FAQ: Closed Loop Stimulation (CLS)

What does CLS measure?
CLS measures cardiac contractility, which is the heart's mechanism to control Stroke Volume (SV). SV multiplied by the Heart Rate (HR) determines the Cardiac Output (CO), which is the amount of blood the heart pumps in one minute.

Does CLS measure cardiac contractility directly?
No. It uses a local intracardiac impedance measurement at the ventricular lead tip. There is a direct correlation between intracardiac impedance and ventricular contractility. Contractility in turn represents the sympathetic nervous stimulation to the heart.

Why CLS is more specific than other sensors?
CLS does 8 couples of measurements per cardiac cycle. This means in a window of about 250ms it delivers stimuli with a frequency rate of 216Hz. Boston's MV sensor has a frequency rate of 20Hz.

Is the impedance measurement of CLS the same as in minute ventilation?
No, it is different to the minute ventilation system. In minute ventilation impedance is measured between the ring electrode of the lead and a small point at the header. Both have roughly the same size. So here the electrical field lines are distributed very uniformly between the 2 poles. The impedance of the whole thorax is important for the signal. So breathing influences the signal of minute ventilation but not the signal of Closed Loop Stimulation.
Also sampling is very different between CLS and MV algorithm. CLS uses 128Hz while MV 20Hz.

If a patient doesn't make any activity but is stressed how can CLS work out?
It's possible to set some parameters in the algorithm in order to have the better response. For this kind of patient CLS dynamic has to be adjusted.
5 different settings of CLS dynamic are available in the device and it is also possible to set maximum resting rate.

Are there special leads required for CLS to work?
No, the proper function of CLS does not depend on the type or fixation position of the lead. Every unipolar or bipolar lead with passive or active fixation is compatible, as long as the lead is positioned in the right ventricle.

Why doesn't CLS need a specific lead or a specific position?
CLS is an algorithm working with unipolar impedance measurement and measurements are only Ohm's law based. It measures relative changes in impedance and is not aware of lead placement. While apical placement is highlighted for illustrative purposes, outflow tract and septal wall placement actually suggest better performance according to a sub-analysis of clinical data.
You may also use any RV lead with CLS: active, passive, bipolar or unipolar.
CLS will work with any RV lead in any RV location.

How can be managed a patient with total AV block?
As for HF patients this issue can be solved programming one of the parameters of CLS: Vp required ON.

What about CLS in combination with beta-blockers?
CLS uses a rolling average of the previous 256 resting curves and therefore can quickly and effectively react to changes in the contraction dynamics. Beta-blockers exert an influence on basic contractility and consequently update the reference curve, delivering optimal rate modulation.

If a patient changes β-blocker therapy or suffers from other cardiac pathologies (AMI or dilated cardiomyopathy) driving in different contractility, how can CLS respond?
CLS is a dynamic sensor that will automatically optimize to drug induced changes in the patient's contraction dynamic. At every cardiac cycle CLS updates its curves in order to obtain a trend corresponding to actual situation. This allows the device to adapt the rate to patient's physiological conditions. Initially the paced rate response will be blunted because beta-blockers will weaken the contraction before the reference waveform is completely rebuilt. However, within about a week CLS will fully optimize to the patient's new contraction dynamic.
After establishing a new baseline, CLS will deliver appropriate pacing therapy based on the demands of the autonomic nervous system.

What about changes in basic contractility (e.g. DCM, infections, AMI)?
The reference curve adapts to the changed state in basic contractility and calibrates CLS automatically to suit these circumstances.

My patient receives a beta-blocker to decrease the rate but as a side effect it also reduces contractility. Does CLS work in such cases and why use CLS at all for patients who require decreased rates?
CLS can also be used in cases of reduced contractility due to beta blockers because the system calibrates itself to suit this situation. The beta-blocker is intended to reduce the resting rate. But, when under stress, the patient should not be subjected

to artificially induced chronotropic incompetence. CLS is integrated in the cardiovascular regulatory circuit and therefore allows for natural rate regulation under stress.

How does CLS respond to heart changes, e.g. in the case of acute myocardial infarction (AMI)?
CLS continually adjusts to match contractility and the contraction pattern. In the case of AMI slight modifications of the contraction dynamics can appear and CLS adapts to these changes automatically.

How can CLS respond in an adequate manner to blood pressure changes?
A study was performed comparing blood pressure changes during CLS stimulation and during DDD pacing. The blood pressure profiles of paced and spontaneous beats were comparable. The onset of paced rhythm in DDD-CLS resulted in a less pronounced decrease in heart rate and fall in diastolic pressure than in DDD

My patient engages in athletic activities often and for long periods at a time (marathon runners, lumberjacks, etc.). If CLS is constantly recalibrating, will this not cause the patient to have a rate that is too low after some time, because CLS calibrates itself to sustained activity as a resting value?
The response of CLS to this unique type of patient can be programmed. In this case, increasing the CLS response to high or very high and increasing the maximum Closed Loop rate would ensure a suitable rate spectrum during sustained athletic activities.

