The poem below is dedicated to all the Thalassemia patients around the world, and for all the family and friends of Thalassemia patients.

"Angel by Your Side"

May you always have an angel by your side,
Watching over you in all things you do,
Reminding you to keep believing in brighter days,
Finding ways for wishes and dreams
to take you to beautiful places.
Giving you hope that is as certain as the sun,
Giving you the strength of serenity as your guide.
May you always have love and comfort and courage,
And may you always have an angel by your side.

May you always have an angel by your side,
Someone there to catch you if you fall,
Encouraging your dreams,
Instructing your happiness.
Helping your hand and helping you through it all.

In all of your days, our hearts are always changing,
Tears come along as well as smiles,
Along the roads you travel,
May the miles be a thousand more times lovely than tedious,
May they give you the kind of gifts that never, ever ends.
Someone wonderful to love
And a dear friend in whom you can confide.
May you have rainbows after every storm.

May you have hope to keep you warm.

And may you always have an angel by your side.

A thalassemia baby in hospital bed.
Thalassemia

The thalassemias (2 forms) are among the commonest inherited blood diseases in the world. Fifteen million people worldwide have clinically apparent thalassemic disorders. Reportedly, thalassemia carriers in India alone number approximately 30 million. These facts confirm that thalassemias are among the most common genetic disorders in humans. They are encountered among all ethnic groups and in almost every country around the world. Countries like Italy, Greece and Cyprus have the highest frequency of thalassemia cases. In Cyprus, 1 in 7 individuals is a carrier for the gene, which translates into 1 in 49 marriages between carriers and 1 in 158 newborns expected to have beta thalassemia major. There exists a thalassemic “belt” that includes the Mediterranean passing through West and Central Asian countries like Turkey, Iran, Afghanistan onto Pakistan, India & Bangladesh and passes on to the South East Asian countries like Indonesia, Burma & Thailand.

Thalassemia may have originated over 50,000 years ago in a valley South of Italy and Greece now covered by the Mediterranean Sea. The word thalassemia was derived from the Greek words of “thalassa” for the Mediterranean Sea and “haima” for blood. But thalassemia was recognized as a clinical entity by Dr Thomas Cooley and Dr. Pearl in 1925. Most of their patients, and those reported by others later, were of Mediterranean ancestry. Hence this group of anemias are also known as Cooley’s Anemia or Mediterranean Anemia.

A hemoglobin molecule is usually made up of 2 α chains and 2 β chains. The amount of β and α chains a person makes is controlled by the hemoglobin gene they inherit from their parents. Where a person has inherited hemoglobin A from both parents they will have TWO usual β genes (β^β), one from each parent, and FOUR α genes (αα/αα), one pair from each parent (diagram 1). They would, therefore, produce normal amounts of β and α to make healthy red blood cells. In people with Thalassemia, the genes that code for hemoglobin are missing or variant (different than the normal genes). Severe forms of thalassemia are usually diagnosed in early childhood and are lifelong conditions.

Alpha Thalassemia

Alpha Thalassemia Trait
Some people do not inherit the usual number of α genes in which case they would have a condition known as α thalassemia minor (trait). The terms “minor” and “trait” refer to the same thing. Alpha
thalassemia trait is not an illness or disease, and it does not make people feel unwell or weak. There are no outward signs to show if a person has α thalassemia trait. They are perfectly healthy. The blood of people who have α thalassemia trait has a slightly lower level of hemoglobin. However they do not need to take iron medicines, unless special blood tests have shown that they are lacking in iron. Millions of people worldwide have α thalassemia trait, especially in areas where malaria is, or was, common.

This characteristic of the blood is passed on from parents to their children through the genes, in the same way that other physical characteristics are passed on through the genes. If a person is born with α thalassemia trait, they will have it all their life but it does not cause them to ill in any way. It may be passed unrecognized through a family for many generations.

There are two types of α thalassemia trait:

**Alpha plus thalassemia trait**
This type of thalassemia trait (diagrams 2 & 3) is most common. It is very common in people of the following parts of the world: Africa, the Caribbean, the Mediterranean islands, India, Pakistan, Bangladesh, the Middle East, and South East Asia.

One thing that it is important to know that if a person has α plus thalassemia trait, it may be mistaken for iron deficiency anemia, and then the person may be treated with iron medicines unnecessarily.

**Alpha zero thalassemia trait**
This type of α thalassemia trait (diagram 4) is less common but can be inherited by people whose families come from any of the following parts of the world: the Middle East, South East Asia, China, and the Mediterranean islands.

