

that subclinical hypothyroidism occurs in 5% to 10% of elderly subjects, and is especially prevalent in elderly women. Subclinical hyperthyroidism is less common, affecting less than 2% of the elderly population. Potential risks of subclinical hypothyroidism in the elderly include progression to overt hypothyroidism, cardiovascular effects, hyperlipidemia, neurological and neuropsychiatric effects. Potential risks of subclinical hyperthyroidism in the elderly include progression to overt hyperthyroidism, cardiovascular effects (especially atrial fibrillation), and osteoporosis. This has not been well studied in geriatric patients.

Methods: A retrospective review and audit of the files from the Geriatric clinic, Universitas Hospital, Bloemfontein between January 1999 and July 2003. Only first admissions were included. Patients were grouped according to their TSH and FT4. Normal TSH=0.35-5.55mIU/l, FT4=11.5-23.2pmol/l.

Results: The total number of patients admitted were 791 (mean age in years =81). Gender: Female 68%, male 32%. Age distribution in years: 65-74(16%), 75-84 (56%), 85+(28%). The following groups were identified after thyroid function tests were done on 488 patients. Of these 58% were normal 4% Rare (normal TSH + increased FT4, not on thyroid replacement therapy), 0.8% Hyperthyroidism, 3.5% Subclinical hyperthyroidism, 27% Hypothyroidism (including patients on Eltroxin), 6% Subclinical Hypothyroidism and 0.2% Non Thyroidal Illness (NTI). Most common admitting diagnosis were delirium, heart failure, pneumonia, hypertension, anemia, dyspnea. Mortality rate for the total group 131/791=17%. Mortality rate % of 488 : normal group 8.6%, Rare group 0.4%, Hyperthyroidism 0.2%, Subclinical Hyperthyroidism 0.4%, Hypothyroidism 3.3%, Subclinical hypothyroidism 1.8%, NTI 0.2%.

Conclusions: We found that thyroid disease, overt and subclinical is common in hospitalized geriatric patients. Follow up is the appropriate gold standard in diagnosis of thyroid disease in hospitalized patients after resolution of the acute illness. However the decision to treat geriatric patients with isolated abnormalities in serum TSH and FT4 levels is complex and requires assessment of an individual patient's risks and benefits with treatment. This study represents a valuable contribution to geriatric world literature.

Cellular and Molecular Changes to Normal and Wounded Human Skin Fibroblasts following He-Ne Laser Irradiation – an *In Vitro* Study.

D Hawkins, and H Abrahamse.

Faculty of Health Sciences, University of Johannesburg.

Low Level Laser Therapy (LLLT) is a form of phototherapy used to promote wound healing in different clinical conditions. Laser therapy has the capability to inhibit or stimulate cellular activity in the absence of significant heating (Pinheiro et al.,1995). The unique properties of laser create an enormous potential for specific therapy of skin diseases (Walsh et al., 1997).

This study aimed to establish cellular and molecular responses of normal and wounded human skin fibroblasts to helium-neon (632.8nm) laser irradiation following a single dose 0.5J/cm², 2.5J/cm², 5J/cm², 10J/cm² and 16J/cm² on two consecutive days. Changes in normal and wounded fibroblast cell morphology (chemotaxis and haptotaxis) were evaluated by light microscopy. Cellular responses were evaluated using the ATP cell viability assay, the ALP cell proliferation assay, LDH membrane integrity assay while genetic integrity was evaluated using the Comet assay for DNA damage.

Morphologically, wounded cells exposed to 5J/cm² migrate rapidly across the wound margin indicating a stimulatory effect of LLLT. A dose of 5J/cm² has a stimulatory effect on wounded fibroblasts with an increase in cell proliferation and cell viability without adversely increasing the amount of cellular and molecular damage. Higher doses (10J/cm² and 16J/cm²) were characterized by a decrease in cell viability and cell proliferation with a significant amount of damage to the cell membrane and DNA. Results show that 5J/cm² stimulates mitochondrial activity, which leads to normalization of cell function with an increase in cell

viability, proliferation and migration of wounded fibroblasts to accelerate wound closure. Laser irradiation can modify cellular processes in a wavelength dependent manner.

A Suspected Case of Familial Benign Hypocalcaemic Hypercalcaemia (FBHH)

M du Plessis, EC van den Berg.

Du Buisson, Bruinette and Kramer Pathologists, Private Physician Zuid Afrikaans Hospital.

Background: An 18 year old patient presented to her GP with non-specific symptoms of fatigue, bone and back pain. Initial biochemical investigations were suggestive of primary hyperparathyroidism, with a total corrected calcium of 2.91mmol/L, ionised calcium of 1.62 mmol/L, PTH of 62pg/mL and 24h urinary calcium-excretion of 5.9 mmol/L. The patient subsequently underwent 3 gland parathyroidectomy with calcium of persistent postop hypercalcaemia.

