Letters to the Editor

Khat chewing is a risk factor of duodenal ulcer

Sir,

Duodenal ulcer is estimated to affect 10% of the population during some time in their life.[1] Duodenal ulcer was found in 14% at the Endoscopy Department in Riyadh Military Hospital.[2] No national figure was traced in recent literature about the situation in Yemen. But increases in attendants at hospitals with duodenal ulcer are noticed. Vast majorities of them are khat chewers.

Khat is a plant known scientifically as Catha ediulis forsk. Its fresh leaves only are used for chewing. Khat chewing is a very common habit in Yemeni society. It is chewed also in southern parts of Saudi Arabia[3] and in several east African countries. Recently khat appeared in USA and several European countries.[4]

Duodenal ulcer is a multi factorial disease, with many known risk factors, eg. genetic predisposition, blood group, HLAP,[5] colonization of *Helicobacter pylori*,[6] smoking, NSAID and alcohol consumption.[5] Khat chewing was found to be associated with gastritis, delayed gastric emptying and decreased gastric cell secretion. Daily khat chewing was found associated with high prevalence of *Helicobacter pylori* and duodenal ulcer, especially in women. However, the study was of small sample size (n= 28) and was not controlled for. Current study was designed to explore association between khat chewing and duodenal ulcer.

Analytical design was employed in this study with cases and controls coinciding in background and confounding variables. Cases were defined as patients who had duodenal ulcer and controls that did not. Both having being diagnosed by gastroduodenoscipy. Exclusion criteria was patients that had duodenitis, erosion, stress ulcer, Zollinger-Ellison syndrome and corrosive ulceration. Any individual who had clinical features of duodenal ulcer was excluded from controls also.

Khat chewer was defined operationally as a person who chewed for => 14 hours per week. Sample size was calculated according to an expected exposure of 37% and worth detecting odds ratio of 1.6 at a confidence level of 95%. Patients and controls were informed about the study and signed on consent forms.

Data was collected through questionnaire forms by the investigators and entered onto a PC, processed and analyzed by SPSS 9 program for the calculation of t-test, Chi square and Odds ratio. P-value was considered significant at < 0.05.

Cases and controls were significantly comparable in all background variables. Levels of significance (p) were 0.54 for mean age, 0.32 for sex, 1.0 for marital status, 0.225 for residency, 0.412 for occupation, 0.708 for educational level and 1.098 for blood group. Differences in confounding variables were also not significant statistically as revealed in Table 1. Regarding khat chewing in relation to duodenal ulcer please refer to Table 2. Mean duration of chewing in cases was 25.09 hours per week compared to 11.01 hours only in controls (t-test = 8.30, P = 0.000).

Results revealed that khat chewing is significantly associated with duodenal ulcer. This effect can be due to stress that follows khat chewing. This phenomenon is very common and is induced by the effect of amphetamine like action of cathine present in khat. Another possible factor can be due to *Helicobacter pylori* associated with khat chewing, beverages consumed during the session or

Table 1 - Variables confound the relationship between duodenal ulcer and khat chewing.

Variable		Cases %	Controls %	Chi²	P-value
Smoking	Yes	38	33	1.111	0.299
	No	62	67		
NSAID	Yes	5	8	1.091	0.623
	No	95	92		
Family history	Yes	17	16	0.076	0.881
	No	83	84		
Alcohol consumption	Yes	3	4	0.074	1.086
	No	97	96		
Chronic disease	No	87	86	0.355	0.949
	Liver	4	5		
	Renal	4	4		
	Other	5	5		

Table 2 - Khat chewing in relation to duodenal ulcer.

Duodenal ulcer	Khat chewing		Total
	Yes No. (%)	Yes No. (%)	
Cases	133 (76)	42 (24)	175
Controls	52 (35)	98 (65)	150
Total	185 (57)	140 (43)	325
Chi square = 56.275, P = 0.000, Odds ratio = 5.968			

insecticides and chemicals used for growing the plant. All these factors together with the chemical constituents of the khat itself needs further investigation to unveil the causation effect on duodenal ulcer.

