Laparoscopicjuxtarenalvaricocelectomy

An important goal of surgery for varicocele is to achieve a diminished incidence of recurrence following operation. Until recently recurrence rates were exceptionally high. An important cause of failure is due to the presence of retroperitoneal collaterals which exit the testis and bypass the ligated testicular vein, rejoining it proximal to the site of ligature near its entrance to the renal vein or inferior vena cava.[1] Accordingly, ligation of the testicular vein near its termination was recently described by Kenawi[2,3] and has given excellent results. It was therefore felt worthwhile to attempt juxtarenal varicocelectomy by a laparoscopic approach to assess the feasibility of the procedure.

Five patients with an average age of 22.3 years were included in this study (Table 1). Scrotal ultrasonography and color duplex ultrasonography confirmed the presence of left varicocele in all, and an associated right varicocele in 4, with evident backflow on Valsalva manoeuvre. The patients were subjected to laparoscopic varicocelectomy. This was juxtarenal on the left side and routine[4] on the right aiming additionally at comparing operative time for both techniques.

Operative technique of left laparoscopic juxtarenal varicocelectomy. The operating table was tilted 15 degrees to the right. The preliminary steps were classic but for the case with left varicocele alone (case 4) the right side operating port was placed to the right of the midline midway between the xiphoid process and the umbilicus. The peritoneum of the left paracolic gutter was divided and the descending colon was mobilized medially. The testicular vessels were identified at the level of the sacral promontory and the vein was dissected proximally towards its insertion into the renal vein where it was clipped (Figure 1). Operating time for left juxtarenal varicocelectomy was greater on the average by 10 minutes compared to the routine technique on the right side.

All patients were relieved of their varicocele as judged clinically and by color duplex ultrasonography where the residual dilated veins showed no blood flow (Figure 2). The scrotal pain disappeared in all and the oligoasthenozoospermia was improved as judged by sperm count and motility. Conception has occurred (case 1). The follow-up period extended from 7 to 8 months (average 30 weeks).

The concept of an ultraproximal interruption of the

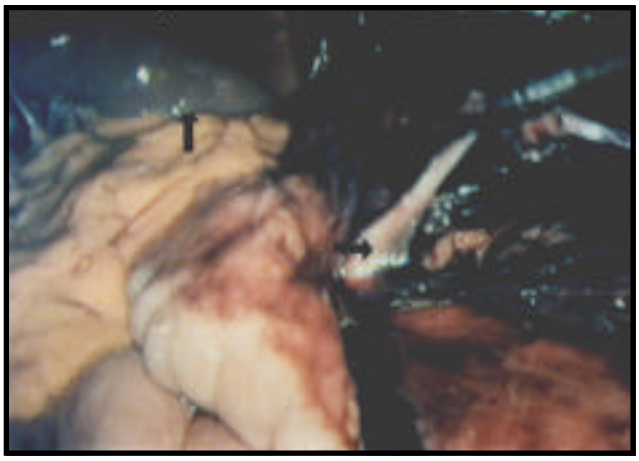

Figure 1 - Laparoscopic view of the terminal part of the left testicular vein (horizontal arrow) before clipping. The spleen is seen in the background (vertical arrow).

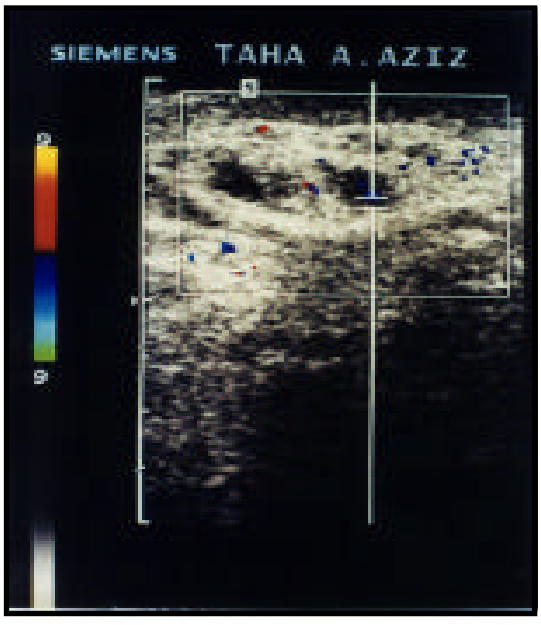

Figure 2 - Color duplex ultrasonography showing the previously dilated pampiniform plexus with no blood flow inside (Valsalva manoeuvre).

