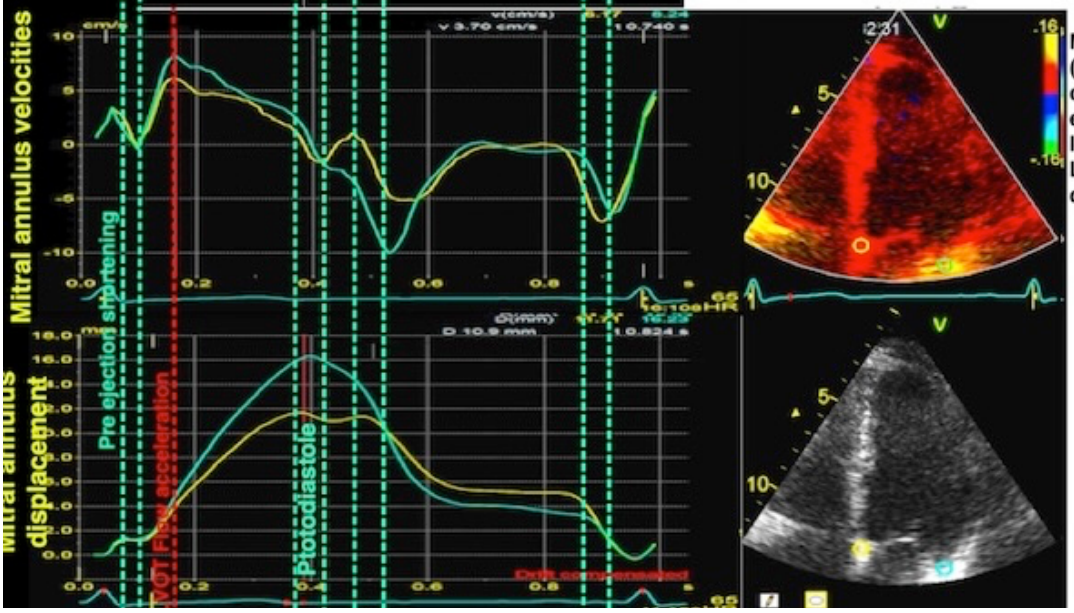
- Blood flow = the rate of volume change

- Blood flow is the first derivative of volume
(and, given constant orifices and compliance; flow velocity)
- Acceleration is the second derivative of volume
- Acceleration force is the pressure gradients
(tension versus load)

Annular motion is related to volume changes
- MAPSE is the absolute LV length change
- MAPSE correlates with SV
- Annulus displacement is thus related to volume changes

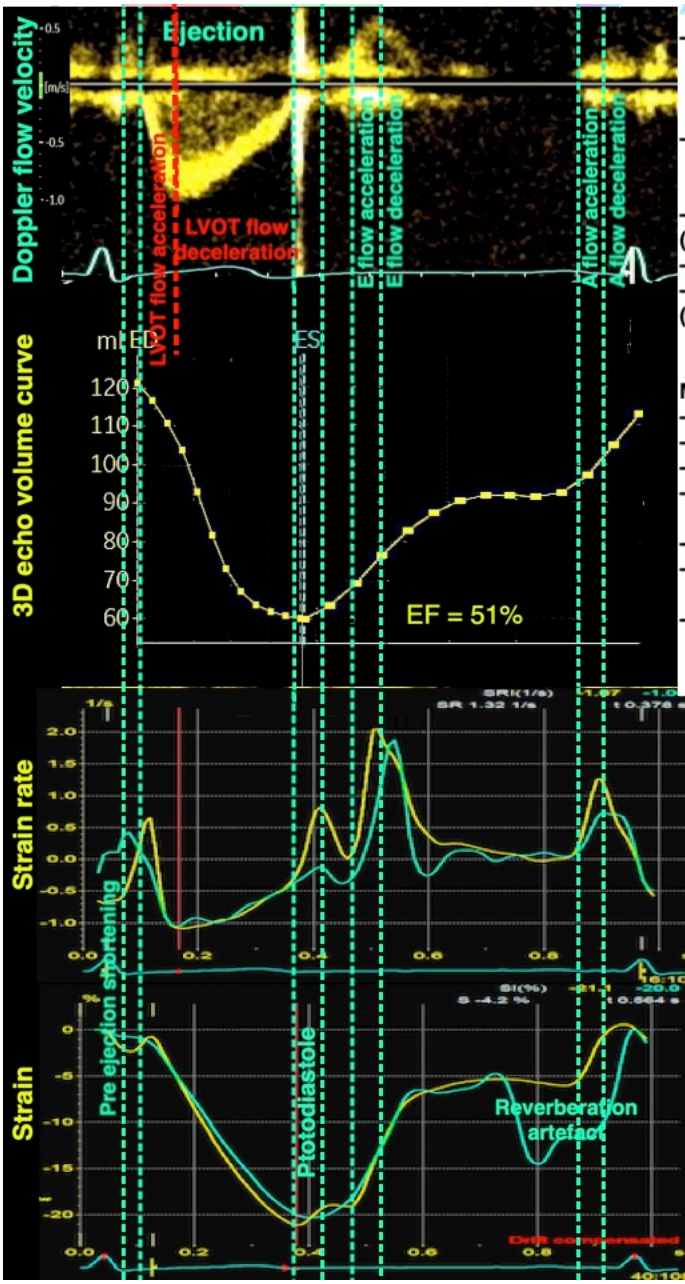
- Annular velocity is the temporal derivative of displacement
- Annular velocity is thus related to flow velocity

- Annular velocities are thusload dependent
(tension versus load) and are thus related to the flow velocities



Mitral annular motiojn
(velocity and
displacement)
encompasses the total
length changes of the
LV in absolute values
during the heart cycles

Vs longitudinal strain and strain rate



Atrial relaxation

- Blood is accelerated by pressure gradients
  accelerates along a gradient
  decelerates against a gradient

- Blood flow = the rate of volume change

- Blood flow is the first derivative of volume
(and, given constant orifices and compliance; flow velocity)
- Acceleration is the second derivative of volume
- Acceleration force is the pressure gradients
(tension versus load)

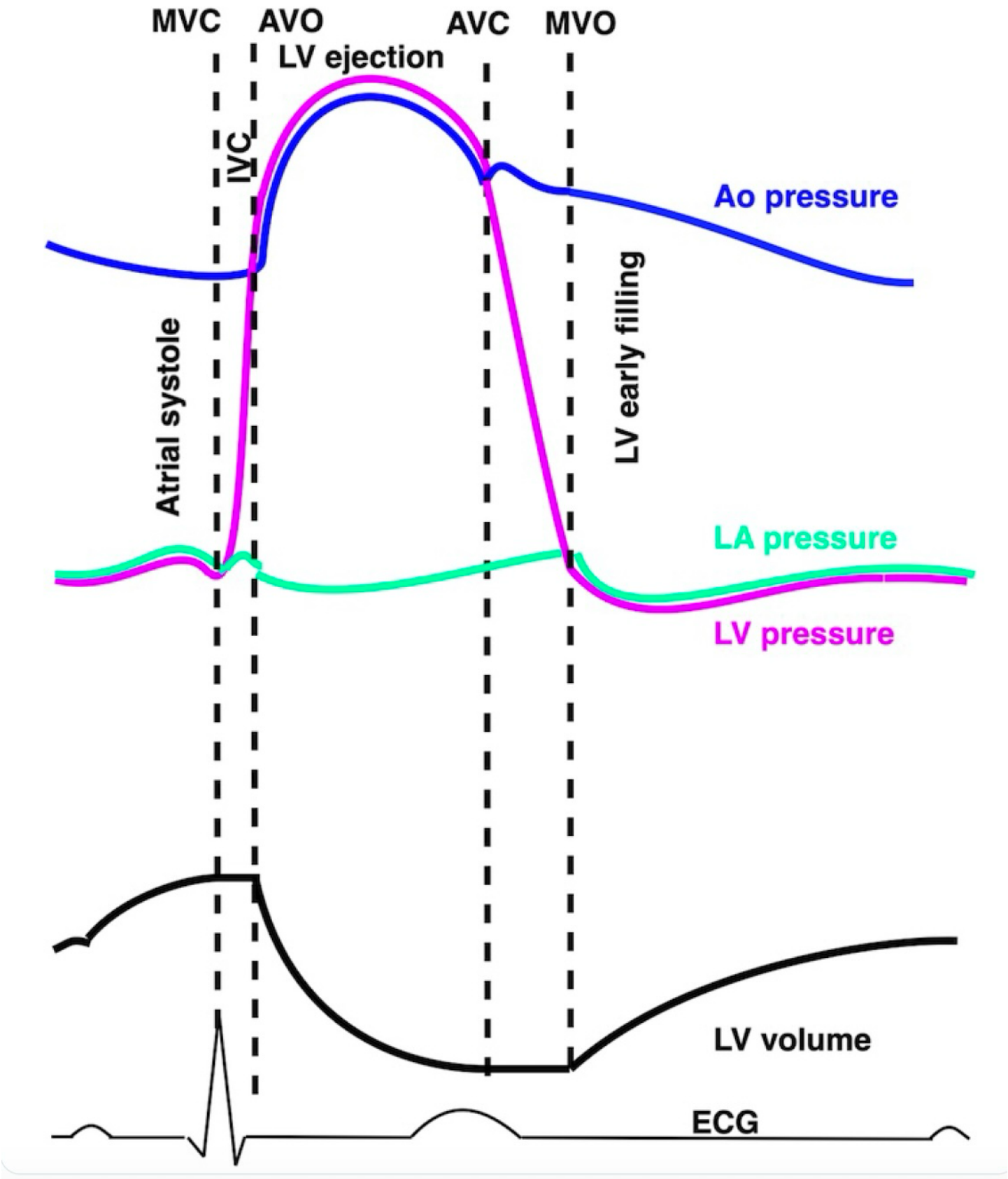
Myocardial deformation is related to volume changes
- Global myocardial strain is the relative LV length change
- End systolic GLS does not correlate with SV
- End systolic GLS correlates with EF (relative SV)
- Strain is thus related to volume changes

