Correspondence

Inhumanity in the autopsy room

It was early 2000. My uncle, a retired army officer, had succumbed to a subdural haematoma at a corporate hospital in Mumbai. He was the leader of a group of people opposing the construction of an artificial port in their small coastal town in Gujarat as it would have submerged several villages. How did he get the subdural haematoma? Was he beaten up in custody? Why did he land up in jail?

Without touching on that issue, I want to highlight a very dark facet of human behaviour—some ask for a bribe even over a dead body.

My uncle's body was taken to a major municipal hospital in Mumbai for post-mortem examination. The examination was performed at night, probably because it was a high-profile political case. After the forensic expert had finished his job, the attendant on duty in the forensic department showed my brother, who is not a doctor, the dissected body of our uncle, with the brain exposed in the opened skull. He said that the head had to be closed. He hinted at a bribe. My brother had never seen such a sight before. He was shocked and nauseated. As soon as he gained his composure, he sent for me and my cousin—both of us are doctors. We filed an official complaint. We are not aware of any action taken on the basis of our representation.

Even after 10 years, that incident still haunts me. Somebody asking for a bribe to suture up a dissected body is simply disgusting. For the autopsy room attendant, it must have been just another dead body and just another autopsy. He was neither concerned, nor worried about the effects of his actions on the minds and hearts of the relatives of the dead person. There were no traces of humanity, service or sensitivity.

What circumstances make us so desperate as to ask for a bribe to suture a dead body?

This reminds me that we, as doctors, should be careful about what we say to the relatives of a person who has died in the intensive care unit. We declare death and carry on our formalities as any 'babu' would in an office. The next moment, we may be sharing a joke in an adjacent cabin. Can we imagine what must be going through the relatives' minds when they see us totally oblivious to their pain and sorrow?

What can be done to prevent such incidents from occurring? What would you have done if you were in my or my brother's place? I do not have an appropriate answer. Do you?

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State governments' decision to stop participating in All-India Medical Entrance Examination system: A retrogressive step

The Maharashtra government is considering scrapping the all-India entrance quota system in state medical colleges, and this would reportedly open up 960 seats for local aspirants.[1] Maharashtra is the second state after Tamil Nadu that plans to abolish the central quota. Tamil Nadu has already done away with the quota through legislation.[2] This change might become operational in Maharashtra from the 2011–12 academic session. The 2 state governments have decided on this move as their analysis shows that students from these states manage to bag only a few seats in the central quota (for example, the Maharashtra government claims only 120 students from the state get admissions in other states through the quota system[1]), and in return, the states surrender many more seats for admission through the quota system. While on face value, this decision seems to be designed to benefit the students of the states, it is a retrogressive step and will be detrimental to medical education in India.

The all-India quota system in medical colleges has been operational for the past many years and was set up through the instructions of the Supreme Court of India. State governments subscribing to the scheme allow for 15% of undergraduate seats and 50% of postgraduate seats in state government medical colleges to be filled through a central entrance examination conducted by the Central Board of Secondary Education at the undergraduate level (*http://aipmt.nic.in/*) and through the All India Institute of Medical Sciences at the postgraduate level (*http://www.aiims.edu/aiims/events/exams-results.htm*). The states of Jammu and Kashmir and Andhra Pradesh opted out of the scheme since its inception and domiciles of these states are routinely not allowed to take part in these entrance examinations (unless they claim exceptions with support of appropriate documentation).

This all-India quota system has several advantages. It provides students the opportunity to undergo their medical training in other states. It also enhances the number of seats available for students to compete for, and this is especially useful for those hailing from smaller states which have only a couple of medical colleges. The central quota system promotes an exchange of students between states, and thus also helps create bonds across languages and regions, a requirement at a time of increasing civil strife and factionalism. The central entrance examination system, at both the undergraduate and postgraduate levels, has evolved over many years to be conducted in a fair and competitive manner (with the option of approaching the Supreme Court in case of disputes). These examinations also set a standard of quality for other state-level and individual medical college entrance tests. The seats allotted through the all-India entrance quota in a state are also accessible to the students from that state, provided these are available at their rank in the merit list during the seat allotment counselling. A few of the students hailing from other states who get admitted through the central quota stay back in the state of training and provide healthcare services locally.

If state governments feel that too few local students are getting selected in the all-India medical entrance examinations, they can try to improve the students' preparation by making resources and training material available. Withdrawing from the scheme is an unfortunate and negative decision, and is not in the interest of the states or the nation.

CONFLICT OF INTEREST

I studied in a government medical college in another state for my MB,BS degree through the all-India entrance examination system.

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An uncommon cause for bilateral peripheral facial nerve paralysis

Bilateral facial nerve paralysis is uncommon. Its causes include Guillain–Barré syndrome, brainstem encephalitis, syphilis, leukaemia, sarcoidosis, Lyme disease and bacterial meningitis.[1,2] A 40-year-old man was admitted to our hospital. Fourteen days earlier he noticed that he was unable to close his right eye. He also had weakness over the right side of his face. Five days later, he could not close his left eye. There was drooling of saliva from both angles of his mouth and there was excessive lacrimation. There was no history of fever, vomiting, photophobia, seizures or loss of consciousness. General physical examination was normal. Examination of the central nervous system revealed bilateral lower motor neuron facial paralysis with normal extra-ocular muscle movements. Fundus examination was normal. He was started on acyclovir 800 mg orally 5 times a day and dexamethasone 4 mg i.v. every 6 hours. Electrical stimulation of the facial muscles was done as part of physiotherapy.