Table 1 - Clinical features and semen analyses.

Case	Age	Marital status	Complaint	Varicocele	Oligoasthenospermia
1	25	Married*	Infertility	Bilateral	Present
2	25	Single	Recurrent pain	Bilateral	Present
3	22	Single	Recurrent pain	Bilateral	Present
4	18	Single	Recurrent pain	Left	Present
5	21	Single	Recurrent pain	Bilateral	Present

*3 years

testicular vein has only been recently introduced by Kenawi.[2,3] This procedure avoids resorting to intentional ligation of the testicular artery which has been carried out by some[5] to ensure a diminished recurrence rate following varicocelectomy. Intentional sacrifice of the testicular artery is not necessary when the operation is carried out near the termination of the testicular vein as excellent results are obtained without this additional step.[2,3] The juxtarenal level of operation is also more physiologic since it lies above the level of the incompetent valves of the testicular vein that are located within several centimeters of its junction with the renal vein or the inferior vena cava. Juxtarenal varicocelectomy has been shown by this study to be feasible by the laparoscopic technique. This only adds a few (average 10) minutes to the operative time. This technique is worthwhile for use as the laparoscopic procedure of choice following recurrence after retroperitoneal varicocelectomy. This latter was the initiator and original indication for open juxtarenal varicocelectomy.[2,3] It is also worthwhile for consideration as the primary operative laparoscopic procedure for varicocele as has been carried out in the reported cases.

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Letters to the Editor

Pseudohypoaldosteronism type 1 in an Omani male infant simulating salt-losing congenital adrenal hyperplasia.

Sir,

Neonates who usually present with symptoms of lethargy, vomiting and dehydration after the first week of life, associated with marked hyponatremia and hyperkalemia, are commonly considered by pediatricians as cases of congenital adrenal hyperplasia due to 21-hydroxylase deficiency until proven otherwise. We present an Omani 10 day old full term male, who was admitted to our unit from a peripheral hospital with history of reluctance to feed and excessive crying for one day. Baby was delivered to a primiparous mother by lower segment cesarean section due to cephalopelvic disproportion and failure to progress. Family history was positive for consanguinity. Apgar scores were 8 at one minute and 9 at five minutes, and his birth weight was 3.270 kg. His initial course was unremarkable and he was discharged home on the second postnatal day. On the ninth day, he had excessive crying and poor feeding and at the nearest medical centre, he was found to be severely dehydrated and laboratory investigation revealed hyponatremia (Na^+: 123mmol/L) and hyperkalemia (K^+:8.4 mmol/L), and despite appropriate intravenous fluid resuscitation, hyponatremia and hyperkalemia persisted (Na^+:120 and K^+:9.0 mmol/Litre). A provisional diagnosis of congenital adrenal hyperplasia was made, and baby was moved to our neonatal intensive care unit for further work up and management.

On arrival, baby was sick looking, irritable but well hydrated (on 0.9% normal saline infusion), normotensive, with normal male genitalia and no other systemic abnormality. Laboratory investigations revealed a normal full blood count, while serum Na^+:121mmlo/L, K^+:5.5mmol/L, Cl^- : 87mmol/L, Urea:44umol/L, glucose:4.4mmol/L, Ca^+:3.22mmol/L, Lactate:0.88mmol/L, ABG revealed PH: 7.24, PCO_2:2.4 Kpa,PaO_2: 9.8 Kpa, Hco_3: 11.8mmol/L, BE: -17.9 mmol/L, O_2 sat: 92.6% and urinary PH was persistently 5 despite bicarbonate therapy. Renal ultrasound was normal.