- Global SR is the temporal derivative of strain
- Global SR is thus related to flow velocity

- GLS and SR are load dependent (tension versus load)

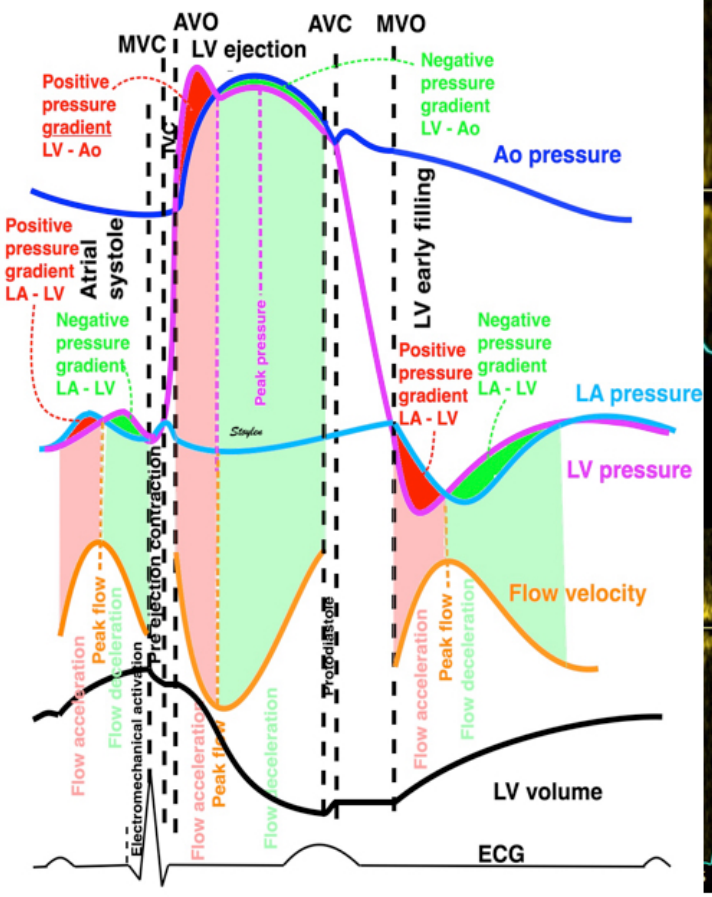
GLS and SR are relative longitudinal LV (or wall) length changes during the heart cycle

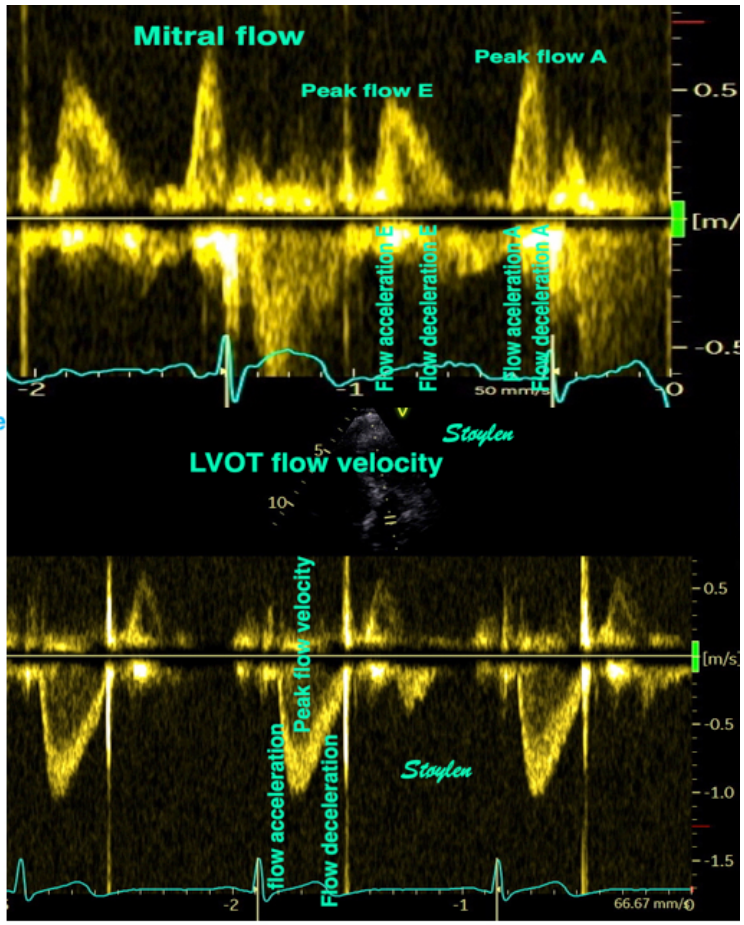
Volume vs pressure



Conventional representation of the LV pressure diagram (Wiggers cycle)

But:





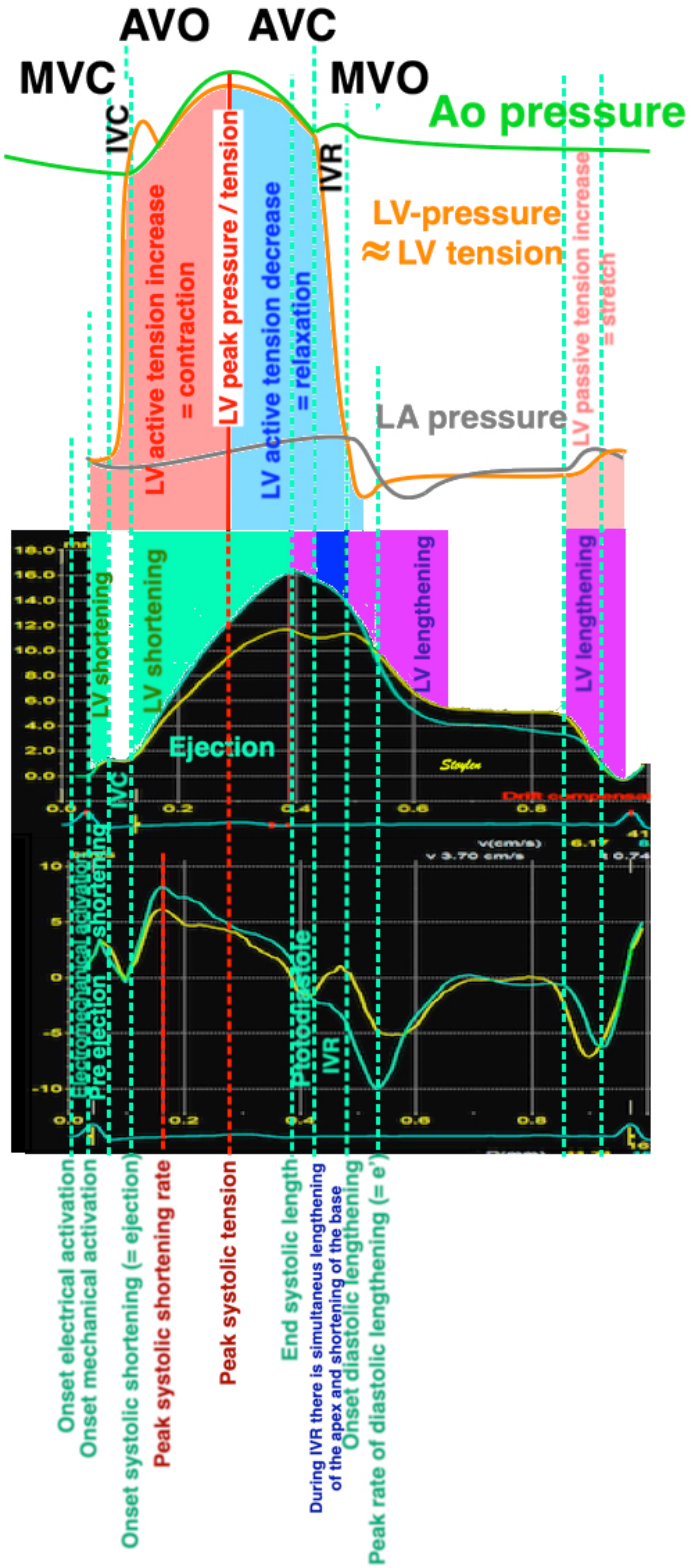
In pulsatile flow, blood is:

