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ST SEGMENT
THE “UPS” AND THE “DOWNS”

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CONTENTS

ST SEGMENT, THE "UPS" AND "DOWNS"

INTRODUCTION

Myocardial disease has become increasingly common even in developing countries (1, 2). As a health care system matures and patients live longer this population, now at a higher risk of myocardial ischemia, tends to get to theatre (2). This therefore means more *at risk* patients will knock at the theatre door more often than not. Perioperative myocardial injury occurs commonly and is one of the independent predictors of poor outcome after surgery (2).

Perioperative physicians are now required to monitor at risk patients and hopefully prevent the complications related to intraoperative myocardial ischaemia. Prevention of myocardial injury is key, so a test that can indicate impending myocardial injury, rather than a test that tells us when the myocardial injury has occurred, will be beneficial. While a leak of myocardial intracellular proteins confirms myocardial injury this may be rather too late.

In this booklet we shall explore briefly the use of the ST-segment peri-operatively; this may indeed encourage us to use this monitor more often; surely we shall need to look at its use's ups and downs.

ECG RECORDING PHYSICS

Normally an electric impulse of the heart is generated by the SA node. The impulse wave then depolarises both the atria and the ventricles in a well organised and sequential manner followed by repolarisation (1). This impulse is Fourier analysed, filtered, amplified and then shown in the monitor we look at. For further information on this subtopic I shall refer the reader to the Friday Morning presentation by Dr G W Jones on the 22nd January 2014.

ST SEGMENT DEVIATION PATHOPHYSIOLOGY

In perioperative myocardial ischaemia two main mechanisms are noted, Type 1 (acute coronary syndrome) and Type 2 (secondary to demand versus supply imbalances) (2).

In Type 1 perioperative myocardial ischaemia an unstable plaque ruptures. Secondary to the thrombogenicity of the contents of the plague a thrombus is formed which completely obliterates the lumen of the coronary vessel. The resultant complete occlusion causes transmural ischaemia and infarction. The vector of electric forces in this type of infarction is epicardial translating to ST-segment elevation (1, 3).

In Type 2 perioperative myocardial ischaemia increased myocardial oxygen demand with/without decreased supply results in sub-endocardial ischaemia. This is the most common type of perioperative myocardial ischaemia, accounting for 55% of perioperative myocardial ischaemia and occurs commonly within the first 3 days post-operatively (1). Tachycardia is the most common cause of Type 2 perioperative myocardial ischaemia, a heart rate of 80 to 90 beats per minute in a patient with baseline heart rate below 60 beats per minute has been associated with significant ischemia (1).

Decreased oxygen delivery to the heart, e.g. in hypovolemia, bleeding, or systemic vasodilatation, anaemia, hypoxemia, and increased demand state like in hypertension (elevated stress hormones, vasoconstriction) and hypercarbia aggravate ischemia. Stress-induced and ischemia-induced coronary vasoconstriction further impairs coronary perfusion (4).

