$P\bar{v},O_2$ was calculated from these n values, assuming an arteriovenous difference of 5 ml of O_2/100 ml of blood. The calculated $P\bar{v},O_2$ fell from 38·5 mmHg to 27 mmHg as COHb concentration rose to 18–20% in normal subjects. In normal subjects and bronchitic patients with different levels of Pa,O_2 the calculated relationship between n and $P\bar{v},O_2$, for Pa,O_2, showed that as n changed from 2·65 to 1·70, the $P\bar{v},O_2$ fell by approximately 11 mmHg in two normal subjects (Pa,O_2 100 mmHg), 7·5 mmHg in patients 1, 2 and 3 (mean Pa,O_2 73 mmHg) and 5 mmHg in patients 4 and 5 (mean Pa,O_2 62 mmHg). Thus carbon monoxide, at the levels often found in bronchitic patients who smoke cigarettes, decreased the Hill coefficient to the same extent as found *in vitro*, resulting in a reduction of $P\bar{v},O_2$.

Poster Communications

61. ULTRASOUND IMAGING IN THE DIAGNOSIS OF MUSCLE DISEASE

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Diagnostic pulse echo ultrasound imaging has recently been described as a method of visualizing pathological change in dystrophic muscle (Heckmatt, Dubowitz & Leeman, 1980, *Lancet*, **i**, 1389–1390). A study is now under way to establish the possible value of the technique as a diagnostic tool in muscle disorders of childhood.

A comparison has been made of the ultrasound appearances of the thigh muscles in 60 normal children, 13 with spinal muscular atrophy, 13 with progressive muscular dystrophy and four with congenital muscular dystrophy. This last-named disease, though showing dystrophic change in the muscle, is non-progressive. The children with spinal atrophy and muscular dystrophy were examined at the time of the diagnostic muscle biopsy. Transverse and longitudinal static 'B' scan ultrasound pictures of the thigh were obtained with a 5 mHz probe.

In normal children three anatomical landmarks, bone, fascia lata and the outer boundary of vastus intermedius, were always clearly seen. In the children with dystrophy and spinal atrophy the ultrasound appearances were altered. In both, increased echo from within the muscle was seen with a corresponding reduction in bone echo. In addition, in spinal muscular atrophy the degree of muscle atrophy was sometimes striking.

As a diagnostic screening tool, ultrasound is of less value than the serum CPK in the early diagnosis of the progressive forms of dystrophy. Ultrasound is potentially of greater value in the diagnosis of spinal muscular atrophy and congenital muscular dystrophy.

62. ENZYMIC ANALYSIS OF HUMAN SKELETAL MUSCLE BIOPSY SAMPLES

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Although there have been many studies of the concentration of various metabolites in needle-biopsy specimens of human skeletal muscle (Edwards *et al.*, 1980, *New England Journal of Medicine*, **302**, 261), little is known of the quantitative alteration in marker enzymes for the various subcellular organelles of these cells. We have combined subcellular fractionation of muscle biopsy homogenates with microassay of organelle marker enzymes in order to investigate the cell pathology (Peters, 1977, *Clinical Science and Molecular Medicine*, **53**, 503) of certain muscular disorders.

The assay conditions were optimized for the following organelle marker enzymes: lactate dehydrogenase (cytosol); 5′-nucleotidase (sarcolemma); *N*-acetyl-β-glucosaminidase, acid phosphatase (lysosomes); catalase (peroxisomes); neutral α-glucosidase (sarcoplasmic reticulum); glutamate, succinate and malate dehydrogenases (mitochondria). Clear resolution of the various organelles was obtained by sucrose density gradient centrifugation and the distribution of Mg^{2+}-dependent, Ca^{2+}-dependent and Na^+,K^+-activated, Mg^{2+}-dependent adenosine triphosphatases were determined. The centrifugation properties and enzyme activities of organelles from control tissue were determined and their alterations in alcoholic myopathy will be reported.

63. CEREBRAL BLOOD FLOW AND OXYGEN AND GLUCOSE UTILIZATION IN THE NEONATAL PIGLET

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Preliminary investigations indicate that the neonatal piglet is a suitable model for the study of glucose metabolism in the perinatal period (Flecknell, Wootton & John, 1980, *British Journal of Nutrition*, **44**, 193–203). Since the brain is probably the major consumer of glucose in the human neonate, attempts to study glucose homeostasis are facilitated by measurement of cerebral metabolism. We have therefore developed a method for measuring cerebral blood flow, simultaneously with cerebral oxygen and glucose consumption rates, in the conscious, unrestrained, newborn piglet.

Blood flow was measured by a modified Kety–Schmidt technique with ^{125}I-labelled iodoantipyrene as the tracer. Mixed cerebral venous blood was obtained from a catheter positioned in the posterior third of the sagittal sinus and arterial blood from a catheter in the femoral artery. Blood samples were withdrawn with modified portable syringe pumps (Pye Dynamics MS16).

In nine normal piglets aged between 4 h and 5 days, cerebral perfusion rate was 90 ± 22 ml min^{-1} 100 g^{-1} (mean ± SD), oxygen consumption 320 ± 150 μmol min^{-1} 100 g^{-1} and glucose consumption 41 ± 25 μmol min^{-1} 100 g^{-1}. The glucose/oxygen ratio was $0{\cdot}83 \pm 0{\cdot}16$ and brain weight $36{\cdot}0 \pm 0{\cdot}6$ g.

Although there appears to be no comparable information from unanaesthetized normal human neonates, our results are similar to the few measurements which have been made in anaesthetized children. For example, Settergren *et al.* (1976, *Acta Paediatrica Scandinavica*, **65**, 343–353) reported cerebral blood flow as 69 ± 24 ml min^{-1} 100 g^{-1} and the glucose utilization rate as $27{\cdot}2 \pm 18{\cdot}6$ μmol min^{-1} 100 g^{-1}. These results suggest that the piglet may be a useful animal model for the investigation of neonatal cerebral metabolism, especially in view of the ethical constraints attached to investigations in children.

64. ALANINE DISAPPEARANCE RATE DURING PROLONGED STARVATION IN OBESE SUBJECTS

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Alanine is an amino acid released from muscle to be taken up by the liver, where it is an important contributor to gluconeogenesis. During starvation the concentration of alanine in blood decreases and this could be due to decreased production of alanine from muscle or increased removal by liver or to combination of these. In an attempt to distinguish between these possibilities we have studied the disappearance rate of intravenously injected alanine in starved obese patients.

Six obese females were studied on three occasions: (a) when they were on a 400 kcal diet, (b) after total starvation for 5 days and (c) starvation for 28 days. After taking control blood samples 112 mmol of alanine was injected intravenously and blood concentration of alanine was measured over the next 60 min. The half-life of the injected alanine ($t_{0{\cdot}5}$) was calculated.

The basal concentration of alanine decreased from 387 ± 34