Physical stress such as occurs during surgical operation or following severe injury or trauma induces a fall in the plasma ascorbic acid (AA). We observed that in major trauma like severe head injury, lacerated injury or burns, plasma AA level was very low, 0.08 \pm 0.14 \text{ S.D. mg/dl}. The mean plasma AA level in a group of patients those underwent major surgical operations was found to be 0.05 \pm 0.07 \text{ S.D. mg/dl}. The whole blood AA levels were practically nil in cases of major trauma like lacerated injury/crush injury, severe head injury and burns. The mean whole blood AA level in a group of patients those underwent major surgical operations was 0.15 \pm 0.14 \text{ S.D. mg/dl} major surgical operations. There was a corresponding increase in the blood dehydroascorbic acid (DHA) content in all the cases of major trauma. The mean blood DHA values as observed in cases of lacerated injury/crush injury, major surgical operations, severe head injury, burns were 0.80 \pm 0.30 \text{ S.D.}, 0.85 \pm 0.31 \text{ S.D.}, 0.92 \pm 0.27 \text{ S.D. and} 0.84 \pm 0.29 \text{ S.D. mg/dl} respectively.

Studies were also made on separate groups of new untreated cases of carcinoma and leukemia. In these cases also the mean blood AA were low, namely 0.28 \pm 0.27 \text{ S.D. and} 0.18 \pm 0.15 \text{ S.D. mg/dl} respectively. The blood DHA levels in the said groups of carcinoma and leukemia were 0.47 \pm 0.38 \text{ S.D. and} 0.44 \pm 0.29 \text{ S.D. mg/dl} respectively.
Pregnancy during the onset of labour is an acute stressful condition. Studies were made on a group of such patients. In these cases also the blood AA level was very low, the mean value being 0.08 ± 0.07 S.D. mg/dl. The blood DHA level was moderately high 0.41 ± 0.23 S.D. mg/dl. Similar low blood AA level and high DHA level were observed in a group of patients suffering from mental derangements (emotional stress). The values were: AA, trace and DHA 0.40 ± 0.13 S.D. mg/dl.

It was observed that after recovery from trauma, blood DHA level practically disappeared and blood or plasma AA level rose to normal value. This was demonstrated in a group of surgical patients by examining their blood before surgery, immediately after surgery and after recovery from surgery. The corresponding mean blood AA values were 0.51 ± 0.13 S.D., 0.15 ± 0.14 S.D., and 0.51 ± 0.16 S.D. mg/dl respectively, and the mean DHA values were 0.05 ± 0.04 S.D., 0.85 ± 0.31 S.D. and 0.07 ± 0.06 S.D. mg/dl respectively. In a similar study it was revealed that plasma AA reappeared and returned to normal level after recovery from surgical stress. The mean plasma AA values before surgery, immediately after surgery and after recovery from surgery were 0.38 ± 0.06 S.D., 0.05 ± 0.07 S.D., 0.41 ± 0.07 S.D. mg/dl respectively.

It was considered that the decrease in the plasma AA level and increase in blood DHA level might be due to (i) increased turnover of AA to DHA and/or (ii) decreased rate of
reduction of DHA to AA. However, the rate of reduction of DHA in the erythrocytes from trauma patients was similar to that from normal subjects. This would indicate that the accumulation of DHA was not due to lack of reduction of DHA to AA. The reduction of DHA in human erythrocytes is carried by reduced glutathione (GSH) and this reduction is dependent on erythrocyte glucose-6-phosphate dehydrogenase (G6PD EC 1.1.1.49) and glutathione reductase (GR EC 1.6.4.2) activities as indicated in the scheme shown below:

![Scheme 1: Schematic representation showing the mechanism of DHA reduction in human erythrocytes.](image-url)
It was observed that the erythrocyte GSH levels and the G6PD activities were similar in normal and traumatic blood. The GR activity was, however, significantly higher ($P < 0.001$) in trauma than that of normal subjects. The reason for this was not clear.

It would, therefore, appear that the accumulation of DHA in traumatic blood was a result of increased turnover of AA to DHA. Increased turnover of AA to DHA in trauma was supported by the results obtained after a single administration of 500 mg AA (i.v.) to traumatic subjects. The blood and plasma AA and DHA levels were estimated and the results were compared with that obtained from normal volunteers under similar conditions. The blood and plasma AA and DHA levels, twentyfour hours after AA administration ($n = 8$), were in mg/dl: blood, AA $0.16 \pm 0.06$ S.D., DHA $0.89 \pm 0.24$ S.D., plasma, AA $0.15 \pm 0.07$ S.D., DHA $0.17 \pm 0.04$ S.D. The corresponding values from normal volunteers ($n = 6$) were in mg/dl, blood, AA $1.26 \pm 0.23$ S.D., DHA $0.12 \pm 0.07$ S.D., plasma, AA $1.07 \pm 0.20$ S.D., DHA $0.06 \pm 0.01$ S.D.

In case of a high turnover of AA to DHA one would expect a high DHA level in the plasma of traumatic patients. Though the plasma DHA level in trauma was significantly higher ($P \ll 0.001$) than that of normal subjects, the mean value was relatively low ($0.17 \pm 0.06$ S.D. mg/dl). This is apparently
because the erythrocytes have no permeability barrier to DHA and that the uptake of DHA by the erythrocytes is very rapid in normal or traumatic subjects. This would explain that even when the turnover of AA to DHA was high in stress condition, plasma DHA level would remain low.

It was observed that high blood DHA and low plasma AA level were also notable features in diabetes mellitus irrespective of age, sex or duration of the disease. Thus, there is a similarity between diabetes and stress conditions in regard to the blood and plasma DHA and AA status. Whereas in stress conditions blood DHA disappears and plasma AA reappears after recovery, the high blood DHA and low plasma AA remain persistent in diabetes. It was further observed that in diabetes mellitus, as found in trauma, the high blood DHA and low plasma AA levels were not due lack of reduction of DHA to AA but due to high turnover of AA to DHA in the body. The high turnover of AA was apparently due to increased rate of oxidation of AA to DHA in diabetic tissues, because oxidation of AA was markedly increased in streptozotocin induced diabetic rats.