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INDIAN SOCIETY OF NEUROANAESTHESIOLOGY AND CRITICAL CARE



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ISNACC 2010

**XI Conference of Indian Society of
Neuroanaesthesiology and Critical Care**
29th - 31st January, 2010

Organised by
Department of Anaesthesiology and Critical Care
Park Clinic, Kolkata

1st Announcement

REGISTRATION TARIFF

Registration Fees	Before 15.08.09 (Early Bird)	16.08.09-31.10.09 (Regular)	After 31.10.09 (Late)
Registration with Accommodation at Conference Venue			
❖ Delegate: (ISNACC member)	Rs 11,000/-	Rs 13,000/-	Rs 1500/-
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IMPORTANT DATES TO REMEMBER

❖ Early Bird Registration	Before 15.08.09
❖ Regular Registration	16.08.09 - 31.10.09
❖ Late Registration	After 31.10.09
❖ Last Date of Abstract Submission	30.09.09
❖ Notification of Abstract Selection	31.10.09
❖ Workshop	20.01.10
❖ Conference	30.01.10 - 31.01.10

REGISTRATION TARIFF

Those who prefer not to stay in the conference venue please contact our official travel agent for non package hotels.

Registration Fees	Before 15.08.09 (Early Bird)	16.08.09-31.10.09 (Regular)	After 31.10.09 (Late)
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From the Editor's desk

A DECADE: IS THIS TOO LITTLE OR TOO MUCH?

Ten years in the life of a Society is neither too little nor too much. But, adequate enough to achieve something very useful. Often, one question keeps bothering all our members, “Have we achieved anything in this period? Presumably, the answer is both “Yes” and “No”. Since the inception of our Society we had pondered about organizing the “Mid-term Executive Meeting” to discuss and finalize some important issues. At last, we have achieved success. The first such meeting” was held on 5th July 2009 in Hotel Hindustan International at Kolkata. We must congratulate our General Secretary Dr G. Parameswara and Prof. Amna Goswami, the Organizing Secretary of 2010, ISNACC conference, for arranging the meeting within such short notice.

The second one pertains to sanction of funds to Dr H.K. Venkatesh from Bangalore to carry out a research project. The research project was presented and approved by all the members in the meeting. The society can now boast of funding some useful scientific research through ISNACC Trust. May I appeal to all our senior members to motivate their junior colleagues to be part of our society and reap the benefits of carrying out some substantive research work in the field of Neuroanaesthesia and critical care.

Finally, the Educational Committee submitted its recommendation for starting different certificate/ superspeciality course in Neuroanaesthesia - this was also debated during the meeting. There was unanimity on one thing; to seek clarification and advice from National Board and Medical Council of India so as to make the course acceptable by different Universities.

In this issue of our News Letter we have published a case report of “unusual blindness following spine surgery in prone position”. To make the article more interesting I have invited Prof. Uma Maheswara Rao to write an Editorial on the subject which is worth reading. May I once again request all our members to submit their scientific research work to our News Letter so that we will review the work and inform you within a month about the suitability for publication in our News Letter. I do hope that you will all think about the Society and will contribute to the News Letter.

H.H. DASH
EDITOR-IN-CHIEF

Stronger Spine and Weaker Vision : The Unsolved Mystery Postoperative visual loss POVL

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Postoperative visual loss (POVL) is increasingly being recognized in recent years. The incidence in general is low when all types of surgery are pooled together. A 2001 report found one case of visual defect unrelated to surgical trauma among 125,234 postoperative patients, which amounts to an overall incidence of 0.0008% [1]. This data from the extremely well-maintained records of Mayo Clinic refer to all types of surgery. The occurrence of POVL is manifold high in patients undergoing spinal surgery [2,3]. Of 14,102 cases of spine surgery, at John's Hopkins University, 4 cases of perioperative ischemic optic neuropathy were identified, leading to an incidence of 0.028%. In 3 of the 4 cases, surgery was performed with the patient in the prone position; the fourth patient was placed in the lateral decubitus position [4].

