ment of acute mountain sickness (AMS). Thanks to the authors for their contribution.

We know that high altitude leads to some negative effects without acclimatizing on pulmonary and cardiovascular systems. AMS is a syndrome due to the rapid ascending to high-altitude in aviators and mountaineers. It is a serious health problem especially in obese subjects. In present study, we want to learn that the subjects were taken to high altitude as volunteers or part of their duties. In our country, we perform like these researches in hypobaric chamber with simulating hypoxia because of legal issues. At hypobaric chamber, we can monitor oxygen saturation, blood pressure and heart rhythm of the subjects so we can easily stop the hypoxia and give oxygen to the subjects. We have some questions about the design of this article. Did the subjects take oxygen when the oxygen saturation was below the threshold value? It could be emphasized that the subjects stayed at high altitude for 24 hours or not and individuals were taken at what speed and which vehicle to high altitude.

In relation to these, we also know that there are some recent studies about the effects of high altitude on cardiac parameters (2). For example we reported a case of cardiac decompression sickness on an aviator (3) and an asystolia during hypobaric chamber training 30.000 feet (4). In another study, we investigated the acute effects of hypoxia on noninvasive electrocardiographic parameters in aviators (5).

In conclusion, although the obese and non-obese subjects had same conditions before high altitude, what happened there and how high altitude was caused problems for the obese. The subject is very important and we believe that these findings will act as a guide for further studies.

Cengiz Öztürk, Şevket Balta, Süleyman Metin[1], Tolga Çakmak[1]
Department of Cardiology, Eskişehir Military Hospital; Eskişehir-*Turkey*
[1]Aerospace Medicine, Gülhane Medical Faculty; Eskişehir -*Turkey*

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Address for Correspondence: Dr. Cengiz Öztürk,
Eskişehir Askeri Hastanesi, Kardiyoloji Bölümü, Eskişehir-*Türkiye*
Phone: +90 222 220 45 30
Fax: +90 222 230 34 33
E-mail: drcengizozturk@yahoo.com.tr
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Author`s Reply

Authors of this mentioned article did not send any reply for this Letter to Editor, in spite of our insistently requests.

Shisha versus cigarette smoking and endothelial function

To the Editor,

The recent report on "Shisha versus cigarette smoking and endothelial function" is very interesting. Selim et al. (1) published, reported in 2013 December issue of The Anatolian Journal of Cardiology that "Shisha smoking has a more hazardous effect on brachial artery endothelial-dependent flow mediated vasodilation compared to cigarette." This conclusion is very interesting and should be discussed. In fact, the recent report showed that there was no difference in aerosol produced by cigarette and shisha (2). There are many factors that affected the final measured outcome. The dosage has to be mentioned. Poredos et al. (3) demonstrated that "smoking is associated with dose-related increase of intima-media thickness and endothelial dysfunction." The genetic underlying of each subject is also important factor to be considered.

Somsri Wiwanitkit, Viroj Wiwanitkit[1]
Wiwanitkit House, Bangkhae, Bangkok-*Thailand*
[1]Hainan Medical University, China; Adjunct professor, Joseph Ayobabalola University-*Nigeria*

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Address for Correspondence: Dr. Somsri Wiwanitkit,
Wiwanitkit House, Bangkhae, Bangkok-*Thailand*
Phone: +234 805 789 7005
E-mail: somsriwiwan@hotmail.com
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Mortal suicidal acetazolamide intoxication in a young female

To the Editor,

Acetazolamide is a carbonic anhydrase inhibitor used in the treatment of glaucoma, epilepsy, benign intracranial hypertension, metabolic alkalosis and is also used as a diuretic. Hyperchloremic metabolic acidosis, renal stones, renal potassium wasting are some toxicities of chronic acetazolamide usage. In elderly or diabetic patients and

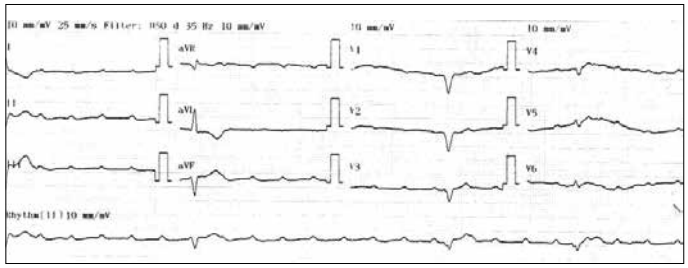

Figure 1. 12-ECG showing complete atrioventricular block and escape beats

patients with decreased renal function, mild to severe metabolic acidosis is seen more commonly (1, 2).