Further investigations: Parathyroid profile and fasting urinary calcium parameters were subsequently performed and suggestive of FBHH. Her parents showed fasting urinary calcium parameters within the FBHH range, but total and ionised calciums were normal. The patient had questionable low bone density on DEXA-scan. Bone markers were appropriate for age and 1,25-vitamin D levels decreased, supporting a diagnosis of FBHH.

Conclusion: The patient displays the typical features of FBHH, namely persistent asymptomatic hypercalcaemia following parathyroidectomy, hypocalcaemia and young age (< 40 years). The negative family screening is however against the diagnosis. Mutational analysis is not yet widely available and of limited use in diagnosis due to the large variety of mostly missense mutations (>40) of the calcium sensing receptor (CaSR) gene, current inability to forecast the effect of a given mutation and absence of a known CaSR gene mutation in up to third of patients. A rare FBHH variant has also been described due to a mutation of another gene. The absence of a family history in our patient could possibly be explained by a de novo mutation, decreased penetrance in the parents, or the presence of a rare acquired condition due to anti-CaSR autoantibodies and associated with other autoimmune disorders.

Exposure of MC3T3-E1 Osteoblasts to Polyunsaturated Fatty Acids modulates Prostaglandin E₂ synthesis and secretion of Osteoprotegerin.

M Coetzee, M Haag, *MC Kruger.

Dept of Physiology, University of Pretoria, Pretoria, South Africa;

**Institute of Food, Nutrition and Human Health, Massey University, Palmerston North, New Zealand.*

Polyunsaturated fatty acids (PUFAs) increase bone formation in animal studies and an anti-resorptive effect on bone has been observed in elderly women after supplementation with PUFAs for three years. The cellular mechanism of PUFA action may be due to modulation of local bone regulating factors such as prostaglandins (PGs), or to regulation of proteins expressed by the osteoblast such as osteoprotegerin (OPG).

In this study, MC3T3-E1 cells derived from murine calvaria that exhibit the phenotype characteristic of osteoblasts *in vivo* were used. In order to measure PGE₂ synthesis, cells were cultured for 24 hours and subsequently exposed to vehicle (0.1% ethanol)(control), and arachidonic acid (AA) or docosahexaenoic acid (DHA) at 20µg/mL for four hours and the culture media collected. For measurement of OPG, cells were precultured for 24 hours, exposed to vehicle, PGE₂ (10⁻⁶M), AA or DHA at 2.5 to 20µg/mL for 24 hours and the conditioned media harvested. In some cases 1µM indomethacin, a cyclooxygenase blocker, was added 45 minutes prior to addition of test substances. Three separate experiments were conducted (n=4).

Exposure to AA increased PGE₂ production 18 fold (p<0.05) over that of the control, while DHA had no effect. AA suppressed OPG secretion in a dose-dependent manner, and this effect was abolished by addition of indomethacin. PGE₂ reduced OPG

secretion by 40% ($P < 0.05$), as compared to control. DHA at higher concentrations also reduced OPG secretion by 20%, but concomitant exposure to indomethacin had a less prominent effect. MC3T3-E1 cells produced significant amounts PGE₂ in response to exogenous AA suggesting significant cyclo-oxygenase activity. AA decreased OPG secretion possibly via PGE₂ formation, as PGE₂ alone also significantly reduced OPG secretion. Abolishment of the effect of AA by indomethacin, confirmed this observation. The slight reduction of OPG by DHA could be due to endogenous PGE₂ production, as DHA itself is not a substrate for PGE₂ synthesis. This work needs to be expanded to include measurements of RANKL secretion, as the ratio between OPG and RANKL is important for regulation of the bone microenvironment. This work was supported by the NRF.

Evaluation of Effects of Nutritional Education on a Group of Patients Suffering from Type II Diabetes.

M Czlapka-Matysik, J Jeszka, J Bajerska-Jarzebowska, B Prządka.

Department of Human Nutrition and Hygiene, Agricultural University of Poznan, Poland.

Background: Most diabetes patients require correct nutritional education to aid their treatment.

Aim: The aim of this study was to measure the impact of diabetes education classes on nutritional status, biochemical blood parameters and knowledge of type 2 diabetes patients.

Methods: We conducted a study in 30 type 2 diabetic patients (15 women; 15 men) aged 46-75 yrs-old, who were attending the out-patient clinic of a local hospital. Over a five-month period, diabetics attended meetings where they received nutritional guidelines. At baseline and at the end of the five months, we assessed their nutritional knowledge and nutritional status – based on anthropometrical and body composition data – and we collected blood samples – in which concentrations of total cholesterol, low and high density lipoproteins (LDL and HDL), triglycerides, glycosylated haemoglobin (HbA1c), and glucose were assessed using standard techniques. Height, weight, waist and hip circumference were recorded from which waist-hip ratio and body mass index (BMI) were calculated. Body composition – fat mass, FM% and fat free mass, FFM% – was assessed with the FUTREX 6100/XL, which is an infrared radiation method. Changes in blood parameters, nutritional status and nutritional knowledge were calculated.