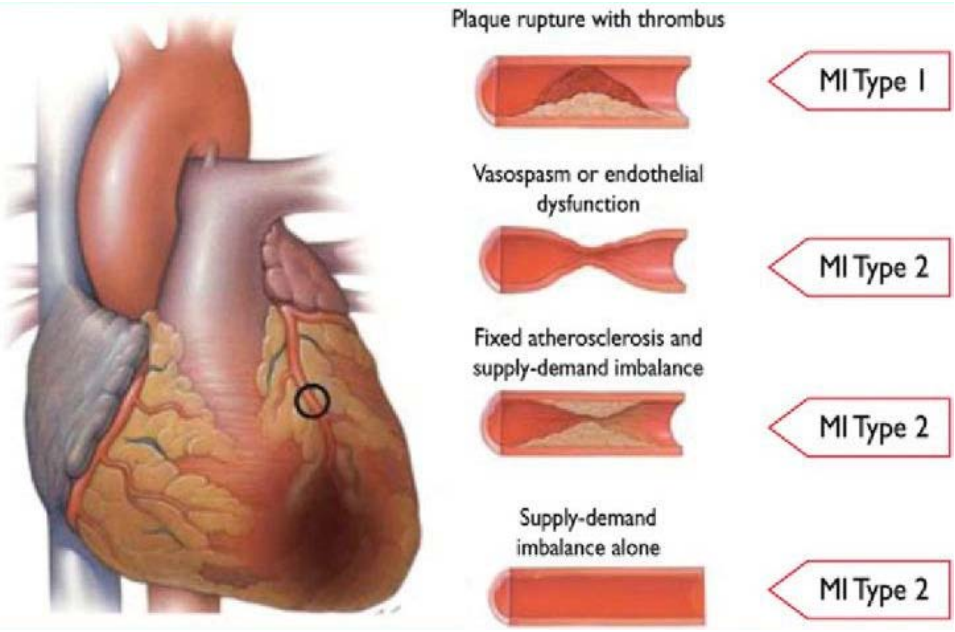

Figure 1

Two theories exist to explain how an ST segment may be depressed in Type 2 myocardial ischemia. These theories are based on the fact that an ischaemic myocardium remains more positive during electrical diastole, has a lower amplitude and a longer duration of an action potential during electrical systole. The above explanation therefore means there is always a potential difference between the ischaemic myocardium and a normal myocardium with resultant electrical currents generated. See Figure 2 below.

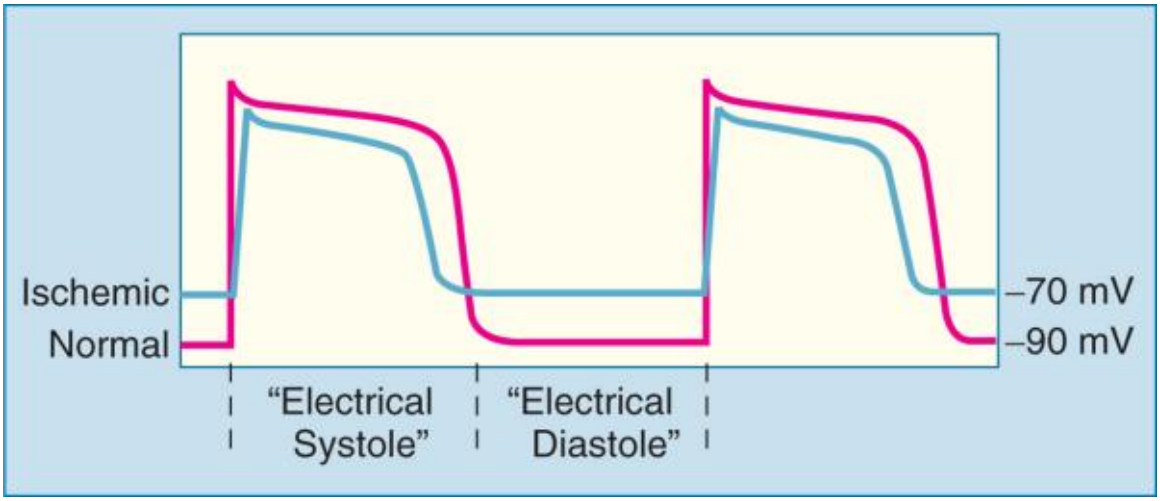

Figure 2

In Theory 1 (systolic injury current) the current flows from a normal myocardium, which is more positive, to the ischaemic myocardium, which is less positive. In Type 2 myocardial ischaemia the ischaemia predominates in the endocardium with resultant net current towards the endocardium, away from the lead directly on top of the myocardium. This results in ST-depression. See Figure 3 below.