- accelerated by a positive pressure gradient and flows with the gradient

- decelerated by a negative pressure gradient and flows against the gradient

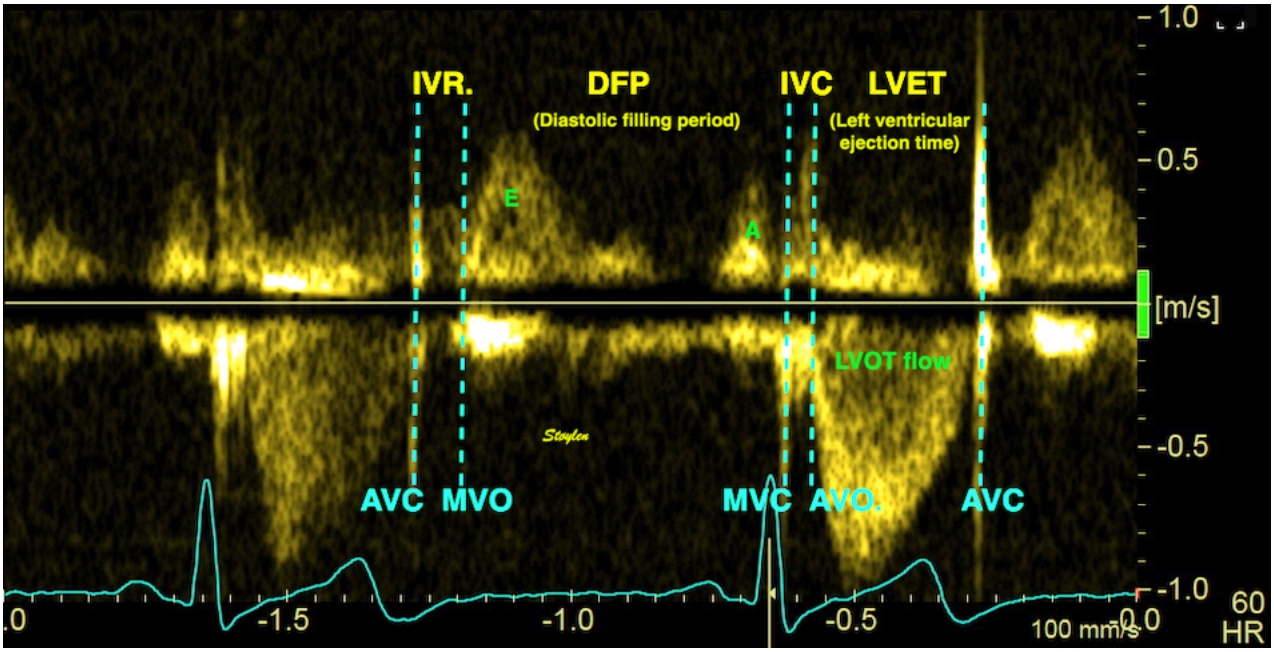
- peak velocity is near pressure crossover

And:

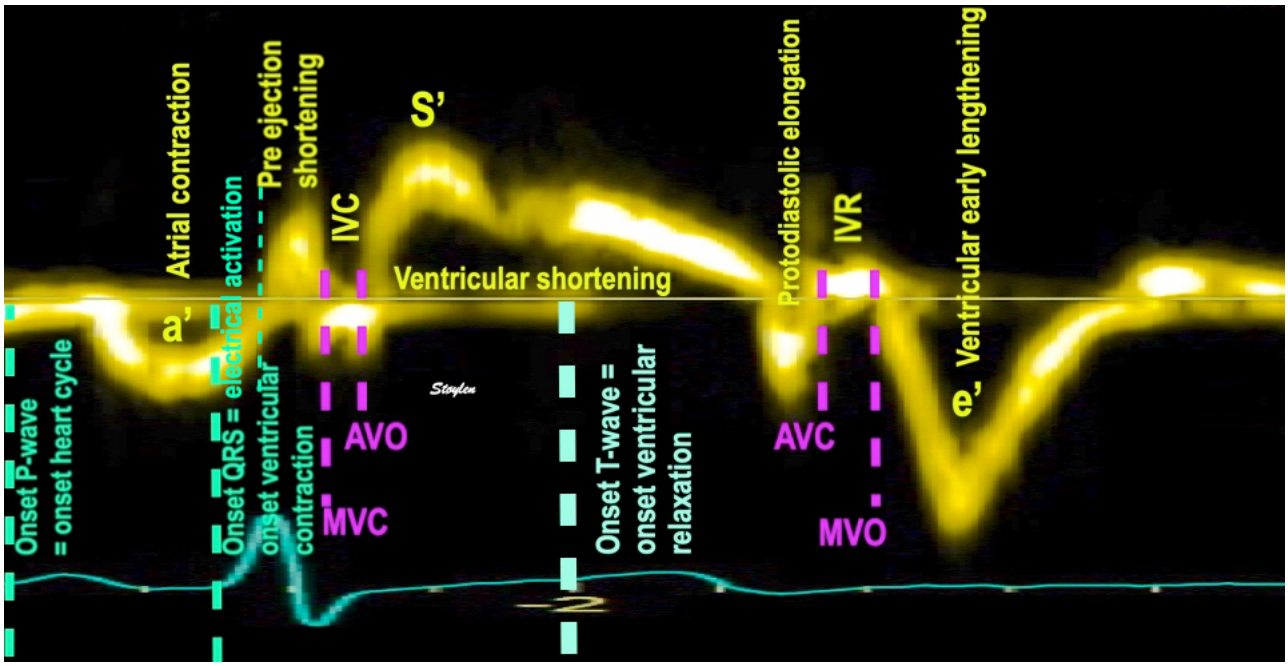


LV contraction does not mean shortening, but tension increase, LV relaxation does not mean lengthening, but tension decrease. Length and tension changes are not simultaneous through the heart cycle.

The heart cycle:

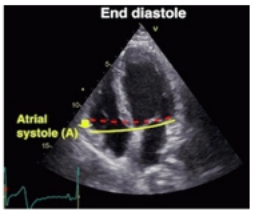


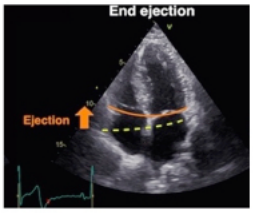
Pulsed wave Doppler with sample volume positioned between the aortic and mitral ostia. Both start and stop of the flows, as well as the valve closure clicks are seen, dividing the heart cycle into the four main phases, ejection, diastolic filling, and between them the isovolumic contraction and relaxation phases..

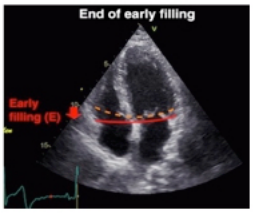


The heart cycle shown by spectral tissue Doppler from the septal base, depicting how the heart cycle in reality starts with the atrial activation (P), and thus the A or a' wave.

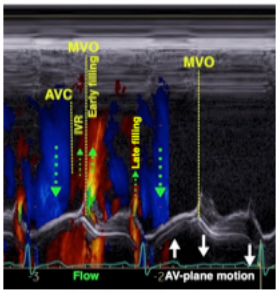
Late LV filling



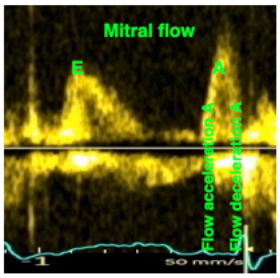


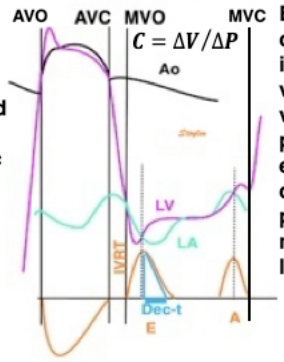


Late filling starts with atrial systole. It is often depicted as the end of a heart cycle, but as the heart cycle starts with the P wave, this is in reality the onset of the next heart cycle. However, it is intimately connected with the previous cycle. During late filling, there is, there is a new phase of basal AV plane motion, expanding the LV. However, the positive early gradient is generated by LV pressure drop (neg. compliance) due to elastic recoil forces, the late gradient is generated by atrial pressure increase due to contraction (pos. compliance, i.e. LV late expansion is pressure driven.

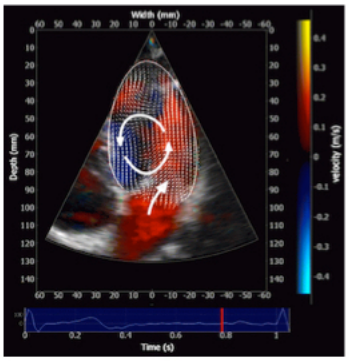


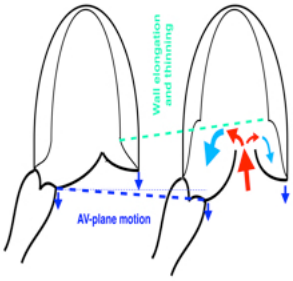
Late flow (A-wave) is accelerated by positive pressure gradient, which reverses by increasing LV pressure due to LV filling, and LA pressure drop due to relaxation.

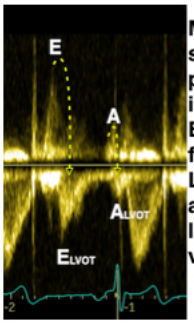




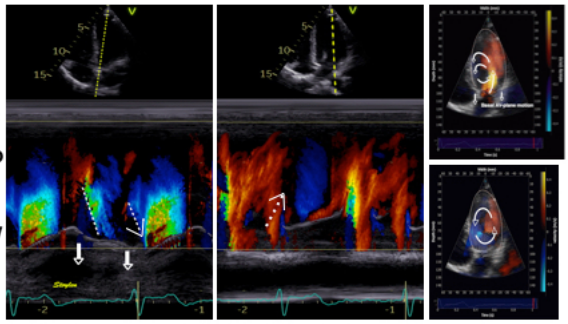
Early filling creates an intra-ventricular vortex, that persists and expands in diastasis, preserving momentum for late inflow.



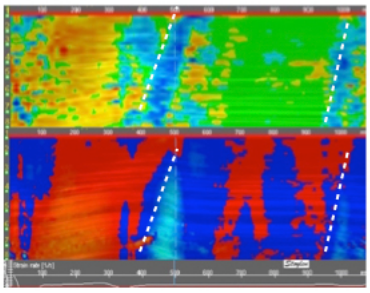




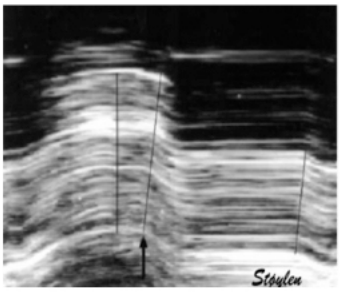
Mitral inflow and simultaneous basal AV-plane motion, deflects inflow into LVOT, as in the E-wave, so there is basal flow along the septum into LVOT, and apical flow along the middle and lateral wall, creating a new vortex.