How does CLS handle Mode Switching?
CLS mode switches to DDI(R) function during atrial arrhythmias. The rate adaptation during Mode Switching is determined by the capacitive accelerometer. The reason is that the contraction dynamics change unpredictably during atrial arrhythmias, and CLS would take a few minutes to adapt to the new signal during Mode Switching. Upon the resolution of the Mode Switch episode, CLS resumes full rate adaptation.

How does CLS handle fusion beats?
In the algorithm there is a special CLS fusion handling and counter, this counts intrinsically conducted Vs events in the fusion zone with a limit of 10 out of 16 Vs events. If the counter is > 10 CLS is disabled.

Why CLS is a better choice in case of VVS than rate drop algorithms?
One of the mechanisms of vasovagal syncope is an increase in ventricular contraction and consequent reaction of nervous system (increase of vagal tone and decrease of sympathetic tone) causing bradycardia and blood pressure decrease.

CLS prevents these problems integrating in the loop and immediately, after changes in ventricular contraction, increasing heart rate.
A common rate drop algorithm activates after decreasing of heart rate and without preventing syncopal symptoms.

What about CLS in combination with Vasovagal Syncope?
CLS has been proven to be very effective for patients with Vasovagal syncope. Due to the early increase in contractility just before the syncopic event, CLS is able to immediately increase the heart rate, preventing the rate drop, which is usually the cause of the vasovagal syncope.

How can CLS reduce ventricular pacing?
As exists a curve about Vp and a curve about Vs, CLS can work even if the pacemaker is not stimulating. There's a particular feature implemented in CLS to allow spontaneous ventricular activity. The PM automatically extends AV delay and if a stable number of V activity is checked then AV will be prolonged to 300ms, thanks to IRS+ algorithm.

When should "Vp required" be programmed ON?
Only when the patient has developed a high degree AV block, because then there is no need to search for intrinsic rhythms.

My patients are all old and quiet inactive. Are there really any advantages to giving them physiological rate regulation?
Especially inactive patients have a worse prognosis because inactivity is a cardiovascular risk factor as proven in studies. These patients receive a pacemaker to be able to master their daily lives better again. And this is exactly what can be achieved by moderate settings in CLS natural rate regulation.

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The response of CLS to this unique type of patient can be programmed. In this case, increasing the CLS response to high or very high and increasing the maximum Closed Loop rate would ensure a suitable rate spectrum during sustained athletic activities.

Sometimes diabetes results in neuropathies, the destruction of nerve tissue. The autonomic nervous system can be affected by this as well. Is CLS suitable for this type of patient?
CLS can also be used in cases of reduced contractility due to neuropathy because the system calibrates itself to suit this situation.

My patient suffers of dilated cardiomyopathy (DCM). The contraction pattern is entirely different from that of patients without DCM. Does CLS work under these conditions?
CLS can also be used in cases of reduced contractility and altered geometry of the ventricle due to DCM because the system calibrates itself to suit this situation. CLS also evens out changes on the long term.

How can CLS reduce AF burden and how can be helpful for AF patients?
Several minutes prior to onset of atrial fibrillation a dominance of the parasympathetic nervous system has been evidenced, causing bradycardia. The balance between heart rate and contractility was disrupted. CLS detects the inappropriate relationship between contractility and heart rate in order to prevent occurrence of atrial tachyarrhythmia beforehand via targeted rate increase.
Furthermore, CLS integrates in a physiological way into the autonomic system. In this way it can influence the mechanisms at the basis of AF onset. It provides, with IRS+, a reduction in ventricular stimulation up to 6%, thus ameliorating the entire hemodynamic system.
AF patients have lost chronotropy and are often on medication to slow AV conduction. Consequently, their heart rate is a result of sporadic AV conduction and medication—not very physiologic. CLS will provide a physiologic heart rate based on the demands of the ANS for these patients.

When should "Resting rate control" be adapted?
In case of vasovagal syndrome, the heart will be paced above the heart rate at rest, even when there is no exercise. In this case the Resting Rate Control should be set between + 20 and + 50 bpm.
If the patient is symptomatic and/or is experiencing heart rate increases at rest during postural changes, the Resting Rate Control can be limited < 20 bpm.

When should CLS be disabled?
In principle CLS adapts to all situations. When there are special situations in which CLS cannot work it will be automatically disabled and automatically resumes its functionality after the situation normalizes again.