



Medication storage temperatures in primary response vehicles

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To the Editor: Paramedics practising pre-hospital emergency advanced life support care carry a range of parenteral medications including antiarrhythmics, analgesics, hypnotics and anticonvulsants.¹ Unless otherwise indicated, medications should be stored at controlled room temperature (CRT) to limit accelerated thermal degradation caused by temperatures above CRT.²

Compliance of storage temperatures with CRT can be determined by measuring the mean kinetic temperature used in place of the arithmetical mean of a set of temperature measurements because the former takes into account the greater contribution of higher temperatures to thermal degradation of medications.³ CRT storage conditions are exceeded if the mean kinetic temperature over a period of time exceeds 25°C, if any temperature over that period of time exceeds 40°C, or if temperature spikes between 30°C and 40°C occur that exceed 24 hours during that period.²

In South Africa, medications are typically kept permanently in a primary response vehicle until used for patient care or are discarded. Currently there is no recommendation, nor is it standard practice, to take any special precautions to keep medication storage temperatures within CRT in these vehicles. Therefore, medications are exposed to storage temperatures which may be above CRT, possibly for long periods.

The aim of this study was to document medication storage temperatures over a continuous 12-month period in the drug storage compartment of a typical primary response vehicle under operational conditions in Johannesburg.

Methods

Two electronic temperature data loggers (Model 42270 temperature/humidity data logger, Extech Instruments, Mass., USA) were placed in the drug storage compartment of a primary response vehicle (2003 Toyota Corolla RSI). Temperatures were recorded at 30-minute intervals between 1 August 2004 and 31 July 2005, and stored in an electronic spreadsheet application. Mean kinetic temperatures were calculated using a validated software application (Stability System II [MKT] version 1.6, ScienTek Software Inc., Calif., USA).

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Results

Over the study period, 17 449 temperature measurements were recorded and downloaded. The highest single maximum temperature recorded was 40.5°C in December 2004. Monthly mean kinetic temperatures were above the threshold recommended for compliance with CRT (25°C) for a period of 6 consecutive months (Fig. 1). The mean kinetic temperature for the period October 2004 - March 2005 (26.9°) was also above CRT, while the overall mean kinetic temperature over the entire 12-month period was 24.7°C.

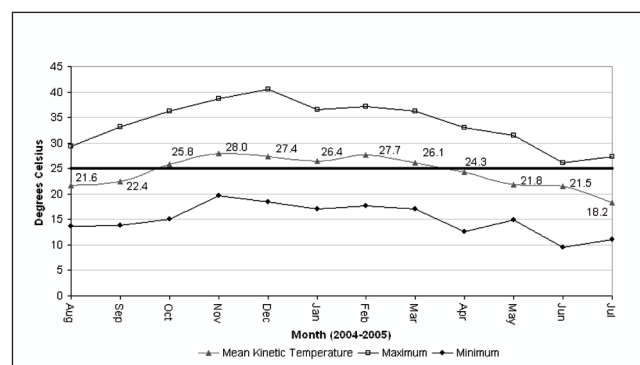


Fig. 1. Medication storage temperature in a primary response vehicle.

Discussion

The effects of deviations from CRT on the chemical stability of any medications were not examined. Concerns may be justified regarding the accelerated degradative effects of high medication storage temperatures, considering that the mean kinetic temperature was above the recommended threshold over the 6 hottest months of the year.

Duration of exposure to higher temperatures must also be an important factor in determining degradative effects on medications stored in primary response vehicles. This knowledge can enable medications with a high turnover to be protected from the damaging effects of prolonged exposure to higher temperatures. The vehicle design can also influence drug storage temperatures; for example, a hatchback allows the vehicle's air conditioning system to reduce temperatures in the drug storage area while driving. The study vehicle was not a hatchback.

A study is under way to quantify chemical degradation occurring in selected medications (adrenaline, atropine, midazolam, diazepam and morphine) after storage in a primary response vehicle.



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Bipolar diathermy for the outpatient control of posterior epistaxis

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To the Editor: Epistaxis remains a major cause of emergency otolaryngology admissions.

Epistaxis is classified as anterior or posterior. Anterior bleeding sites are bleeders anterior to the bony nasal aperture, and are relatively easy to manage because the vessel may be visualised and controlled with basic equipment such as a headlight, nasal suction device and nasal speculum and simple cautery techniques.

Posterior epistaxis, which originates posterior to the bony nasal aperture, poses a more difficult problem. Generally these bleeders are not visible without the use of nasal endoscopy, especially if they occur on the lateral wall of the nasal cavity, a complex anatomical area characterised by the turbinate bones and meati, which have recesses between them.

As a result of these factors the traditional treatment of posterior bleeds has been the use of 'blind' techniques of nasal packing with ribbon gauze soaked in BIPP (bisthmus iodine and phosphate paste) and inflating the balloon of a Foley's catheter in the patient's nasopharynx (this allows for control of the epistaxis where the actual bleeding site is not specifically identified).