Results: In these 30 patients, in both sexes, we found that their nutritional knowledge increased over the five-month period. Similarly, concentrations of glucose, HDL, and HbA1c in their blood increased significantly ($p < 0.001$) over time. While at the same time, their BMI, FM% and LDL, triglycerides decreased significantly ($p < 0.001$). No changes were observed in the other parameters.

Conclusions: Our findings suggest that nutritional education classes for diabetics may have a positive impact on the nutritional status of diabetics and thereby aid their treatment. Such classes could become an integral part of therapy for type 2 diabetes patients.

Hungry Bone Syndrome – A Case Report.

MM Carrhill, SV Delpont.

Paediatric Endocrine and Diabetes Unit, School of Child and Adolescent Health, University of Cape Town.

Primary hyperparathyroidism is rare in the paediatric population, and hungry bone syndrome (HBS) even rarer. The prolonged and marked hypocalcaemia seen with HBS is caused by the aggressive uptake of calcium and phosphate by the bone, hence the name hungry bone syndrome. It has previously been shown that severe underlying bone disease (high PTH value) and an elevated alkaline phosphatase are factors that correlated with predicting HBS following parathyroidectomy.

We present a 16 year old girl with a parathyroid adenoma causing primary hyperparathyroidism. She had brown tumours and

classical subperiosteal resorption of the phalanges and distal tufts, with an elevated PTH and alkaline phosphatase. On day 2 post resection of the adenoma, she developed severe symptomatic hypocalcaemia, hypophosphataemia and hypomagnesaemia. PTH at this stage was within the normal range.

She was commenced on oral calcium carbonate, phosphate sandoz and 1-alpha vitamin D, as well as an intravenous infusion of calcium gluconate. Magnesium sulphate intravenous boluses were also used. Massive amounts of intravenous calcium were required to keep her symptom free. She remained in this infusion dependant state for 3 weeks, before weaning onto full oral supplementation.

This case illustrates the need to be aware of HBS in bone disease secondary to hyperparathyroidism treated operatively, highlighting the need to replace calcium, magnesium and phosphate aggressively, early and often for a prolonged duration.

Genetics of Abdominal Obesity in Different Ethnic Groups.

RE Mohlapamafsi, C van Niekerk, NJ Crowther.

Department of Chemical Pathology, NHLS, University of the Witwatersrand Medical School.

The aim of this study was to measure the prevalence of gene polymorphisms in the glucocorticoid receptor (GLR) and fatty acid binding protein 2 (FABP2) genes, in African subjects, to determine if ethnic differences in visceral fat depot size are due to genetic variation in these candidate genes. We evaluated the FABP2 and GLR polymorphisms using polymerase chain reaction (PCR) and RFLPs in 110 black subjects. The amplified PCR products were digested with restriction enzymes Hha1 and BclI respectively. Polymorphisms were determined by the banding pattern on agarose gels and individuals were typed as homozygous, heterozygous or wild type. We found frequencies of 61.8% heterozygotes, 2.7% homozygotes and 35.5% wild types for FABP2 in African subjects, compared to 41.1% heterozygotes, 13.9% homozygotes and 45% wildtype for a Japanese study. With GLR we found 44% heterozygotes, 2.7% homozygotes and 53.3% wildtype compared to Swedish males where the observed frequencies were 46.2% heterozygotes, 13.7% homozygotes and 40.1% wildtype. These data demonstrate that ethnic differences do exist in genotype frequencies for these candidate genes.

Riedel's Thyroiditis in a Black South African Patient.

C Droste¹, L Nokoane², KRL Huddle¹ and R Shires¹

Divisions of Endocrinology¹ and Anatomical Pathology², Chris Hani Baragwanath Hospital and University of the Witwatersrand, Johannesburg.

Introduction: Riedel's thyroiditis was first described in 1896. Invasive fibrous thyroiditis is an extremely rare condition (0.05% or less of surgical thyroid diseases). It is one component of a systemic fibrosclerotic disease which may involve the retroperitoneum, mediastinum, orbits, the biliary tree and lungs. We present a case where the diagnosis was made post mortem.

Case Study: A 55 year old black female patient was admitted to the Chris Hani Baragwanath Hospital with ascites. She had been diagnosed as hypothyroid the previous year, the aetiology thought to be Hashimoto's thyroiditis. She had been treated with thyroxine, but at the time of admission was clinically and biochemically hypothyroid (FT4 3.3pmol/l, TSH >150mIU/L). She had mild exophthalmos and a small, firm goiter, which moved well on swallowing. An ascitic tap was performed and the fluid found to be chylous. Urea and electrolyte testing done on admission revealed that she was in renal failure (Urea 32.4mmol/l, Creatinine 1676µmol/l). Serum calcium was normal. Abdominal ultrasound showed her to have bilateral hydronephrosis and a multifibroid uterus. Gynaecological opinion was that the enlarged uterus was unlikely to account for the hydronephrosis. An attempt at nephrostomy on the right failed, but a successful nephrostomy was performed on the left. Several attempts at inserting a central