In this instance, no α genes are inherited from one parent, but two normal α genes are inherited from the other. Having this type of α thalassemia trait does not affect health; there is sufficient α genes for making hemoglobin. If both parents have α zero thalassemia trait, and are planning to have children, there will be a 1 in 4 chance that the child could inherit the usual number of α genes, a 2 in 4 chance the child could inherit α zero thalassemia trait, and a 1 in 4 chance the child could inherit α thalassemia major (example 1).

If one parent has α plus thalassemia trait and other parent has α zero thalassemia trait (example 2); each time they are expecting a child there is a 1 in 4 chance the child could inherit the usual number of α genes, a 1 in 4 chance of inheriting α zero thalassemia trait (just like one of their parents), a 1 in 4 chance of inheriting α plus thalassemia trait, (like the other parent) or a 1 in 4 chance of inheriting a condition called hemoglobin H disease.

**Alpha Thalassemia Major**
Alpha genes are essential for making hemoglobin. If a child inherits α thalassemia major, (s)he will not have any α genes and will not be able to make normal hemoglobin, which is essential for carrying oxygen around the body.

Alpha thalassemia major is a serious blood disorder because it affects the baby whilst it is growing in its mother's womb. If a baby inherits this condition, the mother will also be at risk of serious complications during pregnancy.

This condition is life threatening: babies who have α thalassemia major are unlikely to survive pregnancy.
A baby can only inherit α thalassemia major if:
• both parents have α zero thalassemia trait, or
• one parent has a condition known as H disease and the other has α thalassemia trait, or
• both parents have H disease

Hemoglobin H disease
In this condition the baby usually makes sufficient hemoglobin to allow for normal life; however, the red blood cells have less red pigment than usual. On occasions, this condition may need medical treatment; for example, blood transfusion or medication.

Beta Thalassemia

Beta Thalassemia Minor
Beta thalassemia minor occurs when an individual inherits one usual β gene from one parent and one unusual β gene from the other (Hb A βthal). It is found in areas of the world where malaria is, or was, common.

People with β thalassemia minor have red blood cells, which are smaller, paler, and have less red pigment, but this does not cause any health problems. Generally, their red blood cells are still able to carry oxygen around the body as efficiently as someone with the two usual β genes. Because these individuals are themselves healthy, they will not know that they have this unusual trait unless they have a special blood test.

If a couple both have β thalassemia minor, each time they expect a child there is a 1 in 4 chance that their child could inherit a serious blood disease called β thalassemia major.

Beta Thalassemia Major
It is a serious anemia affecting people who have inherited the two unusual β thalassemia genes, one from each parent.

People with β thalassemia major do not produce enough healthy, mature red blood cells; therefore, they become very pale and anemic. From about the age of 3 months children with this condition become very pale, lethargic, lose their appetite and soon fail to thrive, and usually die between 1 and 10 years of age. But with proper medical care they can have long life.

Difference between β thalassemia major and minor
Beta thalassemia minor (trait) is not a disease and cannot change later in life. It is significant when one is considering having children because the child may inherit this unusual gene from both parents.

Beta thalassemia major (disease) is a serious blood condition. Individuals with it are unable to make enough healthy red blood cells and depend on blood transfusions all their life.

Iron overload and iron chelation
Thalassemia causes iron to accumulate in the body. There are two main ways in which patients with thalassemia absorb iron: from the diet, and from transfused blood. If this excess iron is not removed, it can cause damage to important organs such as the liver and heart. Thalassemia patients must therefore use special drugs called chelators, which remove iron from the body.

Iron in thalassemia
In thalassemia major, the body attempts to compensate for the patient’s severe anemia by absorbing significantly more iron from the gut than normal (2-5g/year compared to 0.0015g/year in healthy individuals), in order to make more red blood cells. How much more iron is absorbed depends on the severity of the anemia. Other factors may also play a part in determining the amount of iron absorbed by the
gut. For example, the presence of vitamin C increases the amount of iron absorbed, while tea and some cereals lead to a decrease.

The main source of iron overload in patients receiving transfusions however is blood transfused. In fact, the amount of iron the patient absorbs through blood transfusions is far greater than that absorbed from the diet through the gut. It is therefore important that patients on regular blood transfusions, use iron chelators that bind with iron and remove it from the system.