Our working diagnosis was the same as that of the referring centre and so he was started on intravenous hydrocortisone 75mg/m^2/day (3 times normal acute stage dose), oral 9 alpha fluorocorticol 0.05mg daily and also received a bolus of soda bicarbonate 2ml/kg 8.4% for the metabolic acidosis. After initial fluid resuscitation he was placed on 14.6% oral hypertonic saline, 8meq/kg of Na^+ on which his serum sodium level was maintained. On the fifth day his serum potassium crept up to 8.1mmol/L without any potassium supplementation and so he was placed on calcium resonium (potassium chelator) orally and salbutamol nebulizer. For his metabolic acidosis, he was placed on supplemental oral soda bicarbonate 3meq/kg/day. Later we received his endocrine laboratory results which revealed normal 17-hydroxyprogesterone, normal plasma cortisol, normal urinary 17-ketosteroids and low urinary pregnantriol levels. His serum aldosterone and rennin levels were astronomically high. In view of these results, diagnosis of congenital adrenal hyperplasia was disregarded with normal serum 17-hydroxy-progesterone levels, and so hydrocortisone was stopped. Also because of high rennin levels, oral 9 alpha fluorocortisone was discontinued. By then we were informed that 2 cousins who were diagnosed to have pseudohypoaldosteronism earlier were requiring supplementary sodium along with sodium resonium. But surprisingly both of them now had no metabolic acidosis. Our baby was discharged home on oral hypertonic saline, oral soda bicarbonate as well as calcium resonium. On follow up in the clinic, infant is gaining weight and maintaining Na^+/K^+ within normal range and there is no metabolic acidosis.

Pseudohypoaldosteronism type 1 is autosomal dominant or sporadic, and is a milder disease that remits with age as against the recessive form with manifestation persisting in childhood.[1] The primary defect in this disorder is a lack of functional mineralocorticoid receptors. It is also possible that the elevated plasma concentration of aldosterone down-regulate entirely normal receptors and that the defect occurs elsewhere in the pathway of aldosterone action. In fact, no mutation affecting the action of aldosterone has been detected in the gene for the receptor in any patient studied so far. Infants present in the neonatal period with failure to thrive, vomiting and dehydration, there is acidemia and hyperkalemia.

They do not respond to synthetic mineralocorticoids but are treated with sodium supplements, and sodium chloride required is deduced from the normalization of plasma K^+ concentration and rennin activity.[2,3] Although the primary defect persists for life, improvement may occur beyond 2 years of age, when salt wasting improves as the kidney becomes less dependent upon the distal nephron for sodium recovery. Older children are generally asymptomatic while eating a normal salt intake, but the plasma aldosterone concentration remains elevated.

Pseudohypoaldosteronism type 11 (Gordon's syndrome) is a rare cause of hyperkalemia, hyperchloremic metabolic acidosis, volume expansion and hypertension, which was not present in our patient. Mode of inheritance is autosomal

recessive. Here plasma rennin activity and aldosterone concentrations are suppressed and patient may have hypercalciuria and a tendency to stone formation.[4] This disease represents an inherited tendency to hyperreabsorb the filtered sodium chloride, which leads to short stature and expansion of extracellular fluid volume, with resultant hyporeninemic hypoaldosteronism. Therapy in these patients is sodium restriction and chloruretic diuretics (thiazide). It seems likely that the autosomal dominant and recessive forms of pseudohypoaldosteronism represent distinct genetic defects.

Another differential diagnosis is type 1V renal tubular acidosis and is due to congenital hypoaldosteronism (both true and pseudo) ie end organ failure, and is characterized by hyperchloremic acidosis with hyperkalemia and an acidic urine (PH < 5.5) during acidosis. In the absence of aldosterone-mediated Na^+ reabsorption, hyperlkalemia develops and this suppresses renal ammonia production, resulting in a reduction of ammonium ion excretion and thus acid excretion. It is also seen in conditions where there is direct damage to the distal nephron such as in obstructive uropathy. In these cases, there is salt wasting in very young children, however this condition resolves by five years of age. The syndrome appears to be heterogenous and in patients salt loss involves only the renal tubules, whereas in others salivary and sweat glands may be involved and occasionally the colonic mucosal cells may be effected.[5] Administration of mineralocorticoids is ineffective, and this condition is treated by supplementary Na^+,which may be discontinued as the condition improves usually by two years of age. Also dietary K^+ restriction and a K^+ losing such as frusemide along with sodium bicarbonate may also be indicated.

In conclusion, any neonate with late presentation of dehydration with hyponatremia and hyperkalemia should also be evaluated for pseudo-hypoaldosteronism in addition to congenital adrenal hyperplasia. We hope this prospective provides a basis for the physician to recognize pseudohypoaldosteronism so that an informed decision may be carried out at choosing the best therapy. The physician may also be prepared to reasonably project the prognosis for each patient.

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Neonatal salmonella meningitis.