In this issue of ISNACC News Letter, Kaul and colleagues describe two interesting cases of visual loss after cervical spinal surgery. Both the cases have many intriguing features, which still remain enigmatic in the literature. The patient's head was placed in a horseshoe headrest taking adequate care to avoid pressure on the eyes. Blood pressure in both the patients was maintained within 20% of the preoperative value. The duration of surgery was 8 hrs. in the first patient and, just about intraoperative fluid balance was mildly positive. First patient had complained of loss of vision in the both eyes postoperatively. But, left eye vision improved after few hours and the right eye also had very good recovery after 12 hrs. But, the second patient had loss of vision on both eye following 4 hrs. of surgery in prone position. This patient did not have any predisposing risk factors either. Though the vision of the left eye improved after treatment, the visual loss persisted in the right eye which improved up to perception of hand movement during discharge. This is very much disturbing and highly intriguing.

The American Society of Anesthesiologists (ASA) Committee on Professional Liability established the ASA POVL Registry in 1999 to collect detailed information on cases of POVL occurring after nonocular surgery. Data is collected through voluntary enrollment of POVL cases occurring within 7 days after nonocular surgery. A recent report from this registry provides an analysis of 93 cases associated with spine surgery.

Definitions of the specific lesions:

Recent studies have laid down specific criteria for the diagnosis of various pathological mechanisms responsible for visual loss. Broadly, the mechanisms identified and their definitions are as follows:

Central retinal artery occlusion (CRAO):

Central retinal artery occlusion is diagnosed based on a pale ischemic retina with a pathognomonic cherry-red spot at the macula and a relative afferent pupillary defect or reduced pupillary light reflex.

Anterior ischemic optic neuropathy (AION)

A diagnosis of *anterior ischemic optic neuropathy (AION)* requires an early fundusoscopic examination demonstrating an edematous disc with or without peripapillary flame-shaped hemorrhages and a relative afferent pupillary defect or reduced pupillary light reflex.

Posterior ischemic optic neuropathy (PION)

Criteria for *posterior ischemic optic neuropathy (PION)* include a normal early fundusoscopic examination with a relative afferent pupillary defect or an absent pupillary light reflex.

In spite of such clear definitions, on some occasions, isolated optic nerve pallor remains *unspecified*.

The results of the ASA POVL Registry published in 2006 are as follows: Of the 131 cases entered into the Registry, 93 fulfilled the criteria for inclusion into analysis. Ischemic optic neuropathy was the cause of visual loss in 83 (89%) of these 93 cases, of which 56 had PION, 19 had AION, and 8 were diagnosed with unspecified ION. Ten patients (11%) had CRAO. No particular technique of head fixation had preponderance for the occurrence of ischemic optic neuropathy. Surgery in these patients was performed in a variety of surgical frames or tables, which included a Wilson frame, Jackson spinal table, soft chest rolls, knee-chest tables with the head supported by foam pad, Mayfield pins, donut/gel pad. The mean anesthetic duration was 9.8 ± 3.1 h and in 94% of cases it was 6 h or longer. The mean duration of prone position was 7.7 ± 3.1 h. The median effective blood loss (EBL) was 2.0 L (range, 0.1–25 L), and 82% of cases had an EBL of 1.0 L or greater. Fluid management varied, with colloid (hydroxyethyl starch or albumin) used in 30% of cases and a mean intravenous crystalloid replacement of 9.7 ± 4.7 L. The lowest haematocrit was $26 \pm 5\%$, and 17% of cases had a nadir hematocrit of 30% or greater. Urine output was less than $0.5 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{h}^{-1}$ in 24% of cases, with postoperative increased creatinine in six cases and rhabdomyolysis in three. Anesthetic duration was longer, estimated blood loss was higher, crystalloid infusion was higher, and lowest hematocrit, presence of bilateral disease, use of Mayfield pins, ipsilateral periocular trauma were significantly different between patients who had ION compared to those who had CRAO.

The increased incidence of POVL in spinal surgery remains to be satisfactorily explained. Attention has been drawn to the large volume of blood loss, intraoperative hypotension and anaemia and long duration of surgery as the possible causes [6]. It has been hypothesized that a combination of these factors with local pressure on the eyeball may lead to ischemia. The association of inflammatory response with the causation of ischemic optic neuropathy has been shown recently in trauma patients (7). Extrapolation of these results to spinal surgery may partly explain the occurrence of ION in patients with high volume of blood loss causing hypotension and anaemia (7). Another school of thought is that large volume crystalloid transfusion causes orbital oedema, which some authors prefer to call “eye compartment syndrome.”