The clinical symptoms of iron overload generally appear around the age of 10, although evidence of the toxic effects of iron has been found in the liver of much younger children. Heart disease - one of the most frequent causes of death in thalassemia major - has also been reported within 10 years of the start of a transfusion regime, although heart failure does not usually occur until after 15 years or more.

Iron loading is also the most important cause of delayed sexual maturation in patients with thalassemia, affecting about half of both male and female patients. In addition, iron loading can cause difficulties in women trying to conceive (around 25% of cases), and is frequently a cause in the development of diabetes mellitus. Over the long term, excess iron causes bone complications and damage to other important organs, such as the thyroid and parathyroid. Therefore patients must receive treatment to remove excess iron, which will otherwise accumulate in the body with serious effects on the patient's quality and length of life.

Treatment of β thalassemia major

Over the last three decades, clinical observations and research have established that thalassemia major is a treatable condition. Studies have shown that regular transfusion therapy with safe and appropriately processed blood, combined with regular and effective iron chelation tremendously increase patients' survival and quality of life.

**Blood transfusion therapy**

Regular blood transfusions greatly contribute to the quality and length of life of patients with thalassemia major, and have been a central aspect of the treatment of thalassemia since the 1960s.

If not effectively managed, the severe anemia and over-expansion of the bone marrow characteristic of thalassemia major can lead to:

- poor growth
- facial and other bone deformities
- fragile bones and bone fractures
- enlarged liver and spleen
- impairment of normal physical activities

Patients should only begin transfusion therapy when:

- Hb levels are registered at less than 7g/dl on two successive occasions, more than two weeks apart
- Hb levels are >7g/dl but accompanying physical characteristics are noted, such as:
  - facial changes
  - poor growth and limited weight gain
  - bone fractures
  - extra-medullary hematopoiesis, resulting in tumor masses

Where these criteria are observed, transfusion therapy should not be delayed.

Patients with thalassemia major lack red blood cells. Therefore, patients receiving blood transfusion therapy should ideally receive only red blood cells, which contain none of the other components of whole blood - e.g. plasma, white blood cells and platelets. If a patient receives whole blood, there is a risk that the body's circulatory system will be overloaded, developing complications such as heart failure and pulmonary edema. The removal of white cells and platelets from whole blood also decreases the risk of unwanted effects such as fevers during and after the blood transfusion. Although such symptoms can be treated, every effort should be made to avoid any complications by providing only that component of blood the patient requires.

The volume of blood a patient requires and the rate of transfusion depends on the patient's age and clinical status, the solutions added to preserve the

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<th>Consequences of Excess Iron</th>
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<td>Fibrosis</td>
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<td>Cirrhosis, especially if hepatitis C active</td>
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red blood cells to be transfused, the hematocrit of the donor's RBCs, and the target level of hemoglobin.

In moderate -- as opposed to hyper- or super- -- transfusion regimes (recommended by most thalassemia specialists), patients usually receive 10-15ml of concentrated red blood cells per kg of body weight (volume), transfused over 3-4 hours (based on a 75% hematocrit of the donor's RBCs) (rate), every 2 to 5 weeks (interval). A non-splenectomized patient requires approximately 180ml of pure red blood cells/kg/year, while a splenectomized patient requires about 133ml/kg/year.

Where a patient suffers cardiac problems or where blood transfusions begin when levels of hemoglobin are below 5g/dl, smaller volumes of blood are administered, at a slower rate -- for example, 2-5ml of RBC/kg/hour.

Transfusion regimes should aim to keep patients' hemoglobin levels at between 9-10.5g/dl before transfusion and not more than 15g/dl after transfusion. Under this regime, patients will experience:

- minimal expansion in bone marrow
- normal growth and increased physical energy
- no or delayed enlargement of the spleen
- decreased viscosity of the blood associated with risk of thromboembolic complications, headaches and nose bleeding and
- lower deposits of excess iron on the organs

Iron chelation treatment
As the body has no effective means of removing iron, the only way to remove excess iron is to use drugs called iron chelators (iron binders), which form a compound with iron that can be excreted from the body through the urine and/or stools. As a general rule, patients should begin iron chelation treatment once they have had 10-20 transfusions, or when ferritin levels rise above 1000µg/l.

Desferrioxamine (DFO) was the first iron chelation drug to be manufactured. First produced in the 1960s, DFO was introduced onto the market in the early 1970s for the treatment of thalassemia major.