Sir,

Salmonellosis is a bacterial infection acquired through faecal contamination of water. It can be contracted through meat and dairy products also. Since the discovery of chloramphenicol in the year 1948, mortality and morbidity by salmonella was reduced drastically. But now with emergence of chloramphenicol-resistant strains, cephalosporins like cefotaxime, ceftriaxone and newer fluoroquinolones such as ciprofloxacin are likely to find a place in the treatment modality, especially in cases of established bacteremia and meningitis. We had a case of salmonella septicemia and meningitis which relapsed and died after treatment.

A 3 month old Omani female child was admitted to Rustaq Hospital with complaints of fever, vomiting and off feeds since 3 days. The child was delivered normally at full term and was fully immunized for age.

On examination, the relevant findings were drowsiness and bulging anterior fontanelle. The rest of the findings were normal.

Routine biochemistry parameters were within the normal limits. Hemogram showed a hemoglobin of 8.7 gm% and slightly raised white blood cell (WBC) count, 13.9 K/uL. Lumbar puncture was carried out and CSF examination showed raised protein=01.11g/L glucose=0.5 mmol/L, total WBC count 800 cu.mm with a differential of 80% polymorphs and 20% lymphocytes. Grams staining showed gram negative bacilli and abundant pus cells. CSF when cultured grew Salmonella group D, identified by API - 20 E and serology, showing sensitivity to norfloxacin,

cephradine, ampicillin, chloramphenicol, gentamicin, amikasin, and ceftazidime.

Blood culture carried out at the same time showed the same growth and sensitivity pattern. The patient was put on cefotaxime 250 mg I/V 6 hourly and amikacin 30 mg I/V 8 hourly. During the hospital stay the patient developed convulsion once, which was managed symptomatically. The patient was discharged 2 weeks later with improved condition.

She was readmitted 2 weeks later with high grade fever, convulsions and was started on cefotaxime 300 mg I/V 6 hourly, amikacin 45 mg 8 hourly, chloramphenicol 150 mg I/V 6 hourly (only 2 doses were given as patient developed leucopenia); ampicillin 400 mg I/V 4 hourly 2 doses (after chloramphenicol was discontinued) and antiepileptic drugs along with supportive care. CBC carried out showed WBC = 4.1 K/ul with reactive lymphocytosis.

On examination there were signs of raised intracranial pressure and ultrasonography of cranium showed dilated ventricles. Blood culture showed growth of Salmonella typhi with sensitivity as before. The patient was diagnosed to be in septic shock, coma and was managed accordingly. But the condition deteriorated and the child died the next day.

Meningitis in infants is still a dreaded disease in spite of the advent of antimicrobials. Most common organisms causing meningitis in newborns are group *B streptococci, staphylococcus aureus, Enterococcus* species, and other gram negative bacilli like *Enterobacter* species, Salmonella meningitis is a particularly serious complication of Salmonella infection in neonates and very young children. Cases of Salmonella meningitis have been reported sporadically mostly from India.[1] However, it forms a minor group of causative agents in studies reported from other countries. Jeutsch et al[2] have also reported 2 cases of group D salmonellosis in neonates. Out of these, 2 neonates had septicemia and meningitis. Of these, one patient died and he noted that only children fed with artificial food suffered from salmonellosis and children on breast milk had unremarkable clinical course.

Francis et al[3] in their retrospective survey of neonatal meningitis occurring in Australia stated that mortality was 26% and long term sequelae were common in those with gram negative meningitis and recommended to add third generation cephalosporins to all gram negative meningitis.

Established Salmonella meningitis and bacteremia requires aggressive antimicrobial treatment with aminoglycosides, cephalosporins like cefotaxime and ceftriaxone, which have a high penetration in cerebrospinal fluid, and are proving to be a effective alternative in cases resistant to chloramphenicol. Complicated cases require prolonged treatment than the conventional 10-14 days regime. Koc E et al[4] have reported 2 cases of Salmonella meningitis, one of which relapsed after 4 weeks of cefotaxime treatment and was cured by Imipenem/cilastatin therapy only.

Huang et al[5] in their retrospective review of 15 pediatric patients with Salmonella meningitis reported that 14 surviving patients were treated with a third generation cephalosporin for at least 3 weeks and a high frequency of prolonged fever, neuroimaging abnormalities and neurologic sequelae were seen in these patients.