The occurrence of ION in patients whose heads were placed in Mayfield pins with the eyes free of pressure clearly demonstrates that ION occurs in the absence of pressure on the globe. Lack of retinal ischemia on ophthalmologic examination also confirms that pressure in the globe may not be an important mechanism of ION. The occurrence of ION in many cases without apparent hypotension makes the role of blood pressure management also unclear. Even small series of case control studies failed to show differences in the lowest blood pressure in patients with and without postoperative visual loss [8]. In a very recent publication, from among 126,666 surgical procedures performed, the authors identified 17 patients with perioperative ischemic optic neuropathy (an overall incidence of 0.013%). The hemodynamic variables did not differ significantly

between the ischemic optic neuropathy patients and the matched control patients leading to a conclusion that perioperative ischemic optic neuropathy can occur in the absence of atypical fluctuations in hemodynamic variables during the perioperative period (9). At present, it is not clear if blood flow to the retina and optic nerve is autoregulated and such autoregulatory mechanism is responsible for the preservation of the vision in the face of severe hypotension in most of the patients. Effect of haemodilution on optic nerve blood supply has not been studied. Clinical evidence lacks for a convincing relationship between anaemia and postoperative visual loss.

Since single factors do not explain the occurrence of ION, it may be hypothesized that a combination of factors rather than a single factor seems to be the cause of POVl in spinal surgery. At least one study has shown that a combination of long duration of surgery and large volume of blood loss was associated with visual loss after spine surgery [8], but this data did not try to differentiate the various mechanisms of visual loss such as ION, CRAO, and cortical blindness. Another study in cardiac surgical patients documented that patients with clinically significant vascular disease may be at increased risk for ischemic optic neuropathy after cardiac surgery, especially if the hemoglobin remains low in the postoperative period (10). Association of POVl with prone position in the majority of cases suggests that it may be related to high intraocular venous pressure associated with this position. When this pressure is very high, it may result in a situation akin to the compartment syndrome. Occasional case reports lend support to this hypothesis, but the final answer remains elusive! [11]. The patient in the present issue also had swollen extraocular muscles suggesting that venous congestion and oedema might have led to ischemia. It may be conjectured that preoperative risk factors like diabetes, hypertension, cardiac failure and renal failure would have increased the patient's vulnerability to the ischemic ophthalmic complication. Future studies should aim at identifying the combination of events which predispose the patients for POVl after spinal surgery particularly in prone position.

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Unilateral visual loss after Spine Surgery

Naresh Kaul, Rashid M Khan, Ashok Sumant, Kishore Shetty

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Summary:

Postoperative visual loss is one of the catastrophic and devastating complication following non-ocular surgeries. Here we report two cases of postoperative visual deterioration following spine surgery. In the first case, there was corneal injury and the patient made complete recovery. In the second patient, there was corneal injury and perhaps ischemic optic neuropathy. The patient had minimal improvement in vision.

Key words: corneal injury, prone position, spine surgery, ischemic optic neuropathy.



Postoperative visual deterioration has been described with all types of surgeries apart from head and neck surgeries. Though the exact mechanisms are not known, various risk factors have been identified which could lead to visual loss. The most important among them are hypotension, anemia and prone position [1]. Here, we report 2 cases of visual loss, which occurred after spine surgeries in the prone position.

Patient 1:

A 50-year-old lady, weighing 50 kg, presented with history of low backache of 4 years duration and radiating pain to right lower limb for 4 months. She was diagnosed to have L5-S1 grade I spondylolisthesis with lumbar canal stenosis. Spinal instrumentation was planned under general anesthesia. She had no history of any systemic illness.

Glycopyrrolate and diazepam were given as premedicants. Her preinduction pulse rate was 78bpm and blood pressure was 130/74mmHg. She was induced with fentanyl 100µg and propofol 100mg. Rocuronium was used to facilitate endotracheal intubation with 7.0mm I.D PVC cuffed endotracheal tube orally. After intubation, ointment was placed under the lower eyelids of both eyes and tape applied over the upper eyelids. Eye balls and face were covered with cotton pads. Then, the patient was turned prone onto the Wilson's frame and the head kept over the horseshoe headrest. Head and neck were maintained in neutral position. Care was taken to avoid direct pressure over the eyeball. Invasive arterial blood pressure monitoring was done by cannulating left radial artery with 20G cannula. Bupivacaine 20ml of 0.125% with 5mg morphine was administered through caudal epidural route for analgesia. Anesthesia was maintained with N₂O and O₂ in 2:1 ratio with isoflurane and intermittent doses of vecuronium. The surgery went for eight hours with a blood loss of around one liter. She received three liters of crystalloid, half a liter of colloid and 2U of whole blood. Temperature was maintained around 36 degree Celsius through out the perioperative period. At the end of surgery, patient was turned supine. Patient was reversed with neostigmine 2.5mg and atropine 1.2mg and extubated on the table. There was no evidence of trauma to the face. She was shifted to the ICU for postoperative management.