Desferrioxamine treatment is difficult, time consuming, painful and expensive. It must be adhered to over a lifetime. Although it is a life-saving therapy, DFO treatment does not give patients the sense of immediate benefit they feel, for example, after a blood transfusion.

Deferiprone (1,2 dimethyl-3-hydroxypyrid-4-one, L1) was first licensed for use in 1995 in India, for use by patients who cannot use DFO because of toxicity, or inability to comply with recommended dosage. In most cases, patients are prescribed 75mg/kg body weight per day of L1, taken in three doses.

A recent clinical study has suggested that using DFO in combination with L1 may increase the quantity of iron excreted from the body, perhaps because each drug removes iron from different parts of the body.

A number of other iron chelating drugs are currently under investigation, with particular emphasis being placed on affordable, user-friendly oral chelators. It is hoped that orally active chelators will soon become available for clinical use improving treatment options for patients with thalassemia.

Bone marrow transplantation
Bone marrow transplantation (BMT), if successful, can offer a complete cure to patients with thalassemia major. BMT for thalassemia was started in 1981.

A bone marrow transplant involves taking (harvesting) bone marrow from a healthy individual (called the donor) to be donated to a patient (called the host or recipient). The patient's “unhealthy” marrow is first destroyed by drugs, or sometimes by irradiation, in a process known as conditioning. The healthy marrow, usually taken from the donor's hipbone, is then given as a liquid into the patient's blood stream, in a similar way to a regular blood transfusion. Once in the recipient's blood, the donor's bone marrow cells travel to the large bones where they start producing normal, healthy blood cells. This takes about 2-3 weeks.

Although this procedure can be extremely successful, it also carries serious risks. The risks and benefits of each case must therefore be carefully considered before a transplant is carried out. For the best results,
bone marrow must be given from a brother or sister who is a complete tissue ‘match’. Complications include infection and bleeding, which usually occur before the donor marrow has replaced the marrow of the recipient.

It is important to understand that BMT only treats the bone marrow. So patients planning a pregnancy should be reminded that their genes remain affected by thalassemia and that any affected genes will still be passed on to their children.

Cord blood transplantation
Another transplant technique involves testing the HLA characteristic of a fetus carried by a mother who has an affected child. If the fetus HLA matches that of the older child, cord blood - the blood remaining in the placenta and umbilical cord after the birth of a baby, which is a rich source of the stem cells responsible for producing blood cells - can be transplanted into the affected child instead of bone marrow. However, an important limitation to this technique is that the donor's cord blood must contain a minimum number of nucleated cells ($>1 \times 10^7$) per kg of the recipient's body weight. In addition, as with BMT, the recipient's liver, kidney, heart and lungs must be functioning adequately.

Medical problems associated with thalassemia and its treatment
The treatment of thalassemia major has improved dramatically over the last two decades, leading to a sharp increase in survival rates and significant improvements in patients' quality of life. Nonetheless, patients with thalassemia major may suffer a number of medical problems, some of which are due to the disease itself, while others are the result of inappropriate or poor quality treatment, including inappropriate blood transfusion therapy, lack of blood safety and sub-optimal use of DFO.

Hypersplenism
Many patients with thalassemia major experience problems of the spleen. A normal spleen contains 20-30ml of red blood cells. However, in patients with persistent moderate to severe anemia as a result of inappropriate blood transfusion, the spleen may hold a litre (1000ml) or more of blood. This is because the spleen produces extra red blood cells in a process known as extra medullary erythropoiesis - i.e. the synthesis of red blood cells outside the normal site of production, the bone marrow - in an effort to help the body overcome anemia.

The spleen performs several important functions, including protecting the body from infection by filtering the blood to remove any invading microbes, bacteria and parasites. The spleen is also responsible for removing red blood cells at the end of their lifecycle from the circulation, breaking them down to release the globin and iron from their hemoglobin to be reused in making new cells. In patients with thalassemia major, however, this recycling process does not work properly. Instead the iron is deposited in the spleen or released into the blood stream and transferred back to the spleen. In addition, most of the red blood cells in patients with thalassemia are abnormal in shape and so get stuck in the spleen. As a result, the spleen grows larger and larger, often forcing the abdomen to grow along with it.

In an effort to cope with the increased demands made on it, the spleen often becomes hyperactive - a condition known as hypersplenism - and in the process also destroys the normal red blood cells the patient receives from blood transfusions. As a result, the patient requires more blood at each transfusion, but the transfusions fail to have an effect on the anemia. A hyperactive spleen may also destroy other components of the blood, such as white blood cells and thrombocytes.