Salmonella meningitis is a rare concurrence in cases of salmonellosis, especially children. But due to the associated grave prognosis, this diagnosis should always be kept in mind whenever a bacterial meningitis is suspected in young children. An aggressive treatment should be initiated with third generation cephalosporins, keeping in mind the emerging resistance to chloramphenicol. Also, the treatment should be given for prolonged duration rather than the routine 10-14 days regime.

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Overview of chronic diseases in the Kingdom of Saudi Arabia

Sir,

Non-communicable diseases are expected to count for 7 out of every 10 deaths in developing regions, compared with less than half today.[1] Diabetes Mellitus (DM) is a common, serious and costly health problem. Saudi Arabia is a high prevalence

country 12-16% with DM being associated with a high incidence of complication.[2] Diabetes is a costly condition by virtue of its high prevalence and high costs per person. A large proportion of these costs are related to treating complications of diabetes.[3]

Hypertension (blood pressure >140/90) has been reported to affect more than 20% of adults, however some surveys conducted over the past 2 decades were based on different diagnostic criteria and age composition of the samples studied and therefore are difficult to compare. Studies have also shown a low detection among people with high blood pressure (of 140/90 or higher) were not aware of their elevated blood pressure before the survey. Based on a conservative hypertension prevalence figure of 29% in the population of 20 years of age and older, and assuming that 60% of cases are undiagnosed, it can be estimated that there are over 44 million people in the Eastern Mediterranean region with high blood pressure; more than 26 million having undiagnosed hypertension.[1]

Prevalence of hypertension was surveyed in South Western Saudi Arabia. The overall prevalence was 11.1, and the age-adjusted prevalence was 10.6% in men and 11.4% in women. Prevalence increased significantly by age. Among previously known cases, 76% were receiving treatment, but of these only 20% were found to be controlled. It was concluded that hypertension affects a sizable population of Saudi communities and further efforts are needed to improve the control of the disease.[4] Incidence of asthma among school children in Saudi Arabia was found to be about 10% in Riyadh, compared to about 13% in Jeddah and Qassim and 17% in Abha.[5]

Obesity is an important risk factor for the development of non-insulin dependent diabetes mellitus, hypertension, hyperlipidemia, coronary heart disease, stroke and gall bladder disease.[6] In Saudi Arabia, one study was carried out to determine the prevalence of obesity among Saudi males in the Riyadh region and showed that only 36.6% of subjects were within their ideal weight (BMI<25 kg/m^2) while 34.8% were overweight (BMI 25-29.9 kg/m^2), 26.9% were moderately obese (BMI 30-40kg.m^2), and 1.7% were morbidly obese (BMI>40kg/m^2).[7] Another study showed high prevalence of clinical obesity among Saudi females, as only 26.1% of subjects were their ideal weight (BMI<25 kg/m^2).[8]

In Saudi Arabia, one study was to assess the prevalence of anxiety and depression among patients coming to primary health care centers in Riyadh, and this showed definite cases of 15.3% for anxiety and 12.7% for depression.[9]

Reported mortality statistics have indicated that cancer is emerging as one of the leading causes of death, occupying third place in some countries. Estimates provided by the international agency for research on cancer indicated that over 450,000 new cancer cases occurred in the Eastern Mediterranean Region during 1995.[1]

Road traffic accidents (RTA) are a noticeable common cause of death in Saudi Arabia. RTA are the primary cause of death among dead on arrival cases affecting the most active and productive age group.[10] One study in Saudi Arabia showed that speeding was a contributing cause of 53% of the accidents. More than 80% of the drivers involved in accidents were in the 18-49 age group while 7.8% were under 18 years and 12% were over 50 years.[11]

Treatment of established non-communicable diseases is expensive and for some diseases often ineffective. Prevention is the best way of avoiding growth in the burden of these diseases and in unnecessary health care expenditure.

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Temporal aberrations in dreams

Sir,

The sequential organization of dreams is not well understood. Successive scenes of a dream seem completely discontinuous from a narrative point of view, whereas they are semantically linked.[1] Parts of the world of wakefulness are clearly recognizable in dreams and both worlds merge.[2] Many of us must have had the experience of seeing in a dream a certain object the shape of which perfectly matched a real object that was in his view as he woke up.

Occasionally dreams can justify the happening of unpredictable real events that interrupt them, in such a manner that the real events would seem to be the logical sequels of the dreams. For example, in one dream, the author found himself in a street battle in the course of which a hand grenade was hurled at him. It exploded in his hands just as he awoke to the sound of a real explosion near his apartment in his war torn city.

