Selected Abstracts

Lactoferrin deficiency as a consequence of a lack of specific granules in neutrophils from a patient with recurrent infections. Janine Breton-Gorius, Ph.D. David Y. Mason, MD, Diego Buriot, MD, Jean-Louis Vilde, MD, and Claude Griscelli, MD. American Association of Pathologists. The American Journal of Pathology, Vol. 99, May 1980, pp. 413-428.

Neutrophils from a body suffering from recurrent infections were found to be totally deficient in specific granules when studied by electron microscopy. In contrast, myeloperox-idase-containing azurophil granules were increased in number. This deficiency of specific granules could be detected at the light-microscopic level using an immunocytochemical technique to demonstrate the absence of lactoferrin. Neutrophils also exhibited abnormal nuclear segmentation, nuclear clefts, an abnormally weak cyto-chemical reaction for alkaline phosphatase, and an increased number of mitochondria and ribo-somes. Some granulocytic precursors were abnormal, and many of these cells were phago-cytosed by macrophages in the bone marrow. Despite these multiple abnormalities and the history of severe pyogenic infection, the in vitro bactericidal capacity of the neutrophils was within normal limits, and normal degranulation of azurophil granules occurred following phagocytosis. The precise mechanism by which the deficiency of specific granules in this patient led to an enhanced in vivo susceptibility to infection therefore remains obscure. However, attention is drawn to the fact that in three previously described cases of specific granule deficiency history of recurrent infections was present.

Eosinophilia in the hospitalized neonate. Robert Lawrence, jr. MD. Joseph A. Church, MD. Warren Richards, MD. and Allen 1. Lipsey, MD. American College of Allergists Annals of Allergy, Vol. 44, June 1980, pp. 349-352.

The incidence of and clinical associations with eosinophilia in a cross-section of hospitalized newborns had not been studied previously. The medical records of 200 such infants less than 29 days of age were reviewed. Total eosinophil counts were calculated and associations with a variety of clinical factors were examined. Eosinophilia (adjusted for day of age) occurred at least once in 22% of the infants studied. No significant skew could be detected in the age of onset of eosinophilia. Statistical analysis revealed eosinophilia to be significantly associated ($p<.05$) with length of hospitalization, number of days of antibiotic therapy, and use of paren- teral alimentation. A trend toward eosinophilia was noted in lower birth weight infants ($0.05< p<0.10$) .Eosinophilia was not associated with sex, age, Apgar score at birth, cesarean section delivery, transfusions, phototherapy, specific diagnoses, or type of oral feeding. Multiple regression analysis showed a highly positive correlation of associated factors with hospitalized days as the dependent variable. Eosinophilia appears to be a nonspecific finding in sick neonates and is related to the severity of illness and the number of diagnostic or therapeutic procedures performed.

Scoliosis in symptomatic spondylolisthesis.

I.B. McPhee, FRACS and J.P. O'Brien, Ph.D. The Journal of Bone and Joint Surgery Incorporated. The Journal of Bone and foint Surgery, Vol. 62-B, May 1980, pp. 155-157.

The association between spondylolisthesis and scoliosis was studied in 84 patients who presented during a 30-year period with symptomatic spondylolisthesis. The incidence of scoliosis was 42%, the majority of cases being lumbar or thoracolumbar curves of less than 15°. The incidence was highest in the group of patients with spondylolisthesis at L4-5 where all except one had scoliosis. Scoliosis was present in 47% of patients with dysplastic spondylolisthesis at the lumbosacral junction; in this group, the incidence of scoliosis was greater where the displacement exceeded 25%. The lowest incidence (25%) was found in the group with isthmic spondylolisthesis at the lumbosacral, junction. There appeared to be no relationship between excessive lumbar lordosis or tightness of the hamstrings and

scoliosis.

Epidemiology as a guide to clinical decisions: the associations between triglyceride and coronary heart disease. Stephen B. Hulley, MD, MPH, Ray H. Rosenman, MD Richard D. Bawol, Ph.D. and Richard J. Brand, Ph.D. Massachusetts Medical Society. The New England Journal of Medicine, Vol. 302, June 19, 1980, pp. 1383-1389.

The hypothesis that triglyceride is a cause of coronary heart disease, although unconfirmed and never universally accepted, has nonetheless strongly influenced the practice of preventive medicine. The authors have examined the epidemiologic association between triglyceride and coronary heart disease to evaluate the validity of inferring that there is causal relation between the two. Neither the evidence from published studies nor an analysis of data from the Western Collaborative Group Study provides strong support for the causal hypothesis. Information from other scientific disciplines is also meager, contrasting with the coherence of diverse evidence supporting the hypothesis that cholesterol is a cause of coronary heart disease. These arguments fall short of disproving the belief that lowering triglyceride will prevent coronary heart disease, especially since triglyceride and cholesterol are inextricably associated through mutual lipoprotein carriers. The authors propose that the ethics of preventive medicine place the burden of proof on the proponents of intervention and recommend that widespread screening and treatment of healthy persons for hypertriglyceridemia be abandoned until more persuasive evidence becomes available.