RAPID DETERMINATION OF FREE ERYTHROCYTE PORPHYRIN AND MEAN CORPUSCULAR VOLUME IN THE SCREENING DETECTION OF IRON DEFICIENCY AND THALASSEMIA TRAIT. James A. Stockman, Lawrence S. Weiner, Marie J. Stuart and Frank A. Oski. State University of New York, Upstate Medical Center, Syracuse, New York.

Free erythrocyte porphyrin (FEP) levels and red cell indices (Coalter Counter Model S) were determined in 70 normal subjects, 52 with iron deficiency and 29 with thalassemia trait in order to determine if these procedures were suitable as screening techniques for the detection and initial diagnosis of iron deficiency and thalassemia minor in subjects with microcytosis. FEP levels were increased in 90.2% of subjects with iron deficiency (mean 167 mcg/100 ml RBC's; range 18-700), while in 96.6% of subjects with thalassemia trait, FEP levels were normal (less than 70 mcg/100 ml RBC's). Mean FEP values increased slightly as transferrin saturations decreased but became abnormally elevated when the transferrin saturation was less than 15%. Although subjects with iron deficiency exhibited mean corpuscular volumes in the range 46-84 μ^3, subjects with beta thalassemia trait had values of 70 μ^3 or less (mean 62 μ^3; range 53-70). All non-thalassemic subjects with mean corpuscular volumes less than 70 μ^3 had transferrin saturations of less than 15%. In patients with microcytosis, the combined use of the electronic determination of mean corpuscular volume and rapid assay of FEP should be suitable for initial separation of patients with iron deficiency from those with thalassemia minor.

URBAN HOSPITALIZATION COST ANALYSIS OF PATIENTS WITH SICKLE CELL DISEASE

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A sample, from a 2 year period (1973-73), of hospital records for 57 patients with sickle cell anemia were examined to determine the cost of hospitalization and treatment to assess medical expenses and to obtain a tentative profile of age, length of stay, and type of services utilized. The average age of patients was 16.7 years (R:5 mos.-39 yrs.). Length of stay averaged 13.8 days (R:1-196 days). Breakdown of data per hospitalization is presented as averages: age (N=57) 16.7 yrs., days in hospital (N=57) 13.77, room and board (N=57) $474.73, operating-delivery room (N=2) $96.25, anesthesia (N=4) $38.13, x-ray (N=39) $53.90, laboratory (N=57) $166.13, EKG (N=20) $20.10, physical therapy (N=16) $325.71, med.-surg. supplies (N=51) $34.13, pharmacy (N=48) $32.79, inhalation therapy (N=5) $743.74, I.V. (N=15) $41.71, misc. (N=14) $46.63; total: (N=57) $876.40.

In terms of age categories, the 0-11 year old group average hospital costs were nearly half the cost of the 20-39 year old group. Approximately 50% of the payments were made by health insurance companies, 31% by Medicaid, and 17% by private means.

This information can be utilized in the comprehensive planning and management of patients with sickle cell anemia.

VINCRISTINE (VCR) THERAPY AND INAPPROPRIATE ANTIDIURETIC HORMONE SECRETION. Marie J. Stuart, Myron Miller and James A. Stockman, III (Intr. by Frank A. Oski). Depts. of Pediatrics and Medicine, Upstate Medical Center and the Veterans Administration Hospital, Syracuse, New York.

Isolated examples of inappropriate ADH secretion following VCR therapy have been described. Stimulated by the observation of 3 separate episodes of inappropriate ADH secretion in a patient receiving VCR for acute lymphatic leukemia (ALL), an additional 6 patients with ALL were studied sequentially during initial remission induction. Patients received VCR (2 mg/M^2) weekly x 3 doses and prednisone (40 mg/M^2) daily for a period of 28 days while daily 24-hour urines were collected for ADH immunoassays. In 6 of 7 patients, significant increases in ADH secretion occurred during therapy. The greatest rise was usually seen in the 7th and 8th days following VCR, averaging 18.1 and 14.6 units/M^2/day (normal baseline 3.1/M^2/day), although in 3 of the 7 patients some increase in ADH secretion was also noted on day 3 and 4 post-VCR. In the index patient, ADH secretion reached a value of 283 units/M^2/day and was associated with profound hyponatremia and seizures. The remainder of the patients remained asymptomatic and isosmotic. Increased ADH secretion appears to be a common occurrence following VCR administration. It should be anticipated in order to avoid complications.

ERYTHROPOIETIN, GROWTH HORMONE AND GROWTH. Leonard F. Vitale, T. Donald Eisenstein and N. Shah, Dept of Ped., New Jersey Med. Sch., Newark, N.J. Jorge A. Ortega, Sch. of Med., Univ. of S. Cal.

(Intr. by Franklin C. Behrle)

Growth Hormone (GH) has been shown to stimulate erythropoiesis and increase red cell volume (RCV) in children and animals who had either isolated GH deficiency or surgical hypophysectomy. In an attempt to confirm that this effect is also related to the growth response of this hormone we studied 7 GH deficient children. 6 of the children had reduced RCV (mean 15.5 $\pm$ .8 ml/kg). The 7th (AK) had a normal RCV of 22 ml/kg (N 22-27 ml/kg). The mean erythropoietin level in the 6 children who had reduced RCV was .6 $\pm$ .1 % RBC^{59}Fe Inc. After 6 weeks of GH the RCV was 23.7 $\pm$ 1.2 ml/kg. The erythropoietin increased to a value of 3.2 $\pm$ .6 %RBC^{59}Fe after 1 week of therapy in the 6 children with reduced RCV and decreased to 2.0 $\pm$ .4 % RBC^{59}Fe after 6 weeks of GH therapy. The growth of these 6 patients was 3.5 $\pm$ .25 inches for the 1st year. AK did not increase her erythropoietin during therapy. Her growth was 1.25 inches during her 1st year of GH. Her GH assays were repeated and it was again decreased (less than 1 nanogram/ml by insulin tolerance test).

We believe that the erythropoiesis that occurs after GH administration in a GH deficient child with a reduced RCV is related to erythropoietin secretion and this response maybe related to the hormones'growth effect.

ASPIRIN INDUCED INHIBITION OF PLATELET LIPID PEROXIDATION. Marie J. Stuart, Joan R. Urmson, James A. Stockman and Frank A. Oski. State University of New York, Upstate Medical Center, Syracuse, New York.

Platelet aggregation is accompanied by lipid peroxidation (LP) when agents such as N-ethyl malimide (NEM), thrombin or epinephrine are employed as aggregating agents. We have found that aspirin (ASA) inhibits lipid peroxidation, as measured by thiobarbituric acid, both in vitro and in vivo. Eight subjects were studied for 10 days after ingestion of 600 mg of ASA. Results of LP (µmoles malonaldehyde/10^9 platelets) were:

Agent	Day 0	1	2	4	6	8	10
NEM (.001 M)	.83	.11	.19	.35	.60	.72	.84
Thromb. (.5 U/ml)	.30	.03	.04	.11	.12	.25	.28
Epineph. (50 µM)	.16	.01	.07	.09	.08	.10	.18

Results demonstrate that LP was significantly reduced post ASA ingestion, and gradually returned to normal in 10 days. Concomitant platelet aggregation studies returned to normal after 4 days with epinephrine and by day 8 with thrombin. The inhibitory effect of ASA on platelet LP parallels its effect on aggregation but is a more sensitive index of the "aspirin defect" as it remains present for the entire lifespan of the platelet. These findings suggest an interaction between ASA and platelet membrane sulhydryl groups in the mechanism of aggregation.

SECOND PRIMARY TUMORS IN CHILDREN WITH CANCER AND LEUKEMIA. Jaw J. Wang, Juliet Hananian and Lucius F. Sinks. Roswell Park Memorial Institute (RPMI), Buffalo, New York and University of Miami, Miami, Florida.

Children who have cancer or leukemia usually have an abbreviated life span in which another tumor could manifest itself as compared to an adult population. This study includes a survey of children with leukemia or cancer below 16 years of age seen from June 1946 to December 1971 at RPMI. Sixteen of 884 cases (1.8%) showed the presence of a second primary tumor (SPT). There were 12 malignant and 4 benign lesions. Among 371 cases of acute leukemia, only 1 SPT was found. Three cases of SPT, including one each of Hodgkin's Disease, fibrosarcoma and osteosarcoma were observed in retinoblastoma patients. Among 41 cases of Wilm's Tumor, 2 SPT, including one chondrosarcoma and one osteochondroma were found. One patient with Hodgkin's Disease and chronic myelocytic leukemia had Positive Philadelphia Chromosome. One patient with xerodermia developed both squamous cell carcinoma and malignant melanoma. Two children with glioblastoma multiforme and rhabdomyosarcoma developed hepatoblastoma. A neuro-astrocytoma was observed in one child with acute leukemia and in one with adenocarcinoma of the colon. One child, who was cured of glioblastoma multiforme, developed reticulum cell sarcoma. In 7 children the SPT were detected at the time of diagnosis of the primary malignancy. In 4 cases the SPT occurred 2.5-16 years after radiotherapy and were possibly radiation induced.