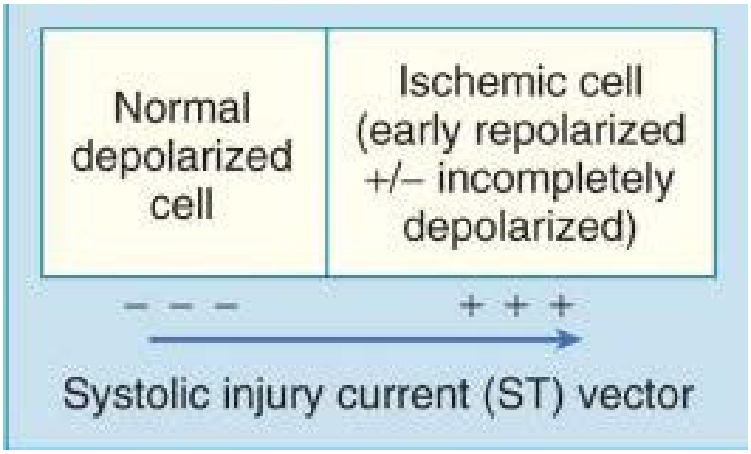

Figure 3

Theory 2 (diastolic injury current), called diastolic current theory, states that an area of localised ischaemia remains with a decreased (more positive) membrane potential which causes an increase in the ECG baseline i.e. the T-Q segment, which represents the electrical diastole. In sub-endocardial ischaemia current moves from the ischaemic myocardium to the normal myocardium which is more negative. This means that the ST-segment does not increase but rather that the baseline of the ECG moves up relative to the ST-segment see Figure 4 below [5].

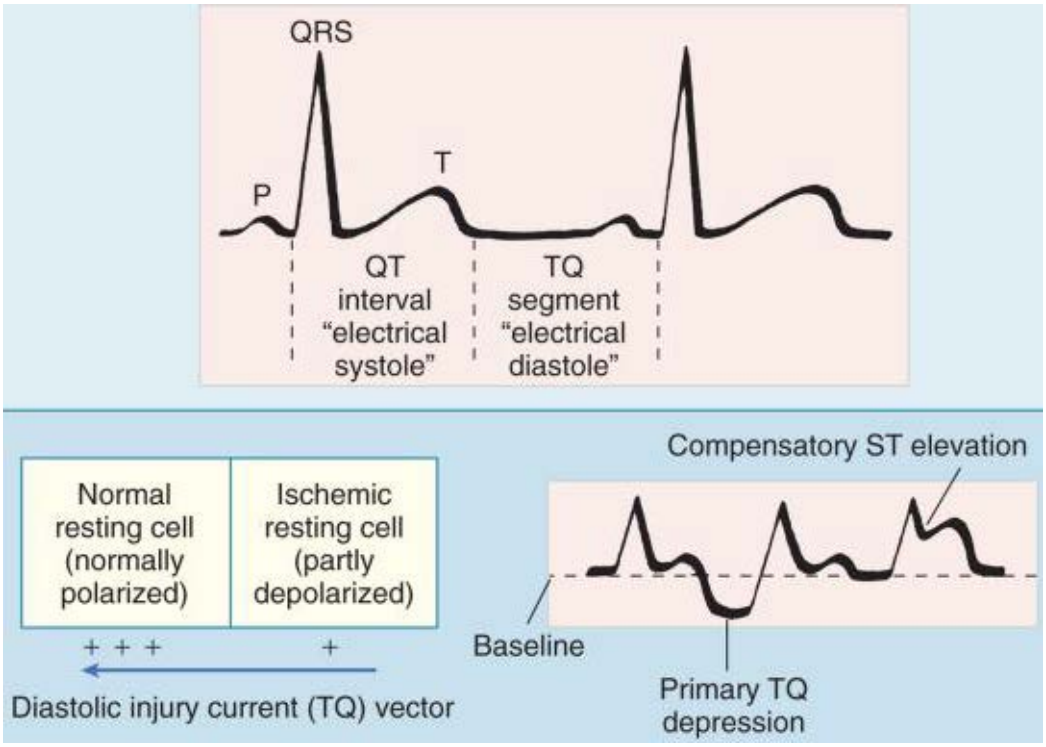

Figure 4

PREVALENCE OF PERI-OPERATIVE MYOCARDIAL ISCHAEMIA

Perioperative myocardial injury has been noted to be a big contributor to 30-day mortality post operatively. If the post-operative period was a disease it would have been the third leading cause of mortality in the United States with half of the deaths being a direct result of cardiovascular complications - mainly perioperative ischemia. The prevalence is higher in females, although males tend to develop ischaemia at a younger age compared to females. The average age of the population presenting to theatre is increasing, and so is the incidence of coronary artery disease amongst the surgical population. Therefore, the incidence of intraoperative myocardial injury, with or without myocardial necrosis, is common and exceeds 2% in the general population and more than 5% in high risk patients (4). Significant advances have been made in understanding peri-operative myocardial injury.