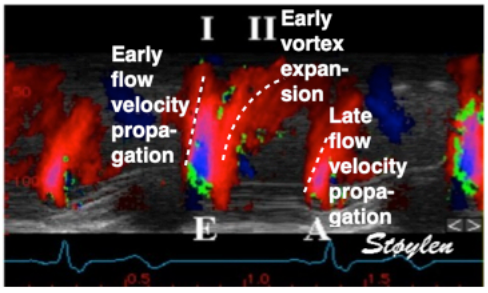
The vortex fuses with the vortex from diastasis, preserving the momentum along septum into the LVOT for the subsequent ejection.

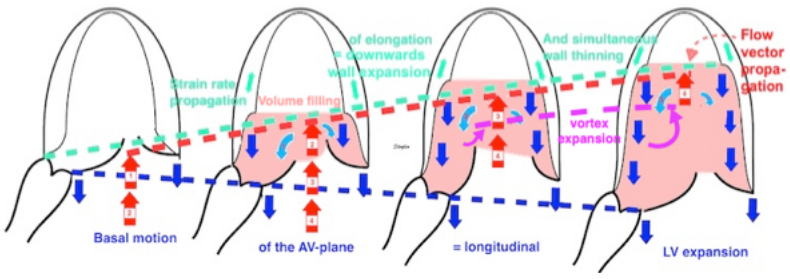


As in early filling, deformation propagates from base to apex. The wave can be imaged by onset of motion, velocity, acceleration or strain rate. It's also called the "atrial kick"



The expansion wave in late filling is driven by the pressure wave from atrial contraction, and is thus pulse wave propagation, analogous to the arterial pulse wave propagation. This in contrast to the early filling wave, which is a negative pressure wave of elastic recoil. Both waves, however are wall elongation and thinning, creating a volume expansion that must be filled by flow.





Flow velocity propagation is the propagation of velocity vectors. As flow is deflected to fill the increasing ventricular volume flow from behind replaces the apical flow, so it is the velocities that follow the pulse wave propagation, not the volume per se.

Annichen Søyland DaaeAS, Wigen MS, Fadnes S, Løvstakken L, Støylen A. Intraventricular Vector Flow Imaging with Blood Speckle Tracking in Adults: Feasibility, Normal Physiology and Mechanisms in Healthy Volunteers, Ultrasound in Medicine & Biology, 2021, https://doi.org/10.1016/j.ultrasmedbio.2021.08.021.

Stoylen A, Slordahl S, Skjelvan GK, Heimdal A, Skjaerpe T. Strain Rate Imaging in Normal and Reduced Diastolic Function: Comparison with Pulsed Doppler Tissue Imaging of the Mitral Annulus. J Am Soc Echocardiogr 2001, 14:264-274.

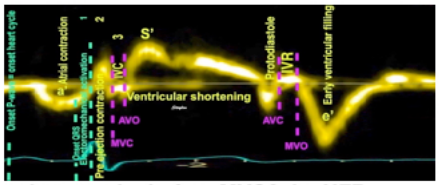
LV pre ejection

Pre ejection consists of:

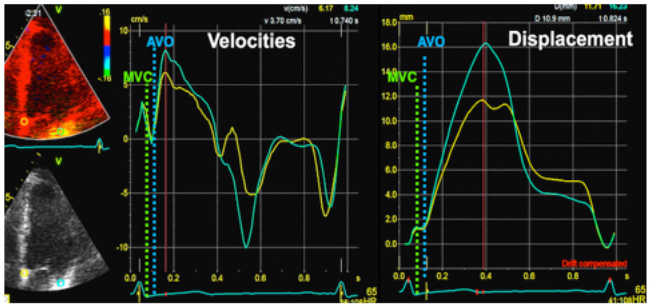
1: Electromechanical activation - conduction of activation from AV-node to initial activation occuring in mid septum and mid lateral wall, ca 15 ms after onset of ECG[1] and excitation - contraction coupling of 20-30 ms[2].

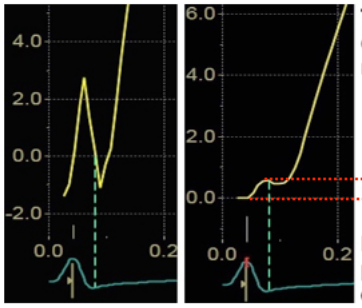
2: Initial (pre ejection) contraction in the lateral wall and septum - before MVC[3].

3: Isovolumic contraction - from MVC to AVO, increasing tension / pressure, without deformation, ending with AVO.

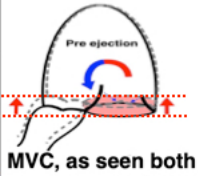


The pre ejection spike has previously been assumed to be isovolumetric, but has been shown to be before MVC[3], by HFR ultrasound very close to the peak of the spike[4], while experimental data suggests that the MVC is the mechanism for terminating the spike[5]. pre ejection motion is abruptly terminated by the MVC itself , and thus both the magnitude and duration of both pre ejection velocity and acceleration is determined by timing of MVC, not contractility. Thus, the height of the spike is determined by the duration of the protosystolic interval, not the peak rate of shortening. It has also been suggested to be a recoil from the atrial contraction, but has been demonstrated both in AV-block and atrial fibrillation[4]. already in 1973 ventriculoatrial crossover was demonstrated to occur ca 40 ms before MVC[6], demonstrating that pre ejection is the onset of tension buildup[6], i.e. that there is active contraction.

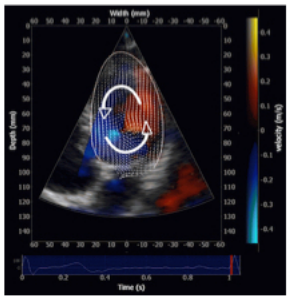




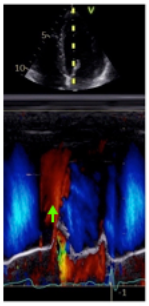
The contraction also causes a small volume reduction of the LV before

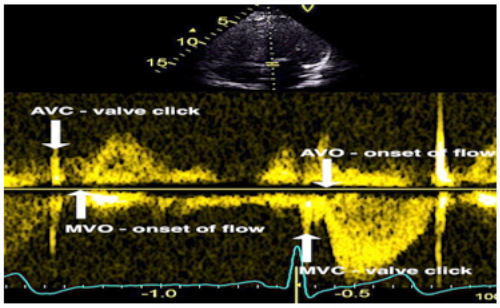


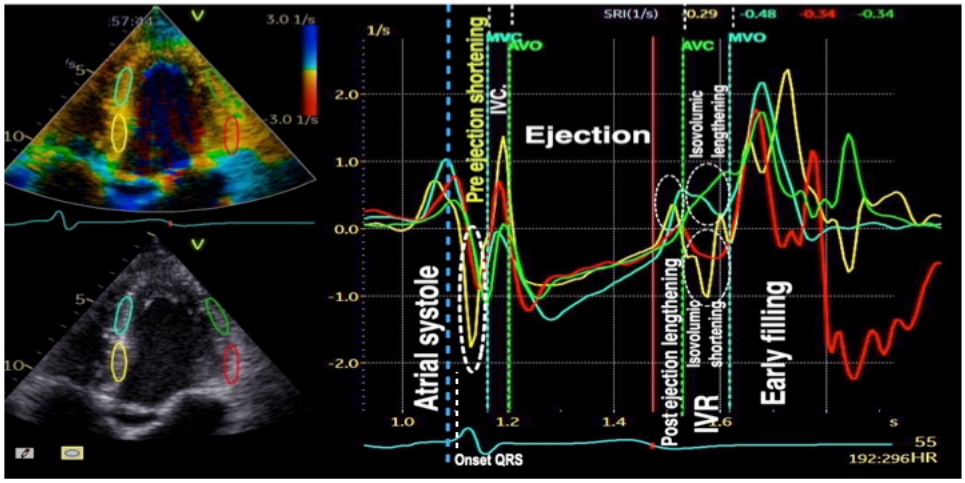
MVC, as seen both by AV-plane motion[3] and experimentally[5]



During pre ejection, the vortex from late filling persists, with basal flow along septum, hitting the anterior mitral leaflet, contributing to the MVC, although annular motion after flow has ended will also contribute, especially for the posterior leaflet.







Strain rate imaging also demonstrating wall shortening both in septum and lateral, both apex and base in pre ejection.

Thus

1: Pre ejection velocity is true ventricular contraction, <u>not</u> atrial recoil

2: Pre ejection contraction is the true LV electromechanical activation timepoint.

3: Peak pre ejection velocity or acceleration is <u>not</u> an LV contractility measure

4: Pre ejection velocity occurs before MVC, and is <u>not</u> isovolumic.

1 Endocardial mapping in humans in sinus rhythm with normal left ventricles: activation patterns and characteristics of electrograms.Circulation. 1984 Jul;70(1):37-42. Cassidy DM, Vassallo JA, Marchlinski FE, Buxton AE, Untereker WJ, Josephson ME.

2 Cordeiro JM, Greene L, Heilmann C, Antzelevitch D, Antzelevitch C. Transmural heterogeneity of calcium activity and mechanical function in the canine left ventricle.Am J Physiol Heart Circ Physiol. 2004 Apr;286(4):H1471-9.