References

1. Miguigan JE. Peptic ulcer. In: Harrison's textbook of Medicine. 14th ed. New York: McGraw-Hill; 1998. p. 1232-1236.
2. Elkarawi M, Ali A, Mohamed AE, Mohamed SA, Ghandour ZM. Retrospective study in Endoscopy Department at Riyadh Armed Hospital. Saudi Medical Journal 1999; 20: 598.
3. Soufi HE, Kameswaran M, Malatani T. Khat and oral cancer. J Laryngol Otol 1991; 105: 643-645.
4. Kalix P. The pharmacology of psychoactive alkaloids from ephedra and catha. J Ethnopharmacol 1991; 32: 201-208.
5. Dayal Y, Delelis RE. Stomach and intestine. In: Robbins basis of pathology. 4th ed. Philadelphia: Saunders; 1989. p. 850-854.
6. ElGuneid A, Al-Sherif A, Murray-Lyon I, Zureikat N, Shousha S. Effects of chewing khat on mucosal histology and prevalence of Helicobacter pylori in esophagus, stomach and duodenum in Yemeni patients. Histopathology J 1991; 19: 437- 443.

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Evaluating the efficacy of saline solutions in treatment of chronic sinusitis

Sir,

Chronic sinusitis (CS) is a common cause of morbidity in adolescents. There are significant links between CS, asthma and allergic rhinitis. Predisposing conditions for development of CS are obstruction of ostium secondary to thick secretions, mucocilliary dysfunction, and mocusal edema.[1] Saline nasal washing appears to be of beneficial effects in enhancing mucocilliary clearance in patients with CS,[2] asthma,[3] cystic fibrosis[4] and even in normal subjects.[3] This study was performed to compare the effects of hypertonic saline (HS-0.3%) and normal saline (NS-0.09%) solutions in treatment of CS.

Fifty patients aged between 10 and 19 were divided in two groups. Hypertonic saline solution was used in treating the first group who had a mean age of 17.5 (HS group), and normal saline was applied in the second group with a mean age of 16 (NS group) (December 1998 to September 1999). Chronic cough and repeated throat clearing were the chief complaints of all patients. These symptoms did not respond to anti-allergic drugs. Headaches, halitosis and nasal obstructions were among other complaints. All the subjects had cough and purulent post-nasal discharge (PND). Redness and swelling of nasal mucosa were revealed upon rhinoscopic examinations. Chronic cough and PND together with one or more of the aforementioned complaints established the clinical diagnosis of CS.[1] X-ray evaluation of paranasal cavities (Water's projection) was performed for all patients early on the study. Thickness of mucosa (>4mm) and/or opacification, (>50%) of the sinus cavity confirmed the diagnosis.

Antibiotics, antihistamines, and decongestants were interrupted one week ahead of the study and were not allowed during the course of treatment. The treatment regimen of the HS and NS groups consisted of 5 drops of hypertonic and normal saline, which was applied in each nostril three times a day. Other causes of chronic cough (asthma, allergic rhinitis) were not clinically suggested for any of the patients. Exclusion creteria were pulmonary problems, nasal polyps, fever and major septal deviation. Six subjects discontinued their participation in the study and four were excluded due to complications of treatment (severe burning sensation). Assessment of the severity of coughing and PND in patients was based on the interviewer's impression of the discomfort expressed by them. The changes in the mean PND and cough scores of subjects are demonstrated in Table 1. Assessment was performed at the beginning, two weeks later, and at the end of the study. The scaling criteria were as

Table 1 - Mean symptoms scores of patients with CS who were treated with saline solutions.

Groups		Before	Mid	After
Cough	HS	2.81	1.9	1.3
	NS	2.73	2.4	2.1
Post natal discharge	HS	2.72	1.6	1.2
	NS	2.67	2.1	1.6

HS-hypertonic saline, NS-normal saline

follows: Postal nasal discharge: absent=1, clear=2, purulent=3; Cough: absent=1, mild=2, moderate=3, severe=4.

Due to certain reservations, follow up x-ray examinations were not conducted at the end of the study, although such examinations can help better evaluate the effects of different saline solutions in treatment of CS. Both groups reported an increase in clear and purulent nasal secretions within the first 10 days of treatment, which decreased significantly later during the course of study. Patients did not express any discomfort with secretions, which results in improved drainage and ventilation of sinus cavities.

Our findings also suggest that hypertonic saline solution is more helpful than normal solutions in treatment of CS. This appears to result from the osmotic effects of hypertonic solutions that induce shrinking of swollen mucosa and cause a transient hyperosmolarity of airway surface liquids. The latter trigger calcium ion release and results in increased ciliary beat frequency. All these changes translate into improved mucocilliary clearance.[3,5] In spite of the discomforts experienced during the first few days of treatment with hypertonic saline solutions, it is an inexpensive and effective remedy for chronic sinusitis.

