Sickle cell hemoglobin

The following tests are available:

- **Hemoglobin in EDTA blood (Hb)**

Material: 3 ml EDTA blood
Stability: 7 days at 2 to 8°C
TAT: same day, FML
Method: photometry
Units: g/dl
Ref.- range: see report

- **Hemoglobin, free, in plasma**

Indication: Suspicion of hemolysis
Material: 1 ml heparin plasma
TAT: 3-7 days*
Method: spectral photometry
Units: mg/dl
Ref.- range: see report
Note: determination of haptoglobin recommended

- **Hemoglobin A2**

Material: 2 ml EDTA blood
Stability: 7 days at 2 to 8°C
TAT: 3 days, FML
Method: HPLC
Units: %
Ref.- range: <3.5

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- **Hemoglobin, glycated**

General:

The glycated hemoglobin HbA1 is a naturally occurring hemoglobin component of adult hemoglobin and consists of several subfractions (HbA1a, HbA1b and HbA1c). As there is no enzyme in the erythrocytes, which splits glycated hemoglobin, the glycation persists irreversibly as long as the erythrocyte is alive (120 days). Thus HbA1c indicates the phases of past hyperglycemias and informs about glucose levels during the last 3 months.

Indication:	Monitoring of glucose adjustment
Material:	2 ml EDTA blood
Stability:	7 days at 2 to 8°C
TAT:	same day
Method:	turbidimetric
Units:	%
Ref.- range:	see report
Note:	Our HbA1c is performed according to the IFCC Standard. The IFCC standards are more sensitive and are able to recognize a pathologic glucose tolerance at the earliest.

- **Hemoglobin F**

General:

Fetal hemoglobin, (also hemoglobin F or HbF) is the main oxygen transport protein in the fetus during the last seven months of development. Functionally, fetal hemoglobin differs from adult hemoglobin by its ability to bind oxygen with higher affinity than the adult form, providing the developing fetus better access to oxygen from the mother's bloodstream.

In infants >3 months, fetal hemoglobin is nearly completely replaced by adult hemoglobin (HbA). Children with sickle-cell disease produce a defective form of hemoglobin called hemoglobin S. HbS forms filaments and can change the erythrocyte from round to sickle-shaped under certain condi-tions. These erythrocytes show an increased tendency to stack on top of one another and to block blood vessels. It can lead to painful vasoocclusive epi-sodes, which are a hallmark of the disease.

In adults, fetal hemoglobin production can be reactivated pharmacologically, which is useful in the treatment of diseases such as sickle-cell disease.

The following examinations are available:

- **Fetal hemoglobin**

Indication: Thalassemia syndromes (α-, β-, δ-thalassemia, HbH-disease), hereditary HbF-persistence (HPFH), hemoglobinopathy (HbS, Hb Lepore), anemia

Material: 3 ml EDTA blood

Stability: 7 days at 2 to 8°C

TAT: 3 days, FML

Method: HPLC

Units: %

Ref.- range: see report

Note: HbF increased: hereditery spherocytosis, AML, CML, myelophthisic anemia, untreated pernicious anemia;

- **Fetal hemoglobin (fetomaternal blood transfusion)**

General:

The quantification of fetal erythrocytes in the maternal blood is useful for the identification of a fetomaternal blood transfusion. Physiologically 0.1 - 0.2 ml of fetal blood pass from the fetus to the mother during delivery (in 30 - 50% of the cases). >0.1 ‰ fetal erythrocytes indicate a fetomaternal microtransfusion (0.5 – 15 ml fetal blood transfer), >3 ‰ fetal erythrocytes blood indicate a fetomaternal macrotransfusion (more than 15 ml fetal blood).

Indication: Suspicion of fetomaternal micro-/macrotransfusion, control of blood contamination in amniotic fluid after amniocentesis, vaginal bleeding during pregnancy, differentiation between maternal and fetal blood

Material: 3 ml EDTA blood, amniotic fluid, cord blood

TAT: 5-7 days*

Method: flow cytometry

Units: per mille (per thousand)

Ref.- range: <0.1 per mille

- **Hemoglobin S**

see also **Sickle cell anemia, genetic test**

General:

Sickle cell disease is observed frequently in the African/Asian population as well as in the Mediterranean/ Middle Eastern population. Here, a common problem is a combination of sickle cell and beta thalassemia genes.

Sickle cell anemia is an autosomal recessive inherited disorder caused by a mutation in the hemoglobin gene. The presence of two defective genes from mother and father causes sickle cell disease. If each parent carries one sickle hemoglobin gene (S) and one normal gene (A), each child has a 25% chance of inheriting two defective genes and manifesting sickle cell disease; a 25% chance of inheriting two normal genes (healthy); and a 50% chance of being an unaffected, heterozygous carrier (as with the parents)

The clinical course of sickle cell anemia does not follow a single pattern; some patients have mild symptoms, and some present with very severe symptoms. The sickle-shaped red blood cells tend to stick to small blood vessels thereby blocking the blood flow. The following conditions are observed: hand-foot syndrome, fatigue, paleness, and shortness of breath, pain occurring unpredictably in any organ or joint, eye problems, growth retardation and delayed puberty. Complications: infections, stroke, acute chest syndrome.

Indication:	suspicion of sickle cell anemia
Material:	3 ml EDTA blood
Stability:	7 days at 2 to 8°C
TAT:	3 days, FML
Method:	HPLC
Units:	%
Ref.- range:	undetectable

For complete list of laboratory test offered at Freiburg Medical Laboratory, please visit http://www.fml-dubai.com/parameter-listings/