The paradox in this dream is that the dreamer could not have anticipated the real unpredictable event (the explosion) interrupting his dream. Therefore the anticipatory part of the dream must have developed or been re-arranged from past memory a split second after the real explosion but decompressed to real time and remembered in proper chronological sequence in relation to the real event by the dreamer. If this were not so, we would then have to postulate that clairvoyance and prognosticating abilities are enhanced in dreams.

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Correspondence

Criteria for diagnosis of growth hormone deficiency

Sir,

We read with interest the article "Criteria for diagnosis of growth hormone deficiency: does it exist?" by Al Herbish.[1] There was a mixed response of the participating physicians regarding the criteria used in diagnosing growth hormone deficiency (GHD). This is not a surprise to us because with the availability of recombinant growth hormone, criteria for diagnosis of GHD are now being modified more frequently. In the hands of a practicing physician, measurement of growth velocity is a simple and reproducible way of assessing growth of a child that has to be checked at a minimal interval of 6 months.[2] If growth velocity is retarded it gives sufficient time to the primary physician to make a positive diagnosis for a subnormal growth. Bone-age is an important accompaniment of most of the disorders causing short stature, where bone-age usually corresponds to the height-age with a few exceptions. Delayed bone-age has often a prognostic value regarding the attainment of the adult height, subjects with delayed bone-age have better prognosis in this regard. In growth hormone insensitivity syndrome, bone-age is usually accelerated when compared with height-age.[3] Growth hormone is secreted in a pulsatile fashion, so a random measurement of growth hormone may not be of much use in the diagnosis of GHD. At the same time, a high basal growth hormone in presence of clinical suspicion of GHD may be a pointer towards possible growth hormone insensitivity syndrome entity being not uncommon here in Kashmir Valley.[4] In these patients, high growth hormone levels accompanied with low levels of insulin growth factor-I and growth hormone binding protein-3 would make a definitive diagnosis of growth hormone insensitivity syndrome possible.[5]

Definitive diagnosis of GHD needs at least 2 positive provocative tests of growth hormone stimulation.[6] We at the Sheri-Kashmir Institute of Medical Sciences Srinagar are using an insulin tolerance test as an initial test for the diagnosis of GHD. The test is carried out by a resident under supervision of a senior resident and a consultant on a continuous glucose monitor. We did not come across

any complications so far. In subjects diagnosed to have GHD on insulin tolerance test, it is our routine practice to confirm the same on at least 2 more stimulation tests namely, Levo-dopa and exercise test. Diagnosis of GHD remains only an academic exercise in developing regions like ours where most of the children can't afford costly treatment like growth hormone therapy.

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Reply from the Author

I sincerely thank Dr. Bashir Laway's correspondence in connection to the article "Criteria for diagnosis of growth hormone deficiency: Does it exist?', published in this journal.[1] I am glad to find Dr. Laway agreeable to the suggestion of keeping a strict criteria for growth hormone deficiency. I feel this is very important even in developing regions where most growth hormone deficient children may not afford growth hormone cost. It is important to protect available growth hormone for these children in need for it i.e. growth hormone deficient and perhaps children with Turner's syndrome or chronic renal failure.

I cannot agree more with Dr. Laway in regards to the importance of growth velocity. It is therefore quite possible to follow up short children and extrapulate growth velocity based on the minimum of 4-6 months interval. The exception to this rule may be children with severe short stature, definition of which is very objective, presence of other symptoms like hypoglycemia or evidence of other pituitary hormone deficiency and children entering puberty where age can be critical.[2-7]

I emphasize the low yield of random growth hormone measurement due to the pulsatile nature of growth hormone release into the circulation. Children with lack of the function of growth hormone e.g. biologically inactive growth hormone, growth hormone receptor deficiency (Laron syndrome), congenital 1gF1 deficiency (a condition evident in the African pygmies) and tissue unresponsiveness to both growth hormone and 1gF1 are very rare. I feel in developing countries, short stature due to malnutrition, liver disease and malabsorption (e.g. celiac disease) which may be associated with elevated growth hormone may need to be considered prior to the above mentioned rare conditions.[4,8]

I am in favor of establishing a diagnostic criteria in every country and perhaps the criteria may be unified for many neighboring countries. This will lead to a better utilization of both investigation tools and treatment for short stature children particularly growth hormone deficient children.