References

1. Shapir G, Rachefsky S. Introduction & definition of sinusitis. J Allergy Clin Immunol 1992; 90: 417-418.
2. Shoseyou D, Bibi H, Shai P, Shseyou N, Shazberg G. Treatment with hypertonic saline versus normal saline nasal wash of pediatric chronic sinusitis J Allergy Clin Immunol1998; 101: 602-605.
3. Davices E, Anderson SD, Gondal, Eber S et al. Inhalation of hypertonic saline aerosol enhances mucocilliar clearance is asthmatic and healthy subjects. Eur Respire J 1996; 9: 725-32.
4. Eng PA, Morton J, Douglass, JA, Riedler J, et al. Short-term efficacy of ultrasonically nebulized hypertonic saline in cystic fibrosis. Pediatr pulmonol 1996; 21: 77-83.
5. Pavi D, Thomson MI, Clarke SW. Enhanced clearance of secretion from the human lung after the administration of hypertonic saline aerosol. Am Rev Respir Dis 1978; 117: 199-203.

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Twins and cystifc fibrosis

Cystic Fibrosis (CF) is an inherited disease of the exocrine glands. Symptoms may include pulmonary disease, pancreatic exocrine insufficiency, male infertility, meconieum ileus, and an increase in the concentration of sweat electrolytes, and dehydration. It is the most common autosomal recessive disease in Caucasians with an incidence of 1:2000, but considered to be rare in Arabs and the Far East population.[1] The disease is caused by alterations in the cystic fibrosis transmembrane conductance regulator (CFTR), which functions as a chloride channel and regulator of other channels in epithelial cells.[1] The incidence varies from one Arab country to another and estimated to be in Bahrain as 1 in 5800, in Kuwait as 1 in 3500, and in Jordan as 1 in 2500 and in Saudi Arabia as 1 in 4243.[1] 1548delG is considered the most CFTR mutation in Saudi Arabia and constitute 15-35%. D-F508 which causes CF in 65-85% of the Caucasian population,[1] was found to less frequent in the Saudi population 4-10%. Twins with CF is a rare entity and is estimated that 1 in 22 Caucasian are CF carriers. With calculating random mating, the chance of monozygotic twins with CF would be about 1 in 0.6 million pregnancies; conversely the chance of dizygotic twins with CF would be about 1 in 0.75 to 1.25 million pregnancies.[1] The frequency of CF carriers and twins pregnancy in Saudi Arabia is unknown.

Patient 1. Twin 1, premature delivery with Cesarian section at 27 weeks of gestation. Ventilated for 3 weeks. Referred to our center at 3 years of age with history of repeated chest infection and heart murmur. Echocradiogram (ECHO) at that time showed Atrial septal defect (ASD) secundum about 7 mm in diameter with left (Lt) to right (Rt) shunt. Normal valves, no significant Rt cardiac dilation, good Lt ventricular function and normal pulmonary venous drainage. The ASD finding was out of proportion to the severity of her illness, where she was seen again with her twin sister by the pediatric pulmonologist at 5 years and 6 months of age for further evaluation. Her weight was 13.4 kg and height 101.5 centimeters, both are below the 5th percentile for age. There was no clubbing, or respiratory distress, and chest examination showed bilateral crepitaion and rhonchi. Chest xray (c-xray) showed peribronchial wall thickening, mild cardiomegaly, prominence of pulmonar artery (PA), dilated lung vessels but normal lung fields. Arterial blood gas (ABG) was normal. She under went ASD

Table 1 - Twins with cystic fibrosis literature review.