Recent evidence also suggests that myocardial injury not satisfying the diagnostic criteria for myocardial infarction (according to the joint task force i.e. the European Society of Cardiology, the American Heart Association and World Heart Federation) occurs in many patients peri-operatively, especially patients presenting for non-cardiac surgery. According to the VISION group it is now clear that these injuries contribute significantly to increased risk to perioperative morbidity and mortality in the first year after surgery (1).

DIAGNOSIS OF PERIOPERATIVE MYOCARDIAL ISCHAEMIA

The joint task force states that myocardial infarction is diagnosed if the following criteria are met:

1. detection of rise and/or fall of cardiac biomarkers (preferably troponin) with at least one value above the 99thpercentile of the upper reference limit; and
2. evidence of myocardial ischaemia with at least one of the following:
    a) Symptoms of ischaemia,
    b) ECG changes indicative of new ischaemia (new ST-T wave changes or new left bundle branch block [LBBB] and/or the development of pathological Q waves,
    c) Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality (5, 6).

This definition has been redefined by the results of the VISON study who proposed that early diagnosis of peri-operative myocardial injury should be a priority amongst medical personnel. Even ischaemia diagnosed only by a troponin leak, without any other symptoms or signs, has prognostic significance for these patients (1), especially if it occurs within 30 days post-operatively and other causes of leakage of myocardial proteins, e.g. sepsis, direct myocardial trauma, congestive cardiac failure, etc. have been be excluded (4).

Post-operative troponin elevation is an established method for the diagnosis of myocardial injury; a creatinine kinase MB assay can be obtained in case of difficulty with sampling troponin (2). According to the Vision group a Troponin T leak of 0.02 to 0.03 ng/ml, taken in the perioperative period, is an independent predictor of 30-day mortality [3]. It is unfortunately not possible to have a sensitive bedside test for troponin for early detection of myocardial injury as the results can only be seen after the injury has already occurred.

Myocardial injury is preceded by prolonged myocardial ischaemia; it can take as long as 20 minutes for myocardial injury to occur with prolonged ischaemia. If the treatment of ischaemia is started before myocardial injury occurs the myocardium will resist injury and may end up with myocardial ischemic preconditioning (6). A sensitive and real time monitoring tool for myocardial ischaemia would be a useful tool so that the diagnosis of myocardial ischaemia can be made before the actual myocardial injury ensues and relevant treatment strategies can be embarked on early. In this booklet we shall look at the perioperative use of ST-segment monitoring as a tool for early diagnosis of myocardial ischaemia, which can easily lead to myocardial injury.

Normal and Abnormal ST-Segment Differentiation

A normal ST-segment is isoelectric. This means it is at the same level as the preceding P-R segment and/or the following T-P segment. ST-segment changes are the most sensitive to myocardial ischaemia. Exercise induced ST-segment elevation in abnormal Q wave containing leads is considered normal. However, it is abnormal in leads without Q waves (1, 4).

Tachycardia causes J (junctional) point depression but the ST-segment normally rises faster than 1 millivolt per second and it ends above 0.1 millivolts compared to its baseline when there is no myocardial ischaemia; one needs to verify if the criteria for abnormal ST-segment is met (1).

ST-segment elevation is deemed significant (according to the third universal definition of myocardial infarction) if in V2 and V3:

- V2 and V3 - 0.2 millivolts or above in males above 40 years
- V2 and V3 - 0.25 millivolts or above in males younger than 40 years
- V2 and V3 - 0.15 millivolts or above in woman
- 0.1 millivolts or above in all other leads

Patients must have no features of left ventricular hypertrophy or left bundle branch block for the above to be valid (1). In patients with baseline ST-segment elevation it is not abnormal to see a return to an isoelectric line in cases of myocardial stress, e.g. tachycardia. This, of course, leaves a question of the usefulness of ST-segment monitoring in patients where there is pre-existing left ventricular hypertrophy or abnormal ventricular conduction. Here it is important to note the baseline ST segment to which a comparison will be made if new changes occur peri-operatively.