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Prevention of migraine

Sir,

I have read the interesting article about the prevention of migraine headache. As you mentioned, the first step is non-pharmacologicl management such as limitation in consuming some known specific foods and food materials, eg. old cheese, chocolate, red wines,[1] tyromine containing foods, flavor enhanced like monosodium glutamate, and meat with nitrite preservations[2] or keep an eye on situations like hyper/hypoglycemia which initiate and aggravate migraine headache[3] as well as keeping away from

bright lights[2] or too much sleep which potentiate this troublesome,[4] and recommendation of these preventable factors to the patient should be taken into consideration.

Except the pharmacological prophylactic measurements you have mentioned, taking prednisone in divided doses as well as combination of ergot, atropine and phenobarbital have been recommended as a prophylactic remedy.[5]

But the best selected prophylactic route is that with lowest side effects, lowest cost and the most effectivity, so taking high doses of riboflavin which has been postulated with low side effects (diarrhea and polyuria) with its other favorable conditions, makes this vitamin the best candidate as a prophylactic drug.[6-8]

In addition, some authors believe that routine employment of drug prophylaxis in migraine may be greatly undermined by poor compliance with low efficacy[9] and because some specific causes of migraine headaches such as menstrual ones are often resistant to abortive and preventive medications10 and also some peculiar side effects of prophylactic drugs such as initial exacerbation of migraine attacks on first taking calcium channel blockers[5] and long time prevalence of some types of this disease (eg. menstrual one), with taking too much prophylactic drug for extending period (in this case, taking sumatriptan)[3,10] is distressing, so these make the role of treatment of a generated migraine so important, and between different measurements, with the exception of ergotamine tartrate with caffeine as the leading agent,[2,11] some other techniques such as biofeed back and spinal manipulation are used for lessening the suffering of this affliction.[12,13]

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Reply from the Author

Pahlavan's comments on prophylactic treatment of migraine are certainly well taken. As pointed out in our review paper, we strongly encourage patients to use non-pharmacological prophylaxis, such as relaxation techniques in association with lifestyle adaptation. However, if these measures fail drugs are very often the only alternative.

Many other prophylactic regimens are found in textbooks.[1-5] Although recommended by experts their use is often anecdotal and hard evidence regarding their effectiveness is lacking from properly designed clinical trials.

Although we regret not to have mentioned the potential of the riboflavin in migraine prophylaxis,[6,7] we felt that data at that time were, and still are, very scanty and did not meet our clinical trial evaluation criteria in terms of patient numbers or design. The particular study referred to by Pahlavan had only 28 patients in the riboflavin group and 26 in the placebo group.[7]

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Book Reviews

Vascular Disease. A multi-specialty approach to diagnosis and management 2nd Edition

DARWIN ETON, 685pp. **Price:** US$ 45.00, **Publishers:** Landes Bioscience, USA, **Date of Publication:** 1999, **ISBN:** 1-57059-561-5.

Diseases that affect the vascular tree remain to be an important cause for morbidity and mortality. Among textbooks that discusses the updates of diagnostic tools in investigating a particular vascular disease and its management, is the second edition of "Vascular diseases: A Multi-Special Approach to Diagnosis and Management" by Darwin Eton. Landes Bioscience publishes the textbook in 1999. The contents of the text are contained in 685 pages, through 42 chapters and an index. Areas, which are covered by this textbook, include all aspects that are related to vascular diseases. These areas include epidemiology, pathology, clinical presentations and recent advances in modalities of investigations; like ultrasonography, computerized tomography, magnetic resonant images, angiography and nuclear medicine, which are aided with plenty of clear illustrations. Also, different and recent therapeutic options are well discussed through out the textbook. There are plenty of explanatory figures and simple tables that contain different etiologies, disease classifications and therapy protocols. The topics of the text are carefully titled, well designed and thoroughly indexed. The textbook costs 45 US$ and is relatively small in size and low in weight, which makes easy to access and carry around. I would recommend this textbook for vascular surgeons, neurologists, neurosurgeons, cardiologists, general physicians and radiologists.

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Aviation Medicine

J. ERNSTING, A. NICHOLSON, D RAINFORD, 703pp. **Price:** PS 95, **Publishers:** Butterworth Heinemann, USA, **Date of Publication:** 1999, **ISBN:** 0-7506-3252-6.