Ref	Status 1/2	Age at diagnosis 1/2	Signs/Symptoms	Other diseases 1/2	Alb 1/2	Hb 1/2	Sweat CL 1/2	Comments 1/2
HB	Monozygotic, term females	2.5/5.5 years (yr)	Chest infection, failure to thrive (FTT), pancreatic insufficiency (PI), FVC 65%, FEV1 55%	1. ASD, device implant, 2. VSD, closed spontaneously, FVC 50%, FEV1 50%	39/40	102/112	110/121	Both, homozygous 1548 delG, severe GER, 1. Pseudomonas +ve 2. Negative culture
2	Monozygotic, term femals	4 months	Anemia, decrease Alb, FTT Edema	No	19/17	8/7.3	113/106	-
3	Monozygotic, term ?/?	4 wks	Anemia, Edema	1. E coli septicemia/ 2. Heart murmur	-	6.7/6.1	113/96	2. Blood transfusion, albumin infusion
4	Dizygotic, 37 wks male/ female	9 wks	FTT, Edema, Anemia Jaundice, decrease Alb	No	23/23	6.3/7.5	92/100	Blood transfusion
5	Monozygotic, term ?/?	29 wks gestation	Meconieum ileus (MI)	1. Bowel resection, 2. No surgery	-	-	117/123	1/2 prenatal echogenic pancreas at 29 wks
6	Dizygotic, term ?/?	10 wks gestation/at birth	Chronic villous sample: DNA analysis	-	-	-	-	1. Aborted, 2. Normal
7	?, term ?/?	-	-	-	-	-	-	Similar strains of pseudomonas cultured
8	Dizygotic, term male/ female	4.7/9.7 years	1. Liver disease, PI, mild lung disease, 2. PS, moderate lung disease	-	-	-	-	Compound heterozygous (G85E/?) 1. FEV1 (86/60%)
9	?, term females	At birth	1. MI, colostomy, Aspergillus pneumonia at 6 yr, repeated admission at 12 yr, home IV treatment at 16 yr, ciprofloxacin at 18 yr.	2. No symptoms at birth, aspergillus pneumonia at 6 yr, repeated admission at 10 yr, home I.V. Rx at 21 yr, ciprofloxacin at 18 yr.	-	-	96/100	FVC (62/88%), FEV1 (63/73%), seudomonas at 19 yr, DNA analysis D-F508/ R347H+D979A, Mother-Japanese, Father-German

HB-Present report, Sweat CL-Sweat chloride mmol/L, Hb-Hemoglobin in gm/L, ASD:Atrial septal defect, FEV1-Forced expiratory volume in one second, 1/2-Twin 1/Twin 2, FTT-Failure to thrive, VSD:Ventricular septal defect, FVC-Forced vital capacity, Alb-Albumin, (?)-Unknown/not mentioned, PS-Pancreatic sufficiency.

device implantation with good results and repeat ECHO post operative in 3 occasions showed no residual ASD, and good ventricular functions. She was started on Pancreatic enzymes replacement, fat soluble vitamins and antibiotics. She showed marked clinical improvement with progressive weight gain. Screening for CFTR, showed homozygous for 1548delG.[1] Barium swallow showed severe gastresophageal reflux (GER). Pulmonary function test (PFT) showed moderate obstructive lung disease (Table 1).

Patient 2. Twin 2, required oxygen for 3 weeks after birth. The patient was picked up by screening with her sister at 5 years and 6 months of age. She has similar history of repeated chest infection and failure to thrive (FTT). Echo study showed small muscular ventricular septal defect (VSD) with Lt to Rt shunt, patent foramen ovale with Lt to Rt shunt, no ventricular enlargement and no other abnormality. C-xray showed peribronchial wall thickening. She was given similar treatment regimen as the twin sister. Her growth improved, but remained identical

mutation as her twin sister with homozgous for 1548delG. Barium swallow showed similar finding of severe GER. PFT showed moderate obstructive airway disease (Table 1).

We have identified two twins that combined 3 rare conditions: twins pregnancy, CF and cardiac anomalies in the same patient. This case is considered the first reported monozgotic twins with CF in the Arabian world. We have also shown that, although they have identical CFTR mutations, they differ in their clinical presentation and response to treatment. Review of the world literature has shown 8 similar reports,[2-9] none of the reports showed cardiac anomalies. It also shows that twins with CF, either monozygotic or dizygotic, differ in their presentation, severity of their disease and response to treatment (Table 1). Detailed examination and specific care should be given to each twin according to the severity of disease. Factors other than genetic effect may explain these differences and further studies should be directed to explain this phenomenon.