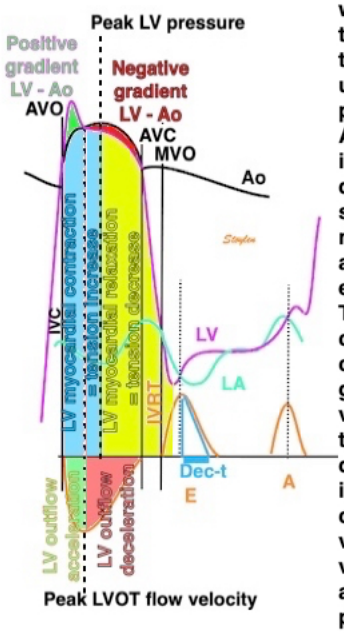
3 Støylen A, Daae AS.Physiological significance of pre- and post-ejection left ventricular tissue velocities and relations to mitral and aortic valve closures. Clin Physiol Funct Imaging. 2021 Sep;41(5):443-451.

4 Brekke B, Nilsen LC, Lund J, Torp H, Bjastad T, Amundsen BH, Stoylen A, Aase SA. Ultra-high Frame Rate Tissue Doppler Imaging.Ultrasound Med Biol. 2014 Jan;40(1):222-31.

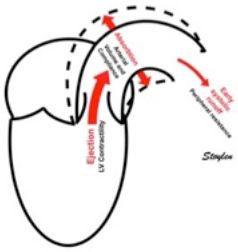
5 Remme EW, Lyseggen E, Helle-Valle T, Opdahl A, Pettersen E, Vartdal T, Ragnarsson A, Ljosland M, Ihlen H, Edvardsen T, Smiseth OA. Mechanisms of preejection and postejection velocity spikes in left ventricular myocardium: interaction between wall deformation and valve events.Circulation. 2008 Jul 22;118(4):373-80.

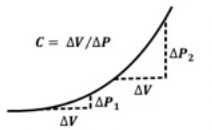
6 Laniado S, Yellin EL, Miller H, Frater RW. Temporal relation of the first heart sound to closure of the mitral valve. Circulation. 1973 May;47(5):1006-14.

LV ejection



After MVC, LV contracts without further deformation, thus no shortening, only tension (pressure) during IVC, until AVO, when LV and AO pressures have equalised. After AVO, blood is ejected into the Ao, causing LV to decrease size (myocardial shortening). This, of course, is not the electromechanical activation, which is the pre ejection contraction. Thedecrease in LV load, causes pressure increase, creating a positive LV - AO gradient accelerating flow velocity. Volume ejected into the aorta causes aortic distension, with pressure increase determined by aortic compliance and the net volume being the ejected volume - runoff volume, which again is determined by the peripheral resistance.

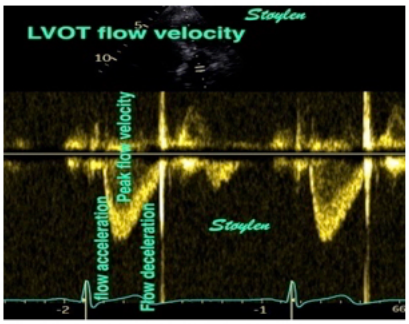




ΔV: Ejected volume – runoff volume relative to arterial volume

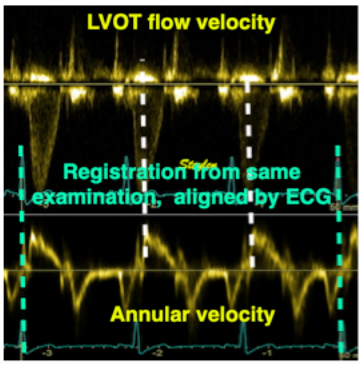
When blood flows out of the LV, as long as LVOT outflow > Ao runoff, increasing Ao distension will cause reduced compliance and increasing pressure.

As myocardial rate of tension increase tapers off, the LV-Ao gradient reverses. The pressure crossover is at peak flow velocity, after this, inertial flow is decelerated, but still continues out of the LVOT until AVC.

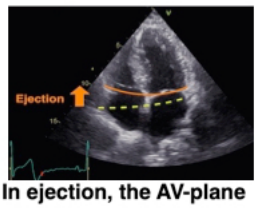


Peak Ao and LV pressure (with negative gradient) is reached when flow is decelerated below arterial runoff, and is later than peak flow. This is the point of peak myocardial tension.

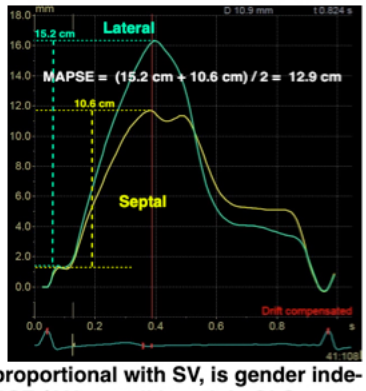
Flow is the volume change rate per time: Q = V / T (l/min), while flow velocity is distance per time v = d / t (m/s). But as d = v x t, and V = A x d, which is the volume circumscribed by the motion with the velocity v, flow and velocity may be assumed to be proportional, if the area is constant, which is reasonable in ejection. The mitral annular velocity is related to the rate of volume change, peak flow velocity and annular velocity are close, although friction and transverse shortening may be factors.



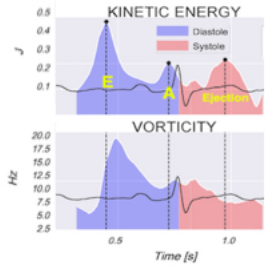
Peak LVOT flow is related to LVOT diameter, and is not a contractility measure[1]. S' is more closely related to contractility, but is both pre- and afterload dependent, is slightly later than peak tension, and is terminated by Ao pressure increase, thus to LV - arterial interaction. Thus, in addition to contractility, S' is dependent on load and arterio-ventricular coupling.



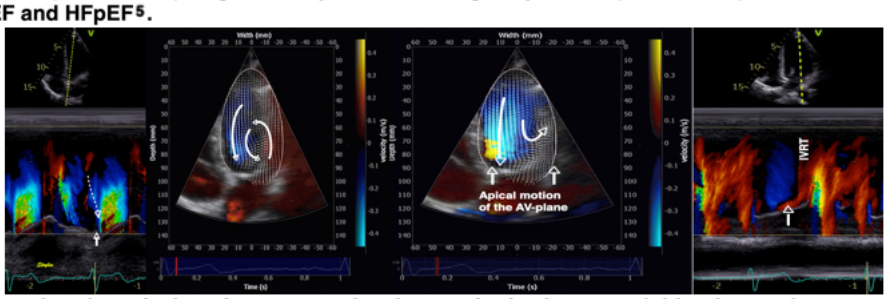
In ejection, the AV-plane moves apically. AV-plane motion is proportional to volume reduction (SV)[2], and the proportion is independent of age and BSA[1 2].



MAPSE is higher laterally than septally, but mean of 2 4 and 6 walls are similar[3]. MAPSE is proportional with SV, is gender independent, but is age dependent (confounder)[3]. It is reduced both in HFreF and HFpEF[4]. MAPSE / LV or wall length is GLS, which s numerically inversely related to BSA (confounder), is gender dependent, and age dependent (confounder)[1]. GLS is not related to SV[3], but reduced in both HFrEF and HFpEF[5].



During pre ejection, there is a persistent vortex, conserving momentum towards the LVOT before ejection.



During ejection,m the vortex disappears, but the apical motion, causes the closes mitral valve to push blood towards apex. This does not create a new vortex, only initiating a uniform blood motion towards apex, that is continued by the isovolumic relaxation[6].

1 Støylen A, Dalen H, Molmen HE.Left ventricular longitudinal shortening: relation to stroke volume and ejection fraction in ageing, blood pressure, body size and gender in the HUNT3 study. Open Heart. 2020 Sep;7(2):e001243.

2 Carlsson M, Ugander M, Mosén H, Buhre T, Arheden H.Atrioventricular plane displacement is the major contributor to left ventricular pumping in healthy adults, athletes, and patients with dilated cardiomyopathy. Am J Physiol Heart Circ Physiol. 2007 Mar;292(3):H1452-9.

3 Støylen A, Molmen HE, Dalen H. Relation between Mitral Annular Plane Systolic Excursion and Global longitudinal strain in normal subjects: The HUNT study. Echocardiography. 2018;35:603-610

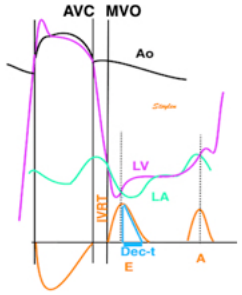
4 Willenheimer R. Cline C. Erhardt L. Israelsson B. Left ventricular atrioventricular olane disolacement: an echocardioaranhic techniaue for ranid assessment of proanosis in heart failure. Heart 1997:78:230-36

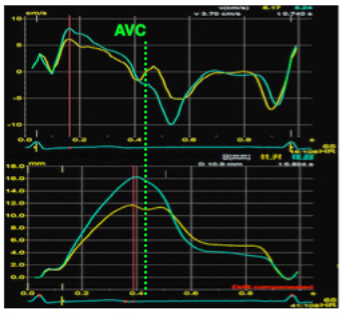
5 Cho GY, Marwick TH, Kim HS, Kim MK, Hong KS, Oh DJ. Global 2-dimensional strain as a new prognosticator in patients with heart failure. J Am Coll Cardiol. 2009 Aug 11;54(7):618-24.

6 Annichen Soyland DaaeAS, Wigen MS, FadnesS, Lovstakken L, Stoylen A. Intraventricular Vector Flow Imaging with Blood Speckle Tracking in Adults: Feasibility, Normal Physiology and Mechanisms in Healthy Volunteers, Ultrasound in Medicine & Biology, 2021, https://doi.org/10.1016/j.ultrasmedbio.2021.08.021.