However, newer technology in the form of nasal endoscopes and bipolar nasal cautery probes potentially permits the localisation and cauterisation of the bleeding site in an awake patient in an outpatient setting. Nasal endoscopy and a bipolar diathermy probe have been found effective in localising the source of bleeding in the outpatient clinic, in the hands of an expert and dedicated nasal endoscopist.^{1,2}

Blind nasal packing is the management protocol for patients presenting with posterior epistaxis in tertiary academic hospitals in South Africa, as was our department's policy before this study. These patients all had their noses packed, were observed as inpatients and had the packs removed 48 hours later. When the pack failed to control the epistaxis the patient would be taken to theatre for localisation of the bleeder and diathermy, or endoscopic sphenopalatine artery ligation. If this failed, embolisation of the internal maxillary artery was performed.

We conducted an audit looking at the management of 100 successive patients with epistaxis admitted to our institution during 2004. The protocol described above resulted in prolonged hospital stays (average for posterior epistaxis 82 hours) and a low incidence of direct localisation of the bleeding site (only 7.7% of posterior sites, 1/13, were localised). By comparison a national survey in the UK revealed that the mean duration of stay in hospital was 70 hours for epistaxis patients, and that 20% of patients admitted to otolaryngology units were managed by direct control of the bleeding point.³

We therefore decided to investigate the viability of localisation and cautery as a primary intervention for posterior epistaxis in the context of a South African training hospital.

Methods

A prospective study was undertaken on patients presenting to the Tygerberg Hospital ENT department with active posterior epistaxis between 1 June 2006 and 1 October 2007. All patients with traumatic epistaxis, any coagulopathy or Osler-Weber-Rendu syndrome, and those younger than 18 years of age, were excluded from the study.

The sample consisted of both patients who presented to our department directly and those referred to us. Any packs inserted before presentation at Tygerberg Hospital were removed. Any anterior bleeding source was cauterised and

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the patient discharged. Only patients with active posterior bleeding sources were included in the study.

The nose was initially cleared of any blood or clots by suction and blowing the nose. Before endoscopy the rate of bleeding was reduced using pledgets soaked in 2 ml of 10% cocaine. The nose was inspected with a 2.7 mm, 25° rigid nasal endoscope and repeated suctioning. Once the source of the bleeding was located, the cocaine pledgets were re-introduced over that specific site to achieve haemostasis.

The bleeding source was then cauterised using a guarded bipolar diathermy probe. Patients in whom the bleeding was controlled were admitted for overnight observation, without any nasal packing. Patients in whom primary control of the bleeding failed were packed with an anterior and, if necessary, a posterior pack and then taken to the operating theatre for definitive management.

Results

Fourteen patients fulfilled the inclusion criteria. Their ages ranged from 28 to 73 years, with a mean age of 58 years. There were 9 men and 5 women. The site of bleeding was localised to the nasal septum in 10 patients (71%) and to the lateral nasal wall in 3 (21%), while 1 patient (7%) had a combination of bleeding sites.

Eleven (79%) of the 14 patients had successful localisation and cautery of the bleeding site, with no subsequent bleeding during the 24-hour observation period. We were unable to control the bleeding in the outpatient department in 3 patients (21%), who were packed and taken to theatre for further management.

Of the 14 patients in the study, 1 returned with epistaxis. He was the first of those needing definitive control in theatre, synechiae having obstructed the initial endoscopic view. He presented the second time 4 months after the sphenopalatine artery ligation, and on this occasion the source was successfully visualised and controlled in the outpatient department using the endoscopic protocol.

Discussion

Our study confirmed that most posterior bleeding sites can be successfully localised using nasal endoscopy and a systematic examination of the nasal cavity. In 10 (71%) of our 14 patients the site of bleeding was the nasal septum, which is in keeping with other authors' findings.⁴ The fact that most bleeding sites are not located in the more complex lateral nasal wall, a significant factor in permitting accurate localisation of the bleeder, contributed to the high success rates in this management protocol.

Correct technique is important, and while active bleeding is necessary for localisation of the bleeder, excessive bleeding makes visualisation of the specific vessel difficult and prevents effective cautery. Attention to haemorrhage control before endoscopy is a key element in successful localisation. Others have used a combination of cocaine and local infiltration of adrenaline through a spinal needle to reduce persistent bleeding.³ We found topical cocaine effective. As the bleeding subsides the endoscopy more easily reveals the site of bleeding, and the cocaine pack can be placed more accurately over the bleeding vessel. We found that the vessel is usually fairly prominent, often protruding from the septum at 90°. Initial attempts at cautery often resulted in the recurrence of bleeding. The cocaine pledget was then repositioned over the bleeder, and cautery repeated once bleeding had stopped.

We admitted our patients for observation overnight because of socio-economic issues limiting access to transport, should the patients rebleed, and because this was a new protocol for our department. Patients in whom a single, prominent bleeding site was localised and cauterised did not rebleed and were discharged the following morning. Their hospital stay was significantly shortened to 20 hours from a previous mean of 82 hours.

Our results indicate that direct localisation of the posterior bleed should become the routine, first-line management in the treatment of posterior epistaxis. However, our numbers are small and the study is limited as there was only one investigator. We are now investigating the applicability of this technique to all registrar trainees in our department.

Conclusion

Endoscopic examination in cases of posterior epistaxis enables the source of bleeding to be localised and controlled in a high proportion of cases. The benefits to the patient, hospital, doctor and health care system are significant. This should become the routine management of posterior epistaxis in ENT departments in South Africa that have the facility of nasal endoscopy, and in private ENT practice.

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