References

1. Banjar H. Overview of cystic fibrosis. Mid East Pediatr 1999; 4: 44-48.
2. Blumenthal I, Fielding DW. Hypoproteinaemia, edema, and anemia: an unusual presentation of cystic fibrosis in dizygotic twins. Arch Dis Child 1980; 55: 812-814.
3. Paxson CL Jr, Lock J. [Letter]. Idiopathic hyoproteinemia in twins. JAMA 1974; 230: 1257-1258.
4. Bass HN, Miller AA. Cystic fibrosis presenting with anemia and hypoproteinemia in identical twins. Pediatr 1977; 59: 126-127.
5. Denholm TA, Crow HC, Edwards WH, Simmons GM Jr, Marin-Padilla M, Bartrum RJ Jr. Prenatal sonographic appearance of meconium ileus in twins. AJR 1984; 143: 371-372.
6. Super M, Ivinson A, Schwarz M, Giles L, Elles RG, Read AP, et al. Clinic experience of preatal diagnosis of cystic fibrosis by use linked DNA probes (published erratum) Lancet 1987; 2: 782-784.
7. Boukadida J, De Montalember M, Lenoir G, Scheinmann P, Veron M, Berche P. Molecular epidemiology of chronic pulmonary colonization of pseudomonas aeruginosa in cystic fibrosis. J Med Microb 1993; 38: 29-33.
8. Vazquez C, Antinolo G, Casals T, Dapena J, Elorz J, Seculi JL, et al. Thirteen cystic fibrosis patients, 12 compound heterozygous and one homozygous for the missense mutation G85E: A pancreatic sufficiency mutation with variable clinical presentation. J Med Genet 1996; 33: 820-822.
9. Hojo S, Fujita J, Obayashi Y, Ohnishi T, Yamaji Y, Okada H, et al. Two cases of cystic fibrosis in Japanese/German twins (Japanese). Nihon Kyobu Shikkan Gakkai Zasshi. Japanese J Thora Dis 1997; 35: 1259-1264.

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Survival of extreme pre-term infants in an intensive care set up

Sir,

It remains a matter of financial and ethical controversy in developing countries regarding how far one should go in salvaging extreme pre term infants, mainly because of the financial and other resource restrictions. At the same time the fertility rate is very high in these countries, leaving the question as to the worth of saving an extreme pre term infant with months of intensive care and sometimes life long morbidity.

In his article[1] Shabih Manzar has noted that in the subgroup of 26 weeks gestation, the survival rate is 44% in the Omani population which is less than the Western reports. A retrospective analysis of the 1998 and 1999 statistics of Sultan Qaboos University Hospital, has shown that the survival rate of 26 weeks gestation babies is 80% (Table 1) which is equivalent to any Western standards. One of the reasons why survival rates of extreme preterm babies in the early days were low, was that, intensive care was provided to these babies only if they survived the initial few days.

Now that the policy has been relaxed and good intensive care being given to these extreme low birth weight (ELBW) babies, the outcome has much improved (Table 1 & 2). At the same time if the baby has grade IV intra-ventricular hemorrhage or severe birth asphyxia we prefer not to take aggressive measures. The incidence of pneumothorax, intra-ventricular hemorrhage, retinopathy of prematurity and necrotizing enterocolitis has all been negligible. Two babies had grade IV intra-ventricular hemorrhage and both these cases were out-born

Table 1 - Neonatal admissions and mortality according to gestational age (Jan 1998-Dec 1999).

Gestational age (in weeks)	Admissions	Mortality No (%)
26	10	2 (20)
28	16	2 (12.5)
30	24	2 (8)
32	48	0 (0)
34	51	2 (4)
36	34	0 (0)
>37	328	15 (5)
Total	511	23 (4.5)

Table 2 - Neonatal admissions and mortality according to birth weight (Jan 1998-Dec 1999).

Birth weight (in grams)	Admissions	Mortality No (%)
750-999	16	2 (12.5)
1000-1499	50	4 (8)
1500-1999	75	4 (5)
2000-2499	80	2 (2.5)
>2500	290	11 (4)
Total	**511**	**23 (4.5)**

babies referred ex-utero to our hospital. Both the babies required ventriculo-peritoneal shunt for post-hemorrhagic hydrocephalus. This again highlights the point that in-utero transfer is better in terms of intact preterm survival. Two-third of 4.5% mortality in the present study (Table 2) is due to congenital anomalies like Potter's syndrome, and non-salvageable inborn errors of metabolism rather than due to prematurity. Another interesting observation is that in addition to survival, the neuro-developmental outcome has been very good on regular follow-up. This good outcome is probably because of antenatal steroids, good obstetric management and intensive neonatal care provided, with minimal and gentle ventilation with permissive hypercapnea, total parenteral nutrition, early trophic feeding and good human, electronic and biochemical monitoring. High caliber nursing care with intensive monitoring, supervision and regular in-service training have to be specially mentioned.

In conclusion, we would like to point out that the survival rate of ELBW pre-term Omani babies is no longer lower than the western counterparts, provided we give standard intensive care with asepsis, as well-evidenced by our statistics over the last two years. To our opinion, 25-26 weeks should be the maximum cut-off point for aggressive intensive care decisions, rather than up to 22 weeks. Long term follow-up of these babies for neuro-developmental outcome and minimal brain dysfunction, however is warranted.