In the ICU, patient complained that she could not see anything. Ophthalmic examination revealed bilaterally reacting pupils and hazy fundus on direct ophthalmoscopy. Vision in the left eye improved within a period of one hour. In the mean time, ophthalmologist's opinion was obtained. Examination revealed bilateral corneal

edema, more on the right eye as compared to the left. Fundus was normal in size, shape, color and margin. Eyes were covered with pads and antibiotic drops were instilled in both eyes. Vision in the right eye improved completely over night.

Patient 2:

A 26-year-old man weighing 50kg, presented with history of left hemiparesis and neck pain of three years duration. MRI spine revealed atlanto axial dislocation, basilar invagination, occipitalisation of atlas and associated spinal cord compression. So, transoral odontoidectomy with posterior fixation of occipito cervical lateral masses was planned under general anesthesia. His preoperative hemoglobin was 9.3gmdL⁻¹.

He received glycopyrrolate as a premedicant. Awake Fiber optic bronchoscopy was done and 7.5mm I.D PVC cuffed endotracheal tube was placed into trachea orally. Total duration of surgery was seven hours. Initially surgery was done in supine position for 3 hours after which patient was turned prone and the surgery went for another four hours. Before turning prone, both eyes were covered with tapes after applying ointments under the lower eyelids. Eye balls and face was covered with cotton pads. Head was kept over horseshoe headrest with neck in slight extension to facilitate surgical exposure and fixation of cervical spine. Care was taken to avoid direct pressure over the eyeballs. Temperature was maintained at around 36 degree Celsius through out the surgery. Invasive arterial blood pressure monitoring was done by cannulating left radial artery with 20G cannula. The lowest systolic blood pressure recorded was 100mmHg in supine position and 98mmHg in prone position. Blood loss was around 300ml and no blood products were transfused. At the end of surgery, patient was turned supine and sent to ICU unreversed, for elective ventilation as per departmental protocol for this specific procedure.

In the ICU, right-sided periorbital and temporal edema were noted. In the right eye, there was no extra ocular movement. Cornea was hazy. Mild proptosis was present. Pupil was fixed and dilated. Perception to light was absent. Left eye was normal. Ophthalmologist's opinion was obtained. He noted pale fundus with normal disk size and shape. Ophthalmologist requested for MRI orbit, to rule out retro orbital pathology as patient had associated proptosis. The use of metallic implants for posterior fixation of spine contraindicated MRI. He had improved minimally, only to perceive hand movements in his right eye at the time of discharge.

DISCUSSION:

General anesthesia leads to loss of protective corneal reflex, Bell's phenomenon and tear production [2]. All these factors can produce dryness of eye. This results in corneal edema and desquamation ending in abrasion [3]. Positive pressure ventilation and improper position can reduce venous return and increase intra ocular pressure. Because of this venous congestion, corneal edema can ensue.

Corneal injuries can occur by direct trauma to eyes like application of facemask during induction and use of surgical cleaning solutions like iodine flowing into the eyes. In prone position, there can be direct pressure to the eye either due to tape dislodgement or improper positioning. Movement of head and neck after positioning can lead to tape dislodgement and also excessive pressure over the eyeballs.

Cucchiara and Black [3] reported an incidence of 0.17% for corneal injuries among 4652 neurosurgical patients. Use of ointments did not prevent the incidence of corneal injuries in their study. Ointment can per se produce red eye due to allergic reactions. In our patient also, corneal injury occurred despite applying ointment.

Batra [4] found an incidence of 44% for corneal abrasions in patients under general anesthesia. The peak incidence occurred at 90-150 minutes after induction. All of their patients had complete recovery at 24 hours postoperatively. All patients had positive fluorescein stains in lower third of corneal crescent indicating improper eye closure as a possible mechanism.

Gild et al., [5] conducted closed claims analysis on eye injuries associated with anesthesia. Out of 2046 patients they analysed, 71 patients had eye injuries. Corneal abrasion was seen in 35% (n=25) and was most frequent among all eye injuries. 16% of them became permanently blind. Only in 20% of them, mechanism of injury was documented. But our first patient made a complete recovery.