Hypersplenism cannot be corrected. Therefore, once it is confirmed that the spleen is performing in a way that is harmful to the body, as described above, it must be surgically removed - a procedure known as a splenectomy. A splenectomy will not cure thalassemia - it simply solves the specific problems that result from an enlarged spleen.

Heart, liver and endocrine complications
Thalassemia major patients often experience problems with the heart, liver and endocrine glands, all of which are usually associated with iron overload. However, unlike heart problems, patients receiving insufficient or no blood transfusions rarely develop liver and endocrine problems, partly because these conditions occur in highly transfused patients on poor iron chelation regimes, and partly because insufficiently transfused patients will not usually live long enough for the condition to develop.

Diabetes mellitus in Thalassemia
Diabetes occurs when the body's metabolism of glucose is disrupted. The organ responsible for the metabolism of glucose is the the β cells of pancreas which produces insulin, the hormone used to
metabolize sugar. Iron can damage these special \( \beta \) cells, and so the body’s ability to utilize sugar is reduced and sugar accumulates in the blood.

**Fertility and reproduction in Thalassemia**

Women with thalassemia can safely complete pregnancy. However, the decision to conceive should be carefully considered by a couple in consultation with their doctor. Women who express the desire to become pregnant should undergo a complete evaluation of their clinical and psychological condition.

Female patients with thalassemia who have a normal menstrual cycle may conceive spontaneously. However, those suffering from primary or secondary amenorrhea will need hormonal treatment in order to stimulate the production of ova and the induction of ovulation. Male patients with thalassemia who suffer azoospermia often respond to a combination of therapeutic hormones, administered over the course of one year.

Once a patient is confirmed to be pregnant, a number of measures should be taken:

- Use of DFO should stop as soon as the pregnancy is diagnosed, as the effect of the drug on the embryo is not clear. However, in animal studies, it has been associated with severe damage to the embryo. If a pregnant patient is extremely iron overloaded or develops severe heart problems, low doses- 20-30mg/kg/day - have been used in the late stages of pregnancy.

- Pregnant women are transfused more frequently with low volume in order to keep Hb at satisfactory levels (10-15g/dl)

- Heart function should be closely monitored. Research has shown that women who begin their pregnancy with lower ferritin levels have better heart function than those with higher ferritin levels

- The patient should be monitored for the development of diabetes mellitus or other endocrinopathies

**Infections in thalassemia**

Patients with thalassemia major have a higher risk of infection because of:

- Anemia
- Splenectomy
- Iron overload
- Blood transfusions
- Use of desferrioxamine

**The choices available for an “at-risk” couple**

Where a woman carrying the \( \beta \) thalassemia trait is considering having a child or is already pregnant, her partner (if not aware of his carrier status) should be tested at once to find out if he also has the thalassemia trait. If they are both carriers, the couple may decide to proceed with planning a family or, if already pregnant, may consider continue the pregnancy and where this is possible, to proceed with testing the fetus for thalassemia, possibly deciding to terminate pregnancy if the fetus is affected.
Other choices considered by “at-risk” couples include separation, adopting, proceeding to in vitro fertilization with foreign healthy sperm or ova. However, parents mainly due to religious beliefs may not decide to find out the status of the child and continue with the pregnancy.

Prenatal testing
Amniocentesis
Amniocentesis is performed in the second trimester of pregnancy, after about 15 (18-22) weeks’ gestation. Using ultrasound as a guide, a trained obstetrician inserts a very thin needle through the mother’s abdomen to withdraw 2-3 tablespoons of amniotic fluid. The fetal cells present in the fluid are then analyzed in the laboratory to determine whether the fetus has thalassemia.

This test is used when the pregnancy is far advanced. It poses no significant risk to the mother. However, the test may cause a miscarriage -- from a few days to a few weeks after the test.

Chorionic Villus Sampling
Chorionic Villus Sampling (CVS) can be performed somewhat earlier than amniocentesis, at about 10-11 weeks’ gestation. Using ultrasound as a guide, the specialist obstetrician removes a small sample of the chorionic villi -- cells that contain the same genetic information as the fetus and which will eventually form the placenta. The cells are removed either by a thin needle inserted through the mother's abdomen (transabdominal) or a thin catheter inserted through the vagina (transcervical). The cells are then analyzed to make a diagnosis.