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References

1. Manzar S. Survival pattern among extreme pre term infants. Saudi Medical Journal 2000; 21: 168-170.
2. Katz VL, Bose CL. Improving survival of the very premature infants. J Perinatol 1993; 13: 261-265.

Correspondence

Hearing loss in a textile factory.

Sir,

Shakhatreh et al[1] have brought out the importance and preventive aspects of occupation related hearing loss in an educative manner. We are submitting our comments as follows:- a) By and large, the risk of incurring hearing loss begins with prolonged exposure to sound approximately 75dB(A),[2] whereas in the authors' series,[1] one of their subjects developed hearing loss at a noise level of 46-73 dB(A). As per the authors, hearing loss in this case was not due to factors other than exposure to noise. Hence, the possible explanation could be hereditary predisposition to noise induced hearing loss.[3] Have the authors' ruled out the possibility of hereditary hearing loss in this subject? This is important from the point of industrial compensation (or) worker's compensation act;

b) As far as the recommendations with reference to noise induced hearing loss in an industrial area is concerned two more things to be included are systematic record keeping and worker notification

when problems are detected;[4] and c) Regarding health education, both the health care workers and the public must be well informed that (i) the ears we get at birth are the only ones we will ever have and so we should learn to treat them with better care, (ii) loud noise will cause damage sooner or later but it is a matter of time, (iii) the heavier the dose and longer the duration of exposure, the greater the damage, and (iv) the cells of the inner ear are incapable of regenerating and hence all damage to the inner ear is irreversible, thus noise induced hearing loss is always permanent .[3]

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Reply from the Author

In response to your letter, I feel grateful of Dr. Uma for his comments on the paper entitled "Hearing Loss in a textile factory". The case under consideration was due to exposure to noise after taking a comprehensive and detailed history of that case. However, Dr. Uma's comment should be highly considered when investigating such an issue. We hardly emphasize on Dr. Uma's points regarding record keeping and worker notification when problems are detected as we have only two ears and their damage due to noise exposure is irreversible.

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References

1. Shakhatreh FM, Abdul-Baqi KJ, Turk MM. Hearing loss in a textile factory. Saudi Medical Journal 2000; 21: 58-60.
2. WHO. Health and environment in sustainable development. Document WHO/EH6/97.8 Geneva, World Health Organization 1977.
3. Karttunen A. Declaring war on noise. Work health safety (Finnish Institute of occupational health, Finland) 1998; 24: 26.
4. Yassi A, Kjellstrom T, Dekok T, Guidotti T. (Edrs) Nature of environmental health hazards (Chapter 2) 2.4 Noise and vibration. In: Basic environmental health: Preliminary version. WHO/EHG/98.19 English, Geneva, World Health Organization 1998; 63-66.

Cardiac troponin and acute coronary syndromes in Saudi Arabia

Sir

In a recent issue of the Saudi Medical Journal, Saadededdin et al,[1] have succinctly reviewed the vital role of markers of myocardial injury in the assessment and management of patients with acute coronary syndromes. Cardiac troponins should have a key role in different regions of Saudi Arabia in an early diagnosis of myocardial infarction. Blood troponin levels would as well assist clinicians in their therapeutic interventions with low molecular weight hearing, glycoprotein IIb/IIIa inhibitors and early percutaneous revascularization. Nevertheless, it would be essential to appreciate possible false-positive and false-negative results during tropnin quantifications.[2] The sensitivity and specificity of troponin assay would relate to assay technology involving either analyzers or employing 1-2 step, simple tests. Troponin I assay is accomplished in laboratory premises by multi-step procedures using analyzers.[2] The assay procedure has also been simplified to an immunochromatographic test which relies on a chromatogaphic membrane, immobilized antibody zones, mobile rehydrable reagent and color particles applied to a filter paper. The reagents are to be stored between 15 to 25^{o}C, the recommended temperature for quantification.

Enzyme immunoassays using analyzers apart, immunochromatographic troponin I assay would be invariably carried out in extremes of ambient temperatures in Saudi Arabia. In many areas, ambient temperatures might be around 10^{o}C with no facilities to maintain such temperatures around 20-25^{o}C. While blankets would protect patients with a suspected myocardial disease, the bedside troponin I assay would be carried out at low temperature only. Similarly, high ambient temperatures around 40^{o}C would alter sensitivity or specificity of troponin I assay at the patient's bedside.