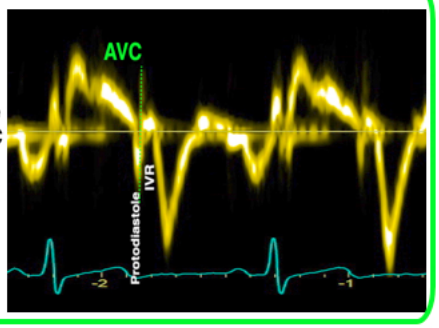
Protodiastole

Protodiastole was first decribed by Wiggers[1]. During ejection, relaxation (tension devolution) starts during ejection at peak pressure, there is simultaneous pressure and tension decline, as well as flow deceleration. Protodiastole ends at AVC. A small LV volume increase has been shown to occur before AVC[2].

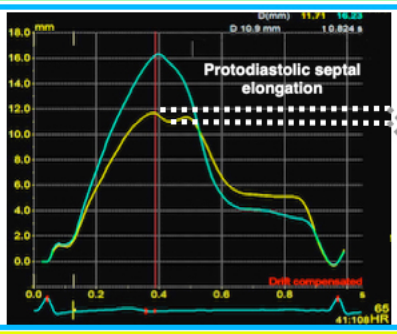


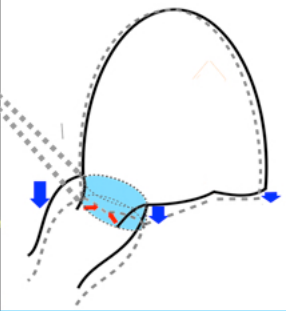


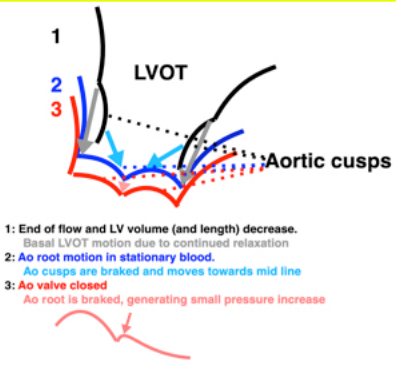
With tissue Doppler, a negative velocity spike can be demonstrated in the septum, and the AVC has been shown to be at the END of this spike, which thus is protodiastolic[2, 3, 4], not isovolumic.

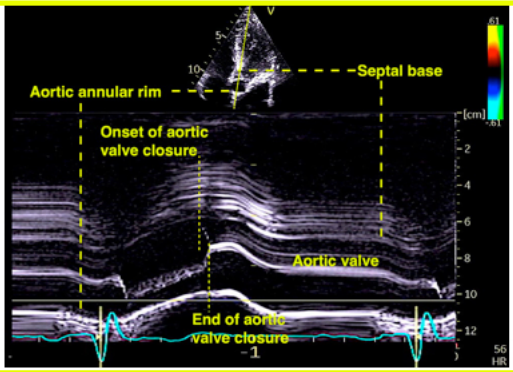


Thus, we see a protodiastolic elongation of the septum at end ejection. The mechanism is that relaxation has already started. As long as there is flow out of LVOT, the LV (including the septum) shortens. When flow ends, the relaxation will induce initial elongation, and if the Ao is open, there is little resistance to elongation, while the closed MV offers considerable resistance, so there is little or no elongation of the lateral wall. This protodiastolic motion has been shown[4], and this will give a small LV volume increase by engulfing an aortic volume without flow, moving in a stationary blood column.









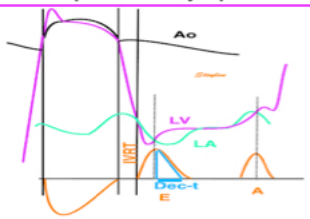
When the aortic ostium moves in a stationary blood mcolumn, tha aortic cusps will be braked and forced towards the middle. Thus, the protodiastole is the time of the closure of the aortic valve. The small pressure increase after the dicrotic notch, is consistent with the residual motion of the aortic root after valve closure.

1 CJ Wiggers. Studies on the consecutive phases of the cardiac cycle American Journal of Physiology--Legacy ..., 1921 - Am Physiological Soc
2 Remme EW, Lyseggen E, Helle-Valle T, Opdahl A, Pettersen E, Vartdal T, Ragnarsson A, Ljosland M, Ihlen H, Edvardsen T, Smiseth OA. Mechanisms of preejection and postejection velocity spikes in left ventricular myocardium: interaction between wall deformation and valve events.Circulation. 2008 Jul 22;118(4):373-80.
3 Aase SA, Torp H, Støylen A. Aortic valve closure: relation to tissue velocities by Doppler and speckle tracking in normal subjects. Eur J Echocardiogr. 2008 Jul;9(4):555-9.
4 Støylen A, Daae AS.Physiological significance of pre- and post-ejection left ventricular tissue velocities and relations to mitral and aortic valve closures. Clin Physiol Funct Imaging. 2021 Sep;41(5):443-451.

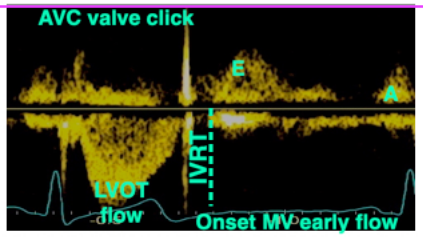
https://stoylen.folk.ntnu.no/strainrate/Basic_physiology.html#Protodiastole

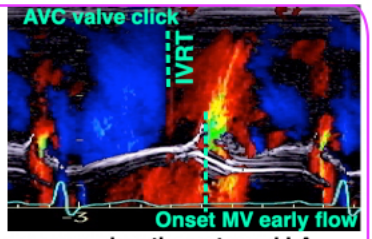
Isovolumic relaxation (IVR)

IVR it the period of LV pressure drop from end ejection at AVC to early filling onset at MVO. In terms of the myocardium, this is relaxation (devolution of active tension), by dissolution of myosin-actin bindings, mediated by active, (energy demanding) calcium transport from cytoplasm to the Sarcoplasmatic reticulum.



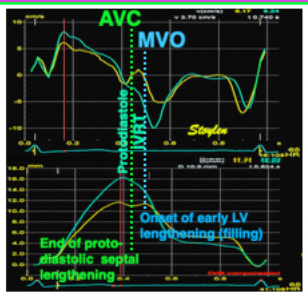
LV pressure = myocardial tension. During IVR, there is reduction of active tension, but with no volume change, no release of passive (elastic) tension. The releaxation rate can be described by the time constant for the pressure drop, tau.

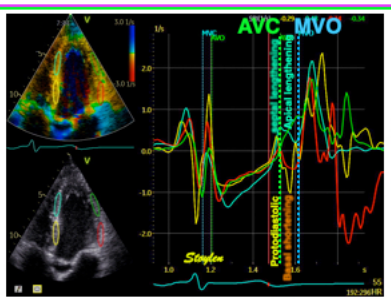


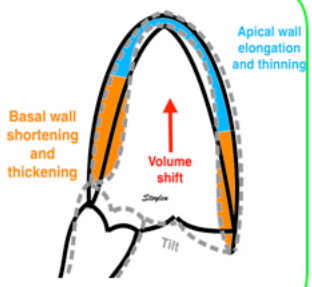


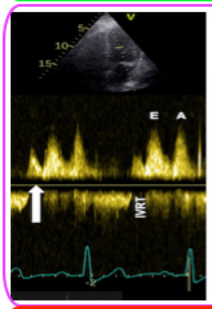
IVRT is dependent on the LV end systolic pressure, relaxation rate and LA pressure. As the IVRT& is defined by AVC and MVO, Doppler flow is the gold standard for IVRT, and in flow recordings, both valve click of AVC and onset of MV flow can be visualised simultaneously in some views.

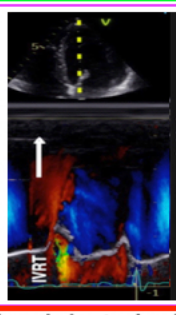
During IVR, the apical part of LV stretches, the basal part shortens[1]. This is not basal contraction, as both parts relax - reducing tension, but uneven tension decline results in shortening / elongation of interacting segments, giving a volume shift = flow from base to apex, and with more shortening of septum also showing a lateral tilt[1].

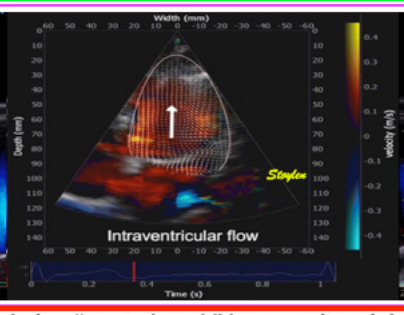


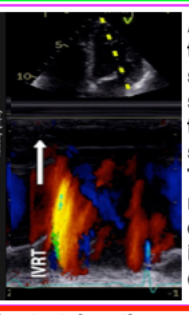










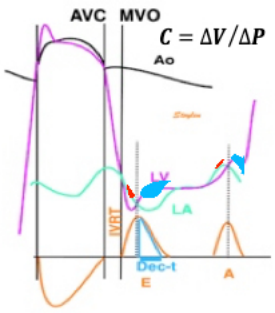


Apically directed blood flow, across the whole width of the LV has been shown both by Doppler[2] and blood speckle imaging[3]. The IVR flow is thus giving a flow momentum for the subsequent early filling.
The basal shortening is thus relaxation, not contraction, and the energy creating the apical gradient, is the relaxation, meaning apical dominance.

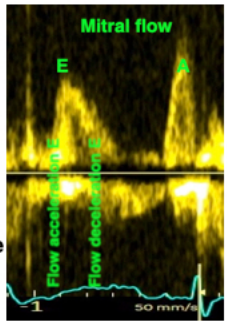
The concept of the basal shortening being "wasted work" because it mainly stretches the apex, is non sensical, firstly, because it isn't work, being relaxation - and the main energy input is from the apex, and secondly because it isnt "wasted", generating a momentum for early filling.