In 1996, Roth et al., [6] did a retrospective study to find out the incidence of eye injuries in patients undergoing non-ocular surgeries under general anesthesia. They noted an incidence of 0.056% (n=34). Among them, the most common was corneal injuries (n=21). Cause was identifiable only in 21%. As the affected patients were treated early with eye padding and antibiotics instillation, none of them had permanent eye damage.

In the first patient the cause could not be identified. But, the patient recovered in the postoperative period probably due to minor compression of the eye ball during intraoperative period. Use of eye ointment and adequate padding may have minimized the pressure.

Our second patient had periorbital edema indicating abnormal pressure over orbits. This trauma could have also led to corneal injury. But patient also had pale disk suggestive of ischemic optic neuropathy (ION). Though ION is described in patients undergoing long duration of surgery (around 8-9 hours), surgery in the 2nd patient lasted for four hours. There were no associated systemic diseases like hypertension, diabetes mellitus and there was no history of smoking. Only probable risk factor in this patient was low hemoglobin (9.3 gm/dl) level. Proptosis and absent extra ocular movements could not be explained by above-mentioned causes. Study of the retro orbital spaces would have identified mass lesions leading to proptosis. But MRI was not done due to presence of metallic implant.

Katz in 1994 [7] published a retrospective data on ischemic optic neuropathy following lumbar spine surgeries. He identified 4 patients over a three-year period. All four had systemic illnesses. Deliberate hypotension was induced in all cases. Anemia was also present in all cases. Another factor was long duration of surgery.

A review of 37 cases by Myers et al., [8], identified hypertension, diabetes mellitus, smoking and vascular diseases as possible risk factors leading to ischemic optic neuropathy following non ocular injuries. Important intraoperative factors noted were massive blood loss, hypotension and long duration of surgery.

Warner et al., in 2001 [9] published a retrospective data on the frequency of perioperative vision loss in non-cardiac surgeries. The incidence of vision loss persisting greater than 30 days was only 0.0008%. Out of the 10,886 patients who underwent spine procedures in prone position, none of them had visual changes persisting beyond 30 days.

Previously optic nerve decompression was tried to treat ischemic optic neuropathy. But the study was stopped in the middle, as it was found to be more harmful [10].

In most of the case series published in the literature, ION has been seen in patients undergoing lumbar spine surgeries in prone position for long duration under deliberate hypotension. But our patient had undergone procedure in the cervical spine for relatively short duration. Even deliberate hypotension was not used.

Manipulation of head and neck during surgery would have resulted in malposition leading to excess pressure in one eyeball. Anemia and hypotension are found in most of published case series as etiological factors in producing ischemic optic neuropathy [11]. But, in our patient the aetiopathology still remains elusive.

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A Surgical Safety Checklist to Reduce Morbidity and Mortality in a Global Population

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Background

Surgery has become an integral part of global health care, with an estimated 234 million operations performed yearly. Surgical complications are common and often preventable. We hypothesized that a program to implement a 19-item surgical safety checklist designed to improve team communication and consistency of care would reduce complications and deaths associated with surgery.

Methods

Between October 2007 and September 2008, eight hospitals in eight cities (Toronto, Canada; New Delhi, India; Amman, Jordan; Auckland, New Zealand; Manila, Philippines; Ifakara, Tanzania; London, England; and Seattle, WA) representing a variety of economic circumstances and diverse populations of patients participated in the World Health Organization's Safe Surgery Saves Lives program. We prospectively collected data on clinical processes and outcomes from 3733 consecutively enrolled patients 16 years of age or older who were undergoing noncardiac surgery. We subsequently collected data on 3955 consecutively enrolled patients after the introduction of the Surgical Safety Checklist. The primary end point was the rate of complications, including death, during hospitalization within the first 30 days after the operation.

Results

The rate of death was 1.5% before the checklist was introduced and declined to 0.8% afterward ($P = 0.003$). Inpatient complications occurred in 11.0% of patients at baseline and in 7.0% after introduction of the checklist ($P < 0.001$).

Conclusions

Implementation of the checklist was associated with concomitant reductions in the rates of death and complications among patients at least 16 years of age who were undergoing noncardiac surgery in a diverse group of hospital.