However, there is scarce information about clinical presentations of patients with acute acetazolamide toxicity. There is only one report defining an accidental poisoning of an infant who was treated successfully with sodium bicarbonate (3). To the best of our knowledge, this is the first case to implicate acute acetazolamide intoxication as a cause of deep metabolic acidosis and secondary total atrioventricular block and death in an adult who committed suicide.

A previously healthy 39-year-old female was brought to our emergency department by her relatives because of drowsiness. Blood pressure and heart rate were 80/40 mm Hg, 19 beats/min respectively. ECG showed complete atrioventricular block and heart rate of 16-19 bpm (Fig. 1). Arterial blood gases under nasal oxygen revealed a pH of 7.119, pO_2; 95 mm Hg, pCO_2; 14 mm Hg, lactate; 12.8 mmol/L, HCO_3; 6.9 mmol/L and although bicarbonate replacement didn't change it. Intermittent boluses of sodium bicarbonate, followed by an infusion, were commenced. She was intubated because of severe dyspnoea. Blood sodium, potassium and calcium levels were normal so didn't replacement it. Dopamine 3 mcg/kg/min and dobutamine 10 mcg/kg were commenced. Transvenous pacing was started immediately. Routine blood chemistry revealed normal electrolytes, elevated troponin, creatinine and liver function tests. Transthoracic echocardiography showed mild global hypokinesia of left ventricle. Additional doses of bicarbonate and other supportive treatment did not work and she died because of circulatory collapse a few hours after admission. Next day, her relatives found an empty acetazolamide box belonged to her father in her bag.

Main pathology in our patient was resistant deep metabolic acidosis. Although the exact time was not known she had ingested 2500 mg acetazolamide before clinical presentation. We think that this was the main reason of the severe acidosis which caused cardiac complications. Lactic acidosis secondary to hemodynamic deterioration probably contributed to the deep acidosis. Most probable reason of elevated troponin, creatinine and liver function tests was ischemic injury because of bradycardia and hypotension.

Life-threatening metabolic acidosis during acetazolamide therapy has been observed only in patients with renal impairment such as diabetics, elderly patients. Overdose with acetazolamide and its management have been reported rarely and we were unable to identify any reports implicating suicide attempt with acetazolamide.

Almost all reports define metabolic side effects of chronic acetazolamide intoxication on brain, gastrointestinal and neuromuscular system, especially in patients with decreased renal function (1, 2, 4, 5). However our case has shown that acute high dose acetazolamide can also cause deep metabolic acidosis and mortality in previously healthy individuals.

In addition to supportive treatment, bicarbonate should be considered as treatment for metabolic acidosis resulting from acetazolamide intoxication. Therefore, hemodialysis may be helpful in the management of acetazolamide overdose, particularly when complicated by renal failure (2, 5). There is no amiable evidence based information to use activated charcoal on acetazolamide poisoning.

Servet Altay, Erkan İlhan[1], Seçkin Satılmış[2], Gülşah Tayyareci[2]
Clinic of Cardiology, Edirne State Hospital; Edirne-*Turkey*
[1]Clinic of Cardiology, Universal Çamlıca Hospital; İstanbul-*Turkey*
[2]Clinic of Cardiology, Dr. Siyami Ersek Hospital; İstanbul-*Turkey*

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Address for Correspondence: Dr. Servet Altay,
Edirne Devlet Hastanesi, Kardiyoloji Bölümü, Edirne-*Türkiye*
Phone: +90 506 377 97 51
E-mail: svtaltay@gmail.com
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