1 Støylen A, Daae AS.Physiological significance of pre- and post-ejection left ventricular tissue velocities and relations to mitral and aortic valve closures. Clin Physiol Funct Imaging. 2021 Sep;41(5):443-451.
2 Sasson Z, Hatle L, Appleton CP, Jewett M, Alderman EL, Popp RL.Intraventricular flow during isovolumic relaxation: description and characterization by Doppler echocardiography. J Am Coll Cardiol. 1987 Sep;10(3):539-46.
3 Annichen Søyland DaaeAS, Wigen MS, Fadnes S, Løvstakken L, Støylen A. Intraventricular Vector Flow Imaging with Blood Speckle Tracking in Adults: Feasibility, Normal Physiology and Mechanisms in Healthy Volunteers, Ultrasound in Medicine & Biology, 2021, https://doi.org/10.1016/j.ultrasmedbio.2021.08.021.

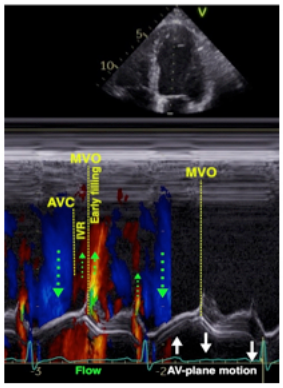
Early LV filling



At MVO, LV pressure continues the drop from IVR, marking the end of relaxation. The volume restriction is eliminated, and the LV can fill (increase in size), consequently, there is pressure drop and volume increase, so there is negative compliance in this phase.



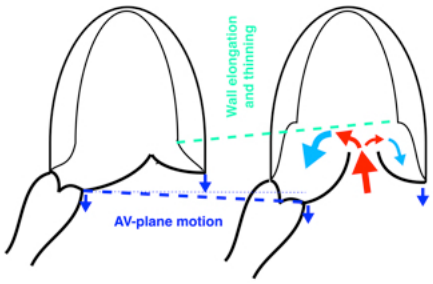
MVO thus marks both onset of early inflow and volume expansion, seen by the basal AV-plane motion[1], as both are manifestations of filling. Intraventricular apical flow is started before MVO[2], transmitral flow starts at MVO.

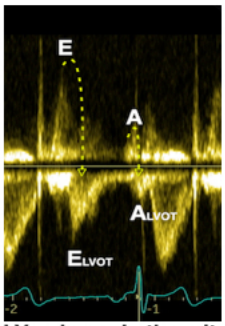


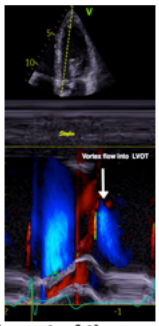
Positive pressure gradient LA - LV

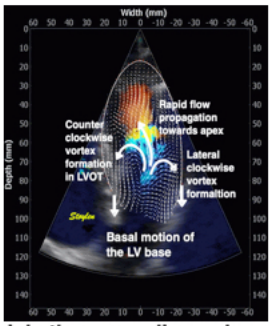
Negative pressure gradient LA - LV

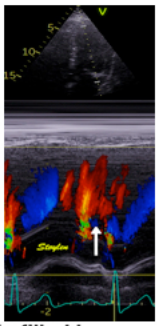
Flow velocity is a function of pressure gradients, accelerated by pos., decelerated by neg. gradients.



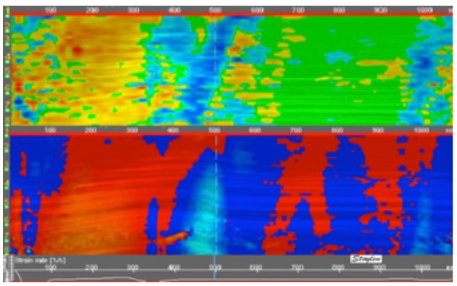




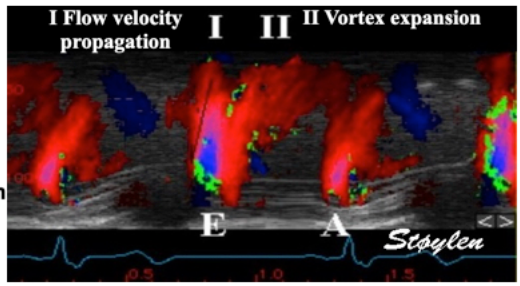




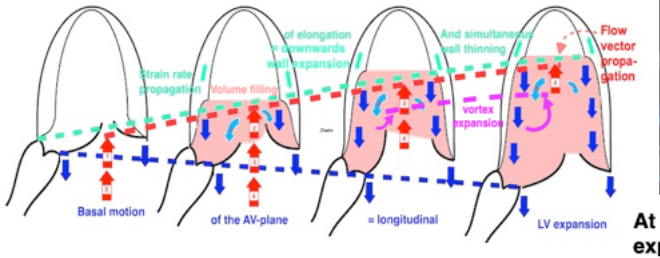
The basal motion of the AV plane expands the LV volume. In the mitraL part of the ventricle the expanding volume is filled by inflow, but behind the closed AV, there is created a void that must be filled by deflection of the inflow, initiating a vortex that originates at the base, not by the deflection of flow by the apex[2]. This can be seen both by pwDoppler, cDoppler and blood speckle tracking.

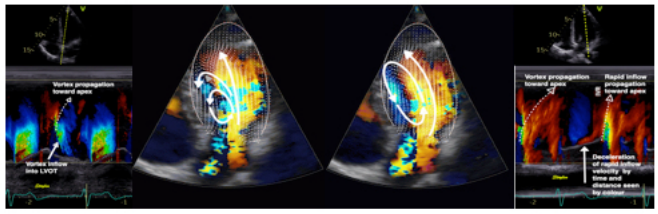


LV wall deformation during early filling can be seen to start at the base and propagate towards the apex, both by colur tissue Doppler and strainrate imaging[3]. This is a necessity, as the expansion must be filled by inflow, which likewise can be seen to propagate from base to apex during early filling (phase 1 of E wave).



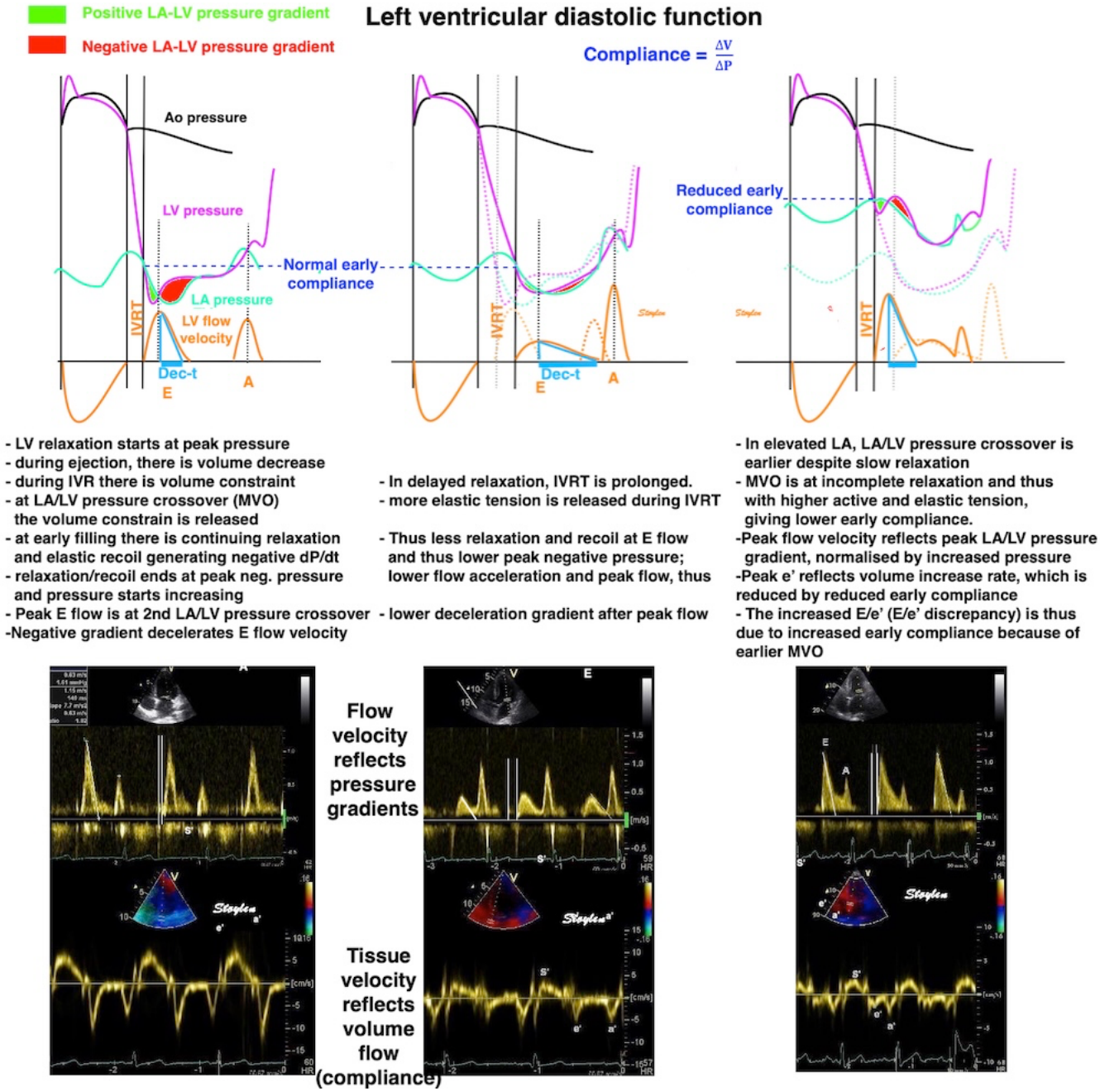
The elongation during early filling generates negative pressure, meaning that much of it is dependent on recoil, as neither relaxation alone, nor the negative pressure can expand the ventricle. Thus, the deformation wave is dependent on the inflow (filling), inflowing blood will fill the expanding space created by the combined elongation and wall thinning, while the blood in the inflowing column will be substituted with later arriving blood. This process will then repeat with the later inflowing blood, filling the cavity expansion moving towards the apex, and later arriving blood is replacing the inflow column. Thus, the flow propagation (I) is the velocity vectors that propagate, while the blood column itself is replaced during filling, and the flow velocity vectors from behind propagate to the wave front. Thus, in the normal physiology, the strain rate propagation and flow velocity propagatin will match[4].