Such pitfalls should be appreciated, they would misguide the clinicians. Prevailing upon the manufacturers to offer environment-resistant kits for troponin assay at the bedside would best eliminate them. Furthermore, it is essential that kits are handled judiciously and not used after their expiry date. Following the use of pre-tested blood for HIV with expired or inappropriately stored antibody reagents, the risk of HIV transmission was at least six times higher than expected.[3]

Certainly, good quality troponin kits when stored carefully and used before the expiry date would be invaluable for detection of minor myocardial

damage, assessment of infarct size and the judicious therapeutic intervention in acute coronary syndrome patients even in remote parts of Saudi Arabia.

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Reply from the Author

We thank Dr. Subhash C Arya for his comments on our paper and we agree with the relevant practical comments made by him. High ambient temperatures can significantly alter the sensitivity and specificity of any qualitative tests. Accordingly, rapid qualitative tests for troponin I and troponin T can be affected especially when the strips used in the test are not carefully stored at the recommended temperature. This is more likely to occur when qualitative troponin rapid tests are carried out in the field by the paramedics. On the other hand, this problem is less likely to occur with troponin tests (qualitative or quantitative) done in the emergency room or in the medical laboratory since such tests are usually carried out under optimal conditions. Also the use of expired kits can significantly alter the results of any test and therefore should not be practiced.

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References

1. Saadeddin SM, Habbab MA, Ferns GA. Cardiac markers for assessing the acute coronary syndromes: A focus on cardiac troponins. Saudi Medical Journal 2000; 21: 228-237.
2. Bock JL. Cardiac troponin. Amer J Clin Path 1999; 112: 739-741.
3. Consten ECJ, Van der Meer JTM, Wolf F, Heij HA, Henry PC, Van Lanschot JJ. Risk of iatrogenic human immunodeficiency virus infection through transfusion of blood tested by inappropriately stored or expired antibody assays in a Zambian hospital. Transfusion 1997; 37: 930-934.

Erratum

In manuscript Double dislocation of the interphalangeal joints in the finger by Mohammed A. Mesmar, Saudi Medical Journal 2000; Vol.21: 493-494, figures 1a and 1b should appear as follows:

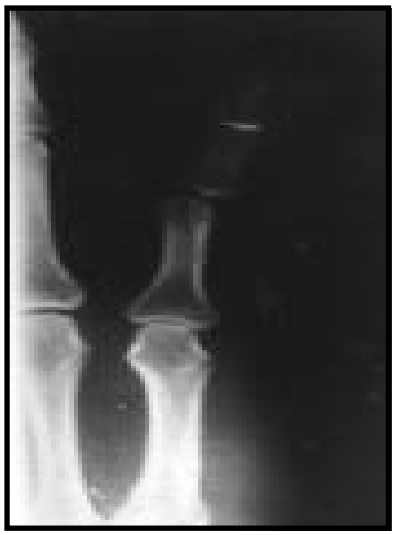

Figure 1a - X-ray films of the right little finger - Anterior-posterior view.

Figure 1b - X-ray films of the right little finger - Lateral view.

Book Reviews

Diabetes Mellitus in the Elderly

JAMES W. COOPER. 84pp. **Price:** USD 39.95. **Publisher:** Pharmaceutical Products Press, USA. **Date of Publication:** 1999. **ISBN:** 0-7890-0682-0.

This book has been co-published simultaneously as Journal of Geriatric Drug Therapy, Volume 12, Number 2 1999. This is a small book consisting of 84 pages and divided into four sections. The book provides an updated and comprehensive review of the diagnosis and treatment of diabetes mellitus in the elderly.

The first section presents a review of the pathophysiology of diabetes in aging. As human aging is associated with significant alterations in glucose metabolism and insulin action, together with a 3-fold higher prevalence of diabetes mellitus and impaired glucose tolerance. Understanding the multi-organ pathophysiology of diabetes in the elderly is clinically relevant, because present and future pharmacologic therapies aim to reverse specific organ defects and often act synergistically to decrease hyperglycemia.

The second section evaluate the various available pharmacologic agents and combination of these agents in the management of type 2 diabetes in the elderly patient, with an emphasis on oral agents. With four oral agents, acarbose, metformin, repaglinide and troglitazone for type 2 disease, and the American Diabetes Association (ADA) revised guidelines that suggest the trial of all oral agents before resorting to insulin, the authors offer practical insight to the rational use of these agents.