ST-segment depression is significant for ischemia if one of the following occurs:

- A Junctional (J) point depression of 0.1 millivolts or deeper and a relatively flat ST-segment slope; or
- J point depression of less than 0.1 millivolts, which remains depressed for greater than 80 milliseconds, i.e. remains below 0.1 millivolts

ST-segment changes must be seen in at least 3 consecutive beats. In cases of tachycardia, where the ST segment is less than 60 to 80 milliseconds, ST depression for as short as 40 milliseconds can be accepted for diagnosis of myocardial ischemia [2]. In patients with a baseline depressed ST-segment a 0.1 millivolts change from the baseline is accepted for the diagnosis of myocardial ischemia, as long as all other characteristics of an abnormal ST-segment are met (1).

Differentiating an ischaemic ST-segment deviation from a non- ischaemic ST-segment deviation.

ST-segment deviation can be caused by different factors. It is important to differentiate between ST elevation myocardial infarction (STEMI), or ST elevation from a non-ischemic ST-segment elevation (NISTE) (7). The same is true for ST-segment depression. According to Deshpande 15% of the average population has NISTE and about 91% of the United States Air Force apparently asymptomatic males between the ages of 16 and 58 years had NISTE between 0.3 and 0.1 millivolts. The incidence of NISTE was less prevalent above 40 years of age. This has been termed the male pattern, although only up to 0.1 millivolts in precordial leads is accepted as NISTE. However, history, physical examination and cardiac proteins in combination with the entire ECG is important in diagnosing ST-segment deviation (7).

Intraoperatively subtle ECG changes may indicate myocardial ischemia hence as low as 0.1 milivolts change in ST segment and T wave changes should be considered abnormal (8). Other differential diagnoses of ST segment changes include: secondary myocardial ischaemia (e.g. aortic dissection), pulmonary embolism, pericarditis, myocarditis, rate related changes and electrolyte imbalances (1). Only ischemia related ST-segment deviation will be discussed further in this booklet.

Not all myocardial ischemia and necrosis is symptomatic. C. Richard Conti et al. noted that after coronary artery occlusion, left ventricular mechanical abnormalities and electrographic changes preceded symptoms (9). This means that by the time there are symptoms and/or troponin leak a significant amount of time has passed with ischemia especially in Type 2 ischemia. An early ECG, or even better, real time ST-segment monitoring is paramount.

Myocardial ischemia can be silent (9). This is true throughout the perioperative period. Intraoperative ischemia can be noted as early as from the induction of anaesthesia. This is usually the supply versus demand ischemia with either a very low blood pressure or very high heart rate. The post-operative period is no exception to silent ischaemia

USES

Pre-operative

High risk patients, that is, those with unstable angina, post-acute myocardial infarction and those with coronary artery disease have been investigated with Halter monitoring which shows that of the patients who had silent myocardial ischemia (i.e. diagnostic ST-segment deviation), 75 per cent had multiple vessel coronary artery disease, 62% had proximal coronary stenosis with greater or equal to 50% diameter reduction, and 39% had proximal coronary lesions with greater or equal to70% stenosis. The short coming of the Halter monitor is that it mainly looks at three leads that is Lead 2, avF and V5.

ST-segment monitoring is a fairly big component of exercise stress testing. Among other things exercise stress testing is stopped once significant ST-segment changes are noted (8, 10). It also helps to note the heart rate at which ischaemia occurs – the ischaemic threshold. Exercise stress testing on its own has sensitivity of 68 percent, but exceeds 85 percent for patients with three vessel disease, the specificity of 77 percent (10).