Continuous Perioperative Insulin Infusion Decreases Major Cardiovascular Events in Patients Undergoing Vascular Surgery

A Prospective, Randomized Trial

Perioperative Medicine, Anesthesiology 2009; 110:970–7

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Background: A growing body of evidence suggests that hyperglycemia is an independent predictor of increased cardiovascular risk. Aggressive glycemic control in the intensive care decreases mortality. The benefit of glycemic control in noncardiac surgery is unknown.

Methods: In a single-center, prospective, unblinded, active-control study, 236 patients were randomly assigned to continuous insulin infusion (target glucose 100–150 mg/dl) or to a standard intermittent insulin bolus (treat glucose > 150 mg/dl) in patients undergoing peripheral vascular bypass, abdominal aortic aneurysm repair, or below- or above-knee amputation. The treatments began at the start of surgery and continued for 48 h. The primary endpoint was a composite of all-cause death, myocardial infarction, and acute congestive heart failure. The secondary endpoints were blood glucose concentrations, rates of hypoglycemia (< 60 mg/dl) and hyperglycemia (> 150 mg/dl), graft failure or reintervention, wound infection, acute renal insufficiency, and duration of stay.

Results: The groups were well balanced for baseline characteristics, except for older age in the intervention group. There was a significant reduction in primary endpoint (3.5%) in the intervention group compared with the control group (12.3%) (relative risk, 0.29; 95% confidence interval, 0.10–0.83; $P = 0.013$). The secondary endpoints were similar. Hypoglycemia occurred in 8.8% of the intervention group compared with 4.1% of the control group ($P = 0.14$). Multivariate analysis demonstrated that continuous insulin infusion was a negative independent predictor (odds ratio, 0.28; 95% confidence interval, 0.09–0.87; $P = 0.027$), whereas previous coronary artery disease was a positive predictor of adverse events.

Conclusion: Continuous insulin infusion reduces perioperative myocardial infarction after vascular surgery.

Intensive versus Conventional Glucose Control in Critically Ill Patients

The NICE-SUGAR Study Investigators, *The New England Journal of Medicine* 2009; 360: 13.

Background

The optimal target range for blood glucose in critically ill patients remains unclear.

Methods

Within 24 hours after admission to an intensive care unit (ICU), adults who were expected to require treatment in the ICU on 3 or more consecutive days were randomly assigned to undergo either intensive glucose control, with a target blood glucose range of 81 to 108 mg per deciliter (4.5 to 6.0 mmol per liter), or conventional glucose control, with a target of 180 mg or less per deciliter (10.0 mmol or less per liter). We defined the primary end point as death from any cause within 90 days after randomization.

Results

Of the 6104 patients who underwent randomization, 3054 were assigned to undergo intensive control and 3050 to undergo conventional control; data with regard to the primary outcome at day 90 were available for 3010 and 3012 patients, respectively.

The two groups had similar characteristics at baseline. A total of 829 patients (27.5%) in the intensive-control group and 751 (24.9%) in the conventional-control group died (odds ratio for intensive control, 1.14; 95% confidence interval, 1.02 to 1.28; $P = 0.02$). The treatment effect did not differ significantly between operative (surgical) patients and nonoperative (medical) patients (odds ratio for death in the intensive-control group, 1.31 and 1.07, respectively; $P = 0.10$). Severe hypoglycemia (blood glucose level, <40 mg per deciliter [2.2 mmol per liter]) was reported in 206 of 3016 patients (6.8%) in the intensive-control group and 15 of 3014 (0.5%) in the conventional-control group ($P < 0.001$). There was no significant difference between the two treatment groups in the median number of days in the ICU ($P = 0.84$) or hospital ($P = 0.86$) or the median number of days of mechanical ventilation ($P = 0.56$) or renal-replacement therapy ($P = 0.39$).

Conclusions

In this large, international, randomized trial, we found that intensive glucose control increased mortality among adults in the ICU: a blood glucose target of 180 mg or less per deciliter resulted in lower mortality than did a target of 81 to 108 mg per deciliter.

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- ❖ Ethics committee's approval is mandatory.
- ❖ Information pertaining to any other financial assistance for the project from other sources must be provided.
- ❖ Four copies of the research project, in the proper format should be submitted to the Secretariat on or before 31st December 2009.

NEWS ITEM

Dr Bidkar Prasanna Udupi passed D.M. Neuroanaesthesiology in May 2009 from Dept. of Neuroanaesthesiology, AIIMS, New Delhi.

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