The aim:

Using tests to clearly identify the compromised fetus at a stage at which intervention will improve the outcome.

What is fetal compromise?

A fetus that is at risk of damage from hypoxia.

Causes of hypoxia:

1 - Uteroplacental insufficiency, fetal growth restriction.
2 - Decreased maternal oxygenation.
3 - Impaired fetal blood supply to the placenta.
4 - Intrinsic fetal conditions.

Physiologically:

The production of ATP requires $O_2$.

Hypoxia leads to anaerobic metabolism with decreased production of ATP (2 molecules vs. 24).

Lactic acid accumulation leads to metabolic acidemia, eventually cell damage and death.

The brain, myocardium and kidneys are the most sensitive organs, eventually fetal death may occur.

Fetal response to hypoxia:

The human fetus demonstrates complex patterns of activity including breathing movements, gross body movements and fine motor movements.
The fetus exposed to hypoxia, will demonstrate adaptations designed to conserve energy and decrease $O_2$ consumption. These include:

- decreased fetal movement.
- redistribution of blood.
- decreased urine output leading to oligohydramnios.
- since fetal growth accounts for substantial fraction of energy of total substrate consumption, therefore in hypoxia growth is decreased leading to fetal growth restriction.

The majority of currently available tests of fetal well-being are designed to detect these adaptive changes, these tests include:

1- Fetal movement counting (kick count):

Depends on maternal perception of fetal movement, therefore it should not be routinely provided.

But those women at high risk of fetal compromise are advised to pay attention to their fetal movement and it is recommended that pregnant females who report a reduction or an alteration in the movement of their fetuses, should be offered some other forms of assessment of fetal well-being.

2- Fetal heart rate recording: which include cardiotocography (CTG) and computerized CTG.

A - Cardiotocography:

Indirect method of monitoring of fetal heart rate. It uses the physical principle of doppler effect to detect fetal heart motion.

physiologically, the fetal cardiac activity is regulated through sympathetic and parasympathetic signals and by vasomotor, chemoreceptors and baroreceptors mechanisms, so pathological events
such as hypoxia modify these signals and eventually alters the fetal cardiac response.

What are the parameters to look for in a CTG trace?

- Baseline fetal heart rate.
- Fetal heart rate variability.
- Fetal heart rate acceleration.
- Fetal heart rate deceleration.
Normal CTG:

- Baseline rate 110 – 160 bpm.
- Variability 10 – 25.
- 2 accelerations in a 20 – 30 min trace.
- No deceleration.

Suspicious CTG:

* Abnormal baseline rate.
* Reduced variability < 10 bpm.
* Absence of acceleration.
* Variable deceleration.

Abnormal CTG:

No accelerations and two or more of the following:

- Abnormal baseline rate.
- Abnormal variability.
- Repetitive late decelerations.
- Variable decelerations with duration > 60 sec, late recovery to baseline, late deceleration component, poor variability between and during decelerations.
**Non stress and stress CTG:**

Non stress: when the patient is positioned comfortably without contraction.

Stress test: refers to what the fetus is experiencing where contractions are stimulated either by nipple stimulation or by oxytocin. A positive test is fetal cardiac decelerations in response to uterine contractions.

**B) Computerized CTG:**

Computer programs have been developed to analyze fetal heart rate recording. By this we can assess short – term variability.

**3) Biophysical profile:**

It depends on ultrasonic assessment over 30 min of 5 parameters then scoring is done so that each parameter is given a score of 2 when normal and zero when abnormal producing a total score of 10.

**The parameters are:**

* Fetal breathing movement.
* Gross body movement.
* Fetal tone.
* Non stress test.
* Qualitative amniotic fluid assessment.
4) Fetal biometry and doppler ultrasonography:

Doppler ultrasound makes use of the phenomenon of doppler frequency shift, where the reflected wave will be at a different frequency from the transmitted one if it interacts with moving structures.

Wave forms from umbilical artery provide information on the fetoplacental blood flow and should be performed on high risk pregnancies.

So if the resistance index measured in the umbilical artery rise above the 95\textsuperscript{th} centile this means faulty perfusion of the placenta with fetal hypoxia. Absent or reversed end diastolic flow in the umbilical artery is particularly serious with correlation to fetal distress and intrauterine death.

Types of deceleration on CTG and there causes:

**Early decelerations**: are benign, They are caused by head compression and in general are a normal physiological response to a mild increase in intracranial pressure. Importantly they are uniform in shape and start and finish with the contraction. They may be said to mirror the contraction.
Variable decelerations are a repetitive or intermittent decreasing of FHR with rapid onset and recovery. Time relationships with contraction cycle may be variable but most commonly occur simultaneously with contractions. The significance of variable decelerations depends on the overall clinical picture and specific features of the decelerations themselves, as well as other features of the CTG. Variable decelerations in association with other non-reassuring or abnormal features change the category of the deceleration to ‘complicated.’
Late decelerations are defined as uniform, repetitive decreasing of FHR with, usually, slow onset mid to end of the contraction and nadir more than 20 seconds after the peak of the contraction and ending after the contraction. Late decelerations are caused by contractions in the presence of hypoxia. This means that they will occur with each contraction and the fetus is already hypoxic. There will be no features of a well oxygenated fetus, like early or typical variable decelerations, normal baseline variability or shouldering. They start after the start of the contraction and the bottom of the deceleration is more than 20 seconds after the peak of the contraction. Importantly, they return to the baseline after the contraction has finished. In the hypoxic fetus, this will include decelerations of less than 15bpm (and occasionally less than 5bpm)