The European experience with acarbose is discussed in the third section. The last section emphasizes the safe use of insulin in the elderly with either type 1 or type 2 diabetes, the co-morbidities, nutritional issues, pharmacodynamics and physiologic effects of various insulin preparations and regimens.

The book is well written, informative, concise and easy to read. Also, it includes numerous references. I would recommend this book for any physician or medical student who wants useful information on diabetes mellitus in the elderly.

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Clinical Diagnosis & Management of Alzheimer's Disease

SERGE GAUTHIER. **Price:** PS 65.00. **Publisher:** Martin Dunitz Publishers, UK. **Date of Publication:** 1999. **ISBN:** 1-85317-655-9.

Alzheimer's disease is a major health problem all over the world and for many years has been under-diagnosed and under-treated. There is an overwhelming interest for better understanding of Alzheimer's disease, as the prevalence of this disease doubles every 5 years, from age 65 to age 85. It is estimated that there are 10 million cases of Alzheimer's disease worldwide, thus escalating socioeconomic costs for all countries.

The first edition of this book was published in 1996. The book became immensely popular among practicing physicians and caregivers of this unfortunate disease. The multidisciplinary authorship of this second edition truly represents global response to this major health problem. The results of recently published clinical trials of epidemiological studies, electrophysiological tests, stabilization therapies, functional brain imaging, molecular genetics have been incorporated in this edition which will have significant impact on the quality of life of patients with Alzheimer's disease.

The principle objective of this edition is clearly focused on clinical aspects of Alzheimer's disease. This edition contains seven chapters beginning with the introduction of the disease process followed by diagnosis, natural evolution, medical management and community and institutional care. The authors have deliberated on ethical and legal issues and finally concluded with the future diagnosis and management of Alzheimer's disease. Each chapter is richly supplemented with comprehensive and up-to-date references, a very rewarding experience for readers and researchers alike. All chapters are dealt with highest level of scientific content and is professionally very rewarding.

The book is recommended for all levels of physicians, internists, neurologists, psychologists, psychiatrists as well as those who are engaged for family, community or institutional care of AD patients. This book is among those very few on the subject, which will have far greater readership, than what may have been anticipated, both for its scientific merit, and the quality of its presentation.

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Book Reviews

Symptom Management in Multiple Sclerosis

RANDALL T. SCHAPIRO. 204pp. **Price:** USD 19.95. **Publisher:** Demos Medical Publishers, USA. **Date of Publication:** 1998. **ISBN:** 1-888799-22-6.

Symptom Management in Multiple Sclerosis, 3rd edition (1998) is 204 pages, small size, easy to read book by Randall T. Schapiro. This book provides a long term experience in managing patients with a wide variety of multiple sclerosis (MS) symptoms.

The book consists of 3 parts. Using simple non-medical language the book starts part one with simple anatomy to define MS and introduce the available medication to treat the disease process.

Part two is the main portion of this book and covers the management of MS symptoms mainly non-pharmacological measures, the symptoms covered include fatigue, swallowing and swollen ankles etc.

The last part of the book covers general health aspects in regard to MS, including diet, exercise and rehabilitation measures.

The book is directed primarily to MS patients and their families as well as all health care professionals caring for MS patients. As a physician, the book would provide you with simple terms and ways to help you in managing and teaching your patient in regard to specific symptomatic problems, mostly in non-pharmacological measures.

The quality of coverage is wide and excellent for the main audience. I would strongly recommend this book for doctors caring for MS patients, especially in non-English speaking communities, to help and assist him to explain to his patient the simple way and measure about the common MS symptoms and how to deal with them as a patient.

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Benign Childhood Partial Seizures & Related Epilepsy Syndromes

CP PANAYIOTPOULOS. 406pp. **Price:** PS 59.00. **Publisher:** John Libbey & Co. **Date of Publication:** 1999. **ISBN:** 0-86196-577-9.

This book's main content is of specific types of epileptic syndrome in childhood. The first part is of general aspects of diagnosis of epilepsy and EEG, roles in the diagnosis and management of epilepsy. Part two covers the rolandic epilepsy and parts 3,4, and 5 covers the occipital seizure and related syndrome.

I found this book of excellent quality in its coverage of the subjects. It is full of valuable information and detail. The style of giving the information is comprehensive and easy to follow.

The first part of this book is important to all physicians in particular non-neurologist to understand the diagnosis and the role of the EEG.

The other parts of the book with the extensive detail are really of interest only to the specialist.

I would recommend this book to be part of the Medical Library to every hospital.

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