Intra-operative

An awake patient may be able to report symptoms that come with myocardial ischaemia. However, the vast number of operations are done under general anaesthesia where a patient will not feel the symptoms. With the known increase in the high risk patients an ST-segment analysis intraoperatively has become almost routine. ST-segment monitoring has become easier with the new automated ECG monitors which even allow viewing of trends. In a patient under anaesthesia there are several interferences that have to be filtered for accurate ECG analyses.

These include the skin impedance, motion artefacts, diathermy, etc. this then necessitates frequency bandwidth filter of 0.05 to 60 or 100 Hz, a narrower filter may affect will affect a low frequency signal like an ST segment. To note is that automated ST segment analysers only monitor and ST segment at a specific point, be it 80 or 60 milliseconds from the J point. It does not matter the slope thereof. A horizontal or descending slope may be more diagnostic of cardiac ischemia (8). In comparison with coronary angiography, an up-sloping ST segment may increase the false positive yield of myocardial injury to about 27% (8). Monitoring lead 1, 2, 3 and V5 has 89% sensitivity to detecting myocardial ischemia (8). V3 is the most sensitive lead in detecting ischemia (86.6%) followed by V4 (78.9%) and V5 (65.8%). V3 and V5 monitoring increases the sensitivity to 97.4% (10).

Automated ST monitoring has made it easier for trend monitoring of ST segments. Caution should be taken in dealing with possible false positives - especially in patients at low risk of myocardial ischemia. In general what I can conclude is that intraoperative ST monitoring is an important tool for the anaesthetists faced with a high risk patient on the operating table. One needs to record the baseline ST segment so as to compare the changes with that, for any change the reason must be sought, be it tachycardia, significant hypotension, significant decrease in arterial oxygen carrying capacity, etc. The management of myocardial oxygen supply versus demand needs to be kept in mind and managed accordingly.

Post-operative

ST-segment ischaemic changes and myocardial injury were compared by Giora Landesburg et al. in ICU where they noted that ST-segment ischaemic changes are common and associated with myocardial infarction if they continued for greater than 10 minutes. The changes occurred in 21% of their ICU patients studied and was associated with an 4.7 fold increase in the risk of troponin leak (10). 12 lead ECG is obviously superior to both five and three lead ECG. Three lead ECG may benefit detection of arrhythmias only. In patients with CAD more patients showed ST segment ischemia related shifts than with 5 lead ECG in Thompson et al.'s study (44 vs. 16 events, $P<0.05$).

The 12 lead ECG ischemic episodes correlate very well in major vascular surgery patients with both troponin leak and clinically significant myocardial infarction. The appearance of ST segment changes in 2 contiguous leads e.g. V2-4 (anterior wall), V5/6, L1 and aVL (lateral wall) and L2/3 and aVF (inferior wall) frequently strengthen the diagnosis of ischemia.

Local use of an ST-segment.

Pre-operatively exercise stress testing used to be done at IALCH, currently it is not available. The use of ST segments is now only on preoperative assessment of patient just on a 10 seconds strip of an ECG.

Intraoperatively ST-segment monitoring is routinely used in cardiac and vascular operating rooms. It is also used for intraoperative monitoring of a high risk patient in other operating rooms besides the above mentioned. No formal audit has been done to assess the use of ST-segment monitoring, but it is probably underused. In one of the UKZN teaching hospital it is applied as standard practice to chart ST-segment trends.

Post operatively only when there is suspicion on a cardiac event a 12 lead ECG is obtained. There is no routine twelve or five lead monitoring on patients at high risk that probably needs to be looked at.

CONCLUSION

It has become clear that perioperative myocardial injury is common and a significant contributor to perioperative morbidity and mortality. Advances in detecting this type of ischemia are still in progression. Better bedside monitors are still lacking and that leaves us with the ECG to monitor myocardial ischemia perioperatively. High risk population benefits more in ST segment monitoring than the general population.

An automated five or twelve lead ECG monitoring is still of paramount importance when used appropriately and has good correlation to troponin leak. One needs to be aware of other differentials to ST segment deviation to be able to productively use this monitor productively. As long as we still do not have a more sensitive real time myocardial ischemia monitor, the ST segment remains our next easily accessible monitor.

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