As with amniocentesis CVS poses no significant risk to the mother. However, there is again a small risk of a miscarriage. If a miscarriage does occur, it can be difficult to know whether it was due to CVS, because many miscarriages happen naturally at around 12 weeks of pregnancy.

There may be an increased risk of the baby’s limbs being malformed if CVS is done very early in pregnancy - i.e. before the 8th week after the last menstrual period. However, there is no evidence of an increased risk of any malformation when CVS is carried out after the beginning of the 9th week after the last menstrual period. This is why the procedure is generally carried out after the beginning of the 10th week after the last period.

Amniocentesis and CVS are both based on DNA testing. In the case of CVS, laboratory scientists study the hemoglobin genes contained in the DNA of cells from the chorionic villi to see if the baby will be normal, a thalassemia carrier or will have thalassemia major. Analysis of the sample usually takes about a week.

Termination of pregnancy
If the test shows that the baby is affected, the couple may decide to end the pregnancy. The role of the genetic counsellor and the obstetrician in these cases is extremely important. Even at this stages decisions have been taken by the couple to continue with the pregnancy accepting the lifelong treatment of the affected child. If pregnancy termination is the choice, however, this is done in one of the two ways, depending on the stage of the pregnancy.

Early termination
Early terminations can be carried out when a woman is less than 14 weeks pregnant. The couple should be informed that termination does not reduce the woman's chance of having another baby. However, it should also be explained that each pregnancy conceived by an at-risk couple carries the same risk of producing an affected child. If the couple wishes to know whether any subsequent babies conceived carry thalassemia, prenatal diagnosis will have to be carried out again.

Late termination
The procedure for terminating a pregnancy at over 14 weeks involves inducing labour by introducing hormones (prostaglandin) into the womb. The labour may last for several hours and the procedure is much more upsetting for the woman than an early termination. Again, this type of termination does not affect the woman’s ability to become pregnant again.
Thalassemia in Bangladesh

Thalassemia is a common problem in Bangladesh. According to World Health Organization (WHO) about 4.8 million people in Bangladesh are now carrying the gene of this silent killer disease, which is four per cent of the total population of the country. It is estimated that approximately 6000 babies with different types of thalassemia are born in Bangladesh each year.

Diagnostic facilities in Bangladesh:

Although, molecular diagnosis of thalassemia is not possible in Bangladesh, still facilities such as peripheral blood film study, reticulocyte counts and radiological diagnosis are available. Hemoglobin electrophoresis is possible in a few institutions and private laboratories at Dhaka.

Treatment of thalassemia & facilities in Bangladesh:

Most of the children with thalassemia require blood transfusion. Unfortunately the facilities for blood transfusion in the country are poor and the cost of iron chelation is far beyond the scope of general people. Professional blood donors are still the principal source of blood for transfusion even with a high seroprevalence of hepatitis B (19-29%) and hepatitis C. Thalassemic children requiring multiple transfusions are at greater risk of acquiring infections and the risk increases when the sources of blood are professional donors. None of the blood transfusion centers in Bangladesh practices the recommended laboratory screening procedure for infectious diseases which possess a major risk of acquiring infections. Even in the transfusion centers of academic hospitals, test for anti-HCV and anti-GHV are not in regular practice. Moreover, pre-transfusion screening of blood is costly as most of them are not done in academic institutions. The situation of voluntary blood donation though improved than the past is still not encouraging. Only few voluntary organizations like ‘Sandhani’ and ‘Red Cresent Society’ are driving to promote voluntary donation.

The unfortunate children should be diagnosed early with the aim to avoid maltreatment and to include them in the ideal transfusion-iron chelation protocol to promote their normal growth and development until Bone Marrow Transplantation (BMT) can be made available in the country. In this respect following few suggestions came out from the clinicians:

1. Facilities for diagnosis should be improved. Hemoglobin Electrophoresis should be made available in all Academic institutions all over the country.

2. Screening program should be set up to detect asymptomatic, previously and known thalassemic heterozygotes for proper genetic counseling.

3. Setting up of ideal pre-transfusion screening facilities in all academic institutions.

4. Voluntary blood donation should be promoted.

5. The cost of iron chelating drugs (desferrioxamine, deferiprone) should be reduced and be made available in the country.

6. Logistics like Infusion pump, Filter (IMUGARD iii-RC; Terumo, Japan) etc, should be made available with reduced cost.

7. Social mobilization to set up special center to augment better services for thalassemic children.

8. Research protocols should be encouraged in this field.

References: