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Titles and Selected Abstracts

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Recent progress in osteomalacia and rickets is reviewed. Tetracycline-based studies of mineralization and remodeling dynamics have been useful in approaching a quantitative definition of osteomalacia which is primarily a disorder of bone mineralization. The diagnosis of osteomalacia can be difficult because clinical findings may be minimal and available laboratory tests normal. The diagnostic utility and relative simplicity of the tetracycline—labeled rib biopsy is emphasized. Current basic information concerning vitamin D and the physiology of parathyroid hormone in osteomalacia and rickets is discussed. Evidence favors a direct effect of vitamin D on bone in addition to its intestinal action. Data concerning the question of the dependence of the skeletal action of parathyroid hormone upon vitamin D is conflicting and this issue is not settled. The major clinical forms of osteomalacia and rickets include primary vitamin D deficiency, gastrointestinal malabsorption, primary hypophosphatemia, renal failure, renal tubular disorders and hypophosphatasia. Important pathogenic factors are lack of vitamin D, phosphate deficiency, azotemia and, possibly, metabolic acidosis and hyperparathyroidism. Osteomalacia can be accompanied by osteoporosis, osteitis fibrosa cystica and osteosclerosis. Treatment is determined by the clinical form but generally requires vitamin D along with other measures.


The description in 1924 of chronic rheumatoid arthritis, splenomegaly and neutropenia represented a specific type of arthritis in combination with a secondary hypersplenic state. Depression of circulating neutrophils may impair the patient’s resistance to infection. Treatment of hypersplenism has consisted of adrenal corticosteroids or splenectomy. Six patients with Felty’s syndrome are reported who underwent splenectomy. Four of the six patients presented with severe infection and the spleen was palpable in all six. All patients after splenectomy demonstrated an increase in the blood leukocytes and neutrophils. Mature neutrophils became apparent in the postoperative bone marrow, whereas preoperative bone marrow examination showed immature forms. The blood neutrophils increased normally during infection in two patients who developed cellulitis and pneumonia four years after splenectomy.
Abstracts


Metabolically normal individuals exhibit different, age dependent remodeling rates and balances at the periosteal, haversian canal, and trabecular-endosteal bone surfaces. During life at these surfaces there is: (1) a slightly positive periosteal balance, (2) an essentially zero haversian canal balance, and (3) a negative trabecular-endosteal balance. This overall balance pattern leads to a net loss (more resorption than formation) of both cortical and trabecular bone with aging. In osteoporosis this pattern of bone loss is accelerated. The patient reported exhibited in the skeleton about 1.0% fluoride in the dry, fat-free ashed bone, which is equivalent to 10,000 ppm fluoride. The overall remodeling rate was increased when compared to the normal. In addition, balance data suggested a trend toward a positive endosteal-trabecular bone surface balance. Such a favorable balance might prevent and/or "counteract" the unfavorable bone loss that occurs in metabolically normal individuals and in patients with osteoporosis.


The sudden unexpected deaths of two young athletes and the findings in their hearts at necropsy are described. Based on the histopathology in and around the sinus node, which was the same in both hearts, a terminal cardiac arrhythmia is postulated as the mechanism of death. Ways in which ischemic pathology in and near the normal cardiac pacemaker may lead to a lethal arrhythmia are discussed. Stress is placed on the importance of considering a cardiac arrhythmia when examining athletes who have suddenly collapsed, since the present observations indicate that appropriate treatment should produce complete recovery.


Diseases which may damage and occlude small coronary arteries are reviewed with classification into three groups: embolic, inflammatory and noninflammatory. In the ventricular myocardium such diseases produce focal degeneration and fibrosis, ultimately leading to cardiac enlargement and failure. When the small arteries normally supplying the cardiac conduction system are involved, the consequences are arrhythmias, conduction disturbances, syncopal attacks and frequently sudden death. Many examples of obscure myocardiopathy may be due to hereditary medial necrosis of the small coronary arteries. The clinical significance of pathology of the coronary arteries, including its possible role in a limited number of examples of angina pectoris, is discussed.


The adverse effect of continuously administered corticosteroids on the retention of nitrogen is established. The effect on the calcium balance has not been consistent. In Harrison's isolated loop studies calcium diffusion across the intestinal mucosa was blocked by corticosteroids. Balance studies were done on eleven children with nephrosis and chronic glomerulonephritis. The interrupted corticosteroid therapy was administered in a single dosage every 48 hours (50-60 mg prednisone) or 40-60 mg prednisone for three days a week. A positive balance was found in all except one patient with glomerulonephritis.


This study tested the use of Micropore tubes to repair crushed and severed nerves and neurolysis of severed nerves six weeks after injury. Thirty nerves in dogs were injured. Fifteen were severed and fifteen were crushed. Bone was placed to obstruct the pathway of nerve regeneration. Five severed and five crushed nerves were decompressed. Five severed and five crushed nerves were treated by resecting the area of injury and bridging the gap with Micropore. Six months later the number of regenerated axons in a standard area of the distal nerve were counted and compared. Five uninjured nerves were used as controls. The average number of regenerated axons were: 1,009 per decompressed crushed nerve, 833 per untreated crushed nerve, 622 per crushed nerve repaired with Micropore tube, 540 per severed nerve with Micropore repair, 186 per decompressed severed nerve, and 108 per untreated severed nerve. The average number of axons in the controls was 1,221. It was concluded that early decompression of crushed nerves is superior to bridging with Micropore. Injured nerves do not regain their normal number of axons in six months.
Abstracts


The study of the dynamics of the local cellular response to an inflammatory stimulus in vivo demonstrated a fault which may explain, in part, the increased susceptibility of diabetics to local and systemic infection. In non-diabetics a prompt response to the inflammatory stimulus by neutrophils is followed by transfer of their intracytoplasmic glycogen to mononuclear cells, the latter appearing 6-9 hours after the neutrophilic exudation. In the patient with catabolic diabetes (ketoacidosis), delay or stagnation of this process occurs; neutrophils become choked with glycogen and fail to transfer it to the juxtapositioned mononuclear cells. Failure of glycogen transfer prevents mononuclear energization, impedes neutrophilic phagocytosis and impairs local host defense. Following appropriate correction of the catabolic state in one diabetic, re-examination showed correction of the previously defined defect. Neutrophils from diabetics with lesser degrees of metabolic derangement transferred glycogen comparable with the non-diabetic. One may infer from this study that "normalization" of these local intercellular responses (promotion of glycogen transfer) by good metabolic control of the diabetes improves the reaction to local inflammation and heightens host defense.


The drug, an analogue of chlordiazepoxide, was administered to 18 patients suffering from anxiety and depression for 3 to 18 months. They had not responded to previous therapy for a period of years. Fifteen were definitely improved: nine became free of depression and three were freed of anxiety. Side effects were minimal. This drug may prove a useful compound in treating anxious depressions.


Forty-five patients with presumed liver disease were examined. The use of both techniques was most successful in the detection of cancer metastatic to the liver. Laparoscopy proved to be the more definitive approach to the diagnosis of cirrhosis, and no supporting documentation by photoscan was required. The diagnosis of liver abscess is best established by means of the photoscan.


Studies are reported of 10 children having physical findings previously reported in cases classified as showing cerebral gigantism. These findings included mental retardation; advanced height, weight, and bone age; and a characteristic facies. Six had pneumoencephalography and all showed communicating hydrocephalus. Karyograms were normal. Urinary 17-KS steroid excretion was increased for chronologic age, but levels were compatible with physiologic age. Adrenal response to dexamethasone suppression and metapyrone administration was normal. The condition is regarded as a reflection of congenital central nervous system dysfunction with mental retardation and altered hypothalamic control of anterior pituitary function.


The authors defined the syndrome as a generalized impairment of tissue perfusion which was due to reduced myocardial contractility and not the results of hypovolemia, pericardial tamponade, arrhythmias, digitalis intoxication, electrolyte imbalance or hypoxia due to hypoventilation. Sixteen patients who died after aortic, subaortic or mitral valve surgery with the signs of low cardiac output were studied. Postmortem coronary arteriograms and special microscopic evaluations were performed. Microinfarctions were found in all 16 hearts and appeared to explain the mechanism of impaired contractility. The microinfarcts were distributed in a patchy manner and showed very early signs of necrosis. Histochemical
stains confirmed the presence of early infarction which did not resemble changes as seen in arteriosclerotic heart disease. The coronary arteriograms showed a normal patent major coronary vasculature. Impaired coronary perfusion, air emboli and blood platelet aggregates produced by the pump oxygenator were believed to be responsible for microinfarctions. Measures to prevent or minimize those factors are summarized and the treatment of low cardiac output syndrome is discussed.


A morphologic study was performed with electron microscopy and a shadow casting technique on the normally elliptical erythrocytes of the llama and on elliptocytes from patients with hereditary elliptocytosis (seven heterozygotes and one homozygote). These studies demonstrated a previously unrecognized enlargement at each pole of the human elliptocyte in contrast to the physiologic llama elliptocyte which did not show this change. Further observation suggested that this polar aggregation in the human elliptocyte was due to hemoglobin massing; the polar areas of the elliptocyte were also shown to be resistant to crenation under appropriate conditions. After lysis of human elliptocytes, the ghost cell retains its elliptocytic outline, unlike the sickle cell ghost which reverts to a disc shape. This implies that one basic defect in hereditary elliptocytosis resides in the cell membrane, and that the further aberration in shape (bipolar aggregation) is the result of hemoglobin massing.


Enamel hypoplasia and hypomaturation may result from direct disturbances of metabolism of the ameloblastic layer of the enamel organ. The specific etiologic agents or events responsible include hereditary factors, systemic disease, tetracycline administration or mechanical trauma. The case reported is one in which mechanical trauma to a primary incisor tooth resulted in areas of enamel hypomaturation and enamel hypoplasia on the succeeding permanent tooth. From the position of the damaged area of enamel on the surface of the tooth crown it was possible to correlate in time the specific event which resulted in the enamel damage.