At the same time, and extending into diastasis, the initial vortex will expand, meaning it proceeds more slowly towards the apex[2] (II).

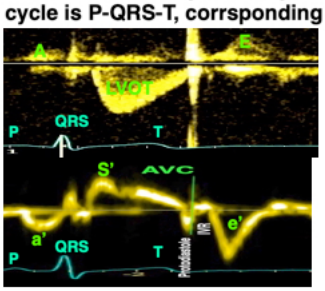
1 Støylen A, Daae AS.Physiological significance of pre- and post-ejection left ventricular tissue velocities and relations to mitral and aortic valve closures. Clin Physiol Funct Imaging. 2021 Sep;41(5):443-451.
2 Annichen Søyland DaaeAS, Wigen MS, Fadnes S, Løvstakken L, Støylen A. Intraventricular Vector Flow Imaging with Blood Speckle Tracking in Adults: Feasibility, Normal Physiology and Mechanisms in Healthy Volunteers, Ultrasound in Medicine & Biology, 2021, https://doi.org/10.1016/j.ultrasmedbio.2021.08.021.
3 Stoylen A, Slordahl S, Skjelvan GK, Heimdal A, Skjaerpe T. Strain Rate Imaging in Normal and Reduced Diastolic Function: Comparison with Pulsed Doppler Tissue Imaging of the Mitral Annulus. J Am Soc Echocardiogr 2001, 14:264-274.
4 Stoylen A, Slordahl S, Skjelvan GK, Heimdal A, Skjaerpe T. Strain Rate Imaging in Normal and Reduced Diastolic Function: Comparison with Pulsed Doppler Tissue Imaging of the Mitral Annulus. J Am Soc Echocardiogr 2001, 14:264-274.



Early diast. velocity (E) is a function of pressure gradient, e' of volume. In delayed relaxation with normal LA pressure, peak flow velocity (E) is reduced, filling starts at hi LV tension, i.e at high early compliance; and thus reduced volume. Thus, E and e' are disconnected E_e' reduced.

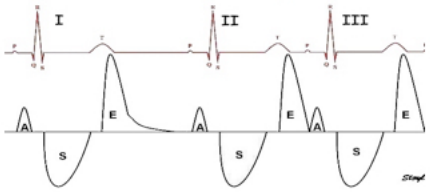
Diastasis

The diastasis is commonly viewed as the interval between early and late filling, which of course is true, but which omits the point that as the heart cycle really starts with the P-wave, the diastasis is in reality the interval between heart cycles, so a single heart cycle is P-QRS-T, corrsponding to a - LVOT - E and a'- S'- e'.

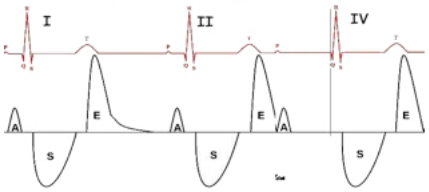


The E - A interval is thus determined by HR (RR-interval), or PQ interval, or both.

Increased HR: shortened RR- (and TP interval)

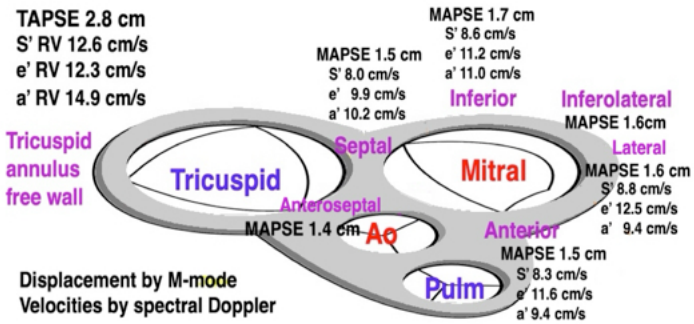


Constant HR (RR-interval), increased PQ interval



This means that the initial mechanical event is the atrial systole, stretching the LV, so it seems that the heart cycle starts with pre stretch, activating both the Frank-Starling mechanism and the passive stretch, thus increasing both active and passive (elastic) tension in ejection.

Mean regional and global AV-plane systolic and diastolic motion in HUNT 3

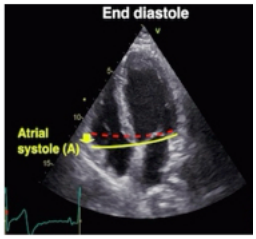


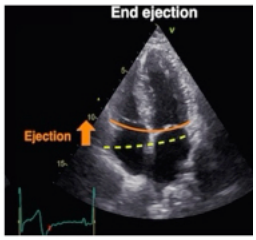
Motion is higher in the tricuspid annulus than the mitral ring

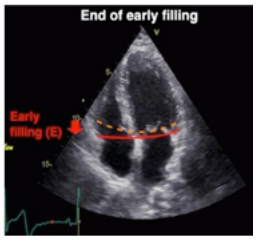
a' is highest inferiorly and septally, lowest lateral and anterior - AV plane tilts towards the right

MAPSE and S' are highest inferiorly and laterally, lowest septally and anteriorly, AV-plane beds in an U-shape, and tilts towards the left in ejection

e' is highest laterally, lowest septally, U shape unbends in early filling (recoil).







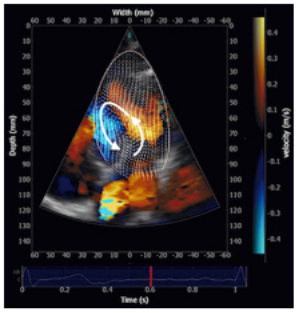
Global LV:

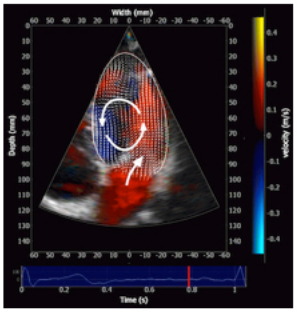
MAPSE:	mean of septal/lateral:	1.56 cm
MAPSE:	mean of septal/anterior/lateral/inferior:	1.58 cm
MAPSE:	mean of septal/anteroseptal/alterior/lateral/inferiolateral/inferior:	1.51 cm
S' :	mean of septal/lateral:	8.4 cm/s
S':	mean of septal/anterior/lateral/inferior:	8,4 cm/s
e':	mean of septal/lateral:	11.2 cm/s
e':	mean of septal/anterior/lateral/inferior:	11.3 cm/s
a:	mean of septal/lateral:	9.7 cm/s
a:	mean of septal/anterior/lateral/inferior:	10.0 cm/s

Global LV correlations:

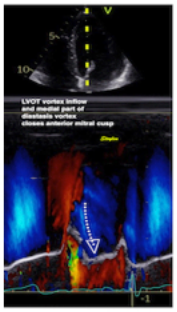
MAPSE with S'	R = 0 .34
MAPSE with e'	R = 0.42
MAPSE with a'	R = 0
S' with e'	R = 0.58
S' with a'	R = 0.18
e' with a'	R = -0.38

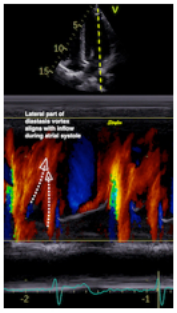
Looking at the AV-plane motion[1], we see that the atrial systole, in addition to moving the AV plane basally and stretching the ventricles, there is tilting of the AVplane towards the right, with more motion in the septal than the lateral mitral ring. The ejection moves the AV-plane apically, compressing the ventricles. In addition, this motion is highest in the RV, and higher in the lateral LV than in the sepotum, thus bending the AV-plane in an U-shape. During early filling, there ios correlation between MAPSE and e' and S' and e', showing a close connection, possibly by e' being recoil of compression form systole. The e' is also highest in the RV and lateral LV, also showing a connection - unbending, possibly also recoil. The a' does not correlate with MAPSE, and only slightly with S', so atrial and ventricular systole are more independent events. The negative correlation between e' and a', however, shows a possible dependence of a' on the preceeding e', possibly by filling volume.





During diastasis, the vortex initiated in early filling expands further, but remains in the LV[2], conserving the momentum from early filling. The vortex goes basally along the septum, contributing to the partial closure of the MV, and apically along the lateral wall, preserving momentum for the late filling during the following atrial systole.





1 Støylen A, Mølmen HE, Dalen H.Regional motion of the AV-plane is related to the cardiac anatomy and deformation of the AV-plane. Data from the HUNT study. Clin Physiol Funct Imaging. 2023 Jul 3. doi: 10.1111/cpf.12845.

2 Annichen Søyland DaaeAS, Wigen MS, Fadnes S, Løvstakken L, Støylen A. Intraventricular Vector Flow Imaging with Blood Speckle Tracking in Adults: Feasibility, Normal Physiology and Mechanisms in Healthy Volunteers, Ultrasound in Medicine & Biology, 2021, https://doi.org/10.1016/j.ultrasmedbio.2021.08.021.

How physiologically correct is this schematic wiggers diagram?