This book has been written by well-known authors, who have a vast experience in teaching and research in the field of Aviation Medicine. Flying environment is hostile, demanding and stressful to human body, aviation medicine as you know is preventive in nature, dealing with man-machine interface specifically Pilot versus Aircraft interface.

The book covers in detail and uses understandable language - the physiology of atmosphere, aviation physiology, aircrew systems, aircraft accident investigation, the aviators fitness to cope with demands and the probable hazards of flying environment and equipment and how to prevent, deal with and manage with special (particular) concern on flight safety.

The quality of the coverage is very good. The content is appropriate for the book title. The different subjects are clearly presented. Tables, figures and illustrations are adequate, clear and of good quality. The references are relevant, recent and properly referred to. This book is addressed to aviation community i.e. flight surgeons, Aviation Medical examiners, flight nurses, teams of aeromedical evacuation, pilots and flight engineers. It is very helpful to civil and military aviators.

Terms in layers of atmosphere such as biosphere and chemosphere are to be explained if possible in the next edition.

Air pollution and air pollutants could be added if possible in the next editions, similarly for Radiation Health Effects. More details in chapter 35 about noise, such as hearing loss, measurements (audiograms, and audiometers) and prevention.

In brief, this book is a reference -nice to have it. It is a very "good buy" to flight surgeons and those who are concerned and interested in aviation medicine.

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Radioguided Surgery

ERIC D. WHITMAN, DOUGLAS REINTGEN 164pp. **Price:** US$ 45.00, **Publishers:** Landes Bioscience, USA, **Date of Publication:** 1999, **ISBN:** 1-57059-495-3.

The book is quite reasonable. It is indeed covering the basis of a successful radioguided surgery based on the long experience of the author Dr. Eric D. Whitman and his colleagues. Basically, radioguided surgery has provided a reliable and accurate way to identify regional lymph nodes at risk for micrometastasis which was used initially in patients with melanoma and now extended for patients with

breast cancer, skin malignant tumors, bone lesions and even for parathyroid localization intraoperatively.

Application of radioguided surgery provides the surgeons in cooperation with the nuclear medicine physicians and pathologists a lymphatic mapping which clearly delineates the lymphatic drainage pattern from primary tumor injection sites to the regional lymph nodes at risk by injection of radioactive colloid with blue dye for staining and by using intraoperative a handheld gamma probe which is capable of detecting the accumulation of intradermally injected radiolabelled colloid within the sentinel nodes.

The authors described a nice algorithms to apply a sentinel node and radioguided surgery including data sheets on different sentinel lymph nodes detection in various diseases (such as in melanoma, breast cancer and parathyroid detection) which is in particular useful for inexperienced institutions. In addition, description of a good gamma probe was mentioned generally without any baize.

In chapter two, the book covered elegantly the basic of radiation, radiation safety which is important to know for the physicians and surgeons who are working with gamma camera probes, even described how to test the probe before starting the procedures and the basic components of the probes and their types and how to use it properly to avoid its complications such as false positives results with the advantage and disadvantage of each type.

The book also described in detail the estimated dose of radiation during the procedure to the surgeons and pathologists and how to use good radiation safety practice which is a very important task to know.

The authors of the book emphasize how training in sentinel nodes radioguided surgery is important and credentiating physicians and surgeons and gave the most important steps to perform an ideal training program for radioguided surgery and emphasize how important to supervise the physicians or surgeons after completing the training program. Not only that but also mentioned is that this program should enable the faculty to personally supervise the first few radioguided surgical procedures performed by the student.

In my opinion, this book seems good which gives a comprehensive and basic technique of the new technology about radioguided surgery and hopefully in the future would be able to try this technique.

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Diarrhea and Constipation in Geriatric Practice

RANJIT N. RATNAIKE 236pp. **Price:** US$ 59.95, **Publishers:** Cambridge University, UK, **Date of Publication:** 1999, **ISBN:** 0-521-65388-6.

There is a global increase in the population of the elderly. Great proportions of health care resources are spent on care of the elderly. This book addressed two of the most common problems of the geriatric population.

The etiology, underlying conditions, pathogenesis and management issues of diarrhea and constipation are described in a lucid, easily understood manner highlighting the vulnerability of the elderly to certain pathological conditions. Written by a group of skilled practitioners the book covers diverse disciplines from immunology to nursing care and nutritional issues.

This is an excellent book to be acquired not only by geriatricians but also by general medical practitioners.

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