His haemoglobin, fasting blood sugar and liver and kidney functions tests were normal. Serum angiotensin converting enzyme (ACE) levels were 74 U/L (normal: 8–65 U/L). The 24-hour urinary calcium was 179 mg (normal: 100–300 mg). Examination of the cerebrospinal fluid (CSF) revealed 50 lymphocytes, no red blood cells, with protein of 156 mg/dl and glucose of 49 mg/dl. Gram stain, acid-fast bacilli stain, India ink preparation and VDRL of the CSF were negative and the CSF culture was sterile. Examination of the bone marrow was normal. Electrophysiological studies of both facial nerves revealed reduced amplitudes with normal distal latencies and conduction velocities, suggestive of axonal involvement. MRI of the brain with gadolinium contrast was normal.

Hepatitis B, hepatitis C and VDRL serology were negative. However, HIV-1 was positive by ELISA and was confirmed by Western blot. The CD4 count was 408 cells/µl. The HIV viral RNA load by real-time reverse transcriptase PCR was 124 824 copies/ml. The patient was heterosexual. He was unaware of his HIV status and denied any history of drug abuse, blood transfusion or high risk sexual behaviour. After 4 weeks of treatment and physiotherapy, the patient had significant improvement in facial weakness. He was able to fully close both eyes and drooling of the saliva had stopped.

Peripheral facial paralysis can occur at any stage of HIV infection.[3] It is seen more often in healthy HIV carriers than in patients with AIDS.[4] Unilateral and bilateral facial nerve paralyses occur with a 100-fold greater frequency in the HIV-1 infected population: 4.1% *v.* ~0.04% in the general population. However, even among patients with acute HIV-1 infection, bilateral facial paralysis is rare.[5] During the early stages of HIV infection, especially in patients with bilateral facial paralysis, corticosteroids may be used because the risks of management are outweighed by the complications of facial neuropathy.

Bilateral facial paralysis is more often due to systemic causes than unilateral paralysis and should spur a diligent search for an underlying cause. Unilateral or bilateral facial paralysis can be the first symptom of HIV infection. HIV should be considered in the investigation of any facial paralysis, especially in areas with a high prevalence of HIV. The peripheral facial paralysis in HIV-positive patients is most often a self-limiting condition.[2]

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Evaluation of awareness and use of emergency access numbers in Delhi

Emergency services in the city of Delhi are, at present, accessible through 3 different telephone numbers: 100 for police, 101 for fire services and 102/1099 for centralized accident and trauma services (CATS) ambulance. In many countries, such services are accessible through a single number called the universal emergency number (UEN). For example, in the USA, this number is 911[1] and in most of Europe, it is 112.[2] A single 3-digit number is easy to remember and can be dialled quickly, and its use ensures that response services dispatch the appropriate team to the site of an emergency. The use of different numbers for different services can lead to a delay in appropriate assistance if the incorrect number is called by a bystander.

To assess the knowledge of the people of Delhi with respect to emergency access numbers and the concept of a UEN, we conducted a questionnaire-based survey. Self-administered, multiple-choice, bilingual (English and Hindi) questionnaires were distributed to 1000 respondents in 5 district commercial centres in Delhi during the evening hours in March 2008.

We analysed the 761 completed questionnaires. The majority of the respondents (78%) were educated above high school level, only 42% had some form of medical insurance and 30% had a family physician.

Only 54 (7%) would have called for an ambulance to transport a patient from the street to a hospital. The rest would have transported the patient themselves (516 [68%]) or called the police (182 [24%]).

The phone number for calling the police was known to 747 (98%), for the fire services to 499 (66%) and for an ambulance service to 535 (70%). Only 7 (1%) knew the CATS number. We found that one-third of the respondents did not know all 3 emergency access numbers correctly. Most of them were reluctant to use ambulance services to transport a patient.

In response to the question about a UEN, 696 (91%) did not want such a facility to be introduced.

Taken together, these facts could lead to an inappropriate response, with the incorrect service being called or ambulance services being underutilized. This, in turn, could make a crucial difference to outcomes for critically ill or injured patients who need to be rapidly transported to a hospital.

The low acceptance of the suggestion for creating a UEN is possibly due to lack of awareness of this concept and its benefits.

Three states in India, Gujarat, Andhra Pradesh and Uttarakhand, are already using 108 as a UEN.[3] It is important to extend this facility to cover the entire country, as it could well translate into saving the lives of a number of people.

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Hand, foot and mouth disease (HFMD): A newly emerging infection in Orissa, India

Hand, foot and mouth disease (HFMD) caused by human enteroviruses such as coxsackievirus A-16 (CA 16) and enterovirus 71 (EV 71), presents as epidemics in different parts of the world.

There is no past record of HFMD from Orissa. During September–October 2009 several patients presenting with fever, papulovesicular rash on the buttocks, knees, hands, feet and lesions on the oral mucosa were seen at different hospitals and dermatology clinics of Bhubaneswar, Orissa. A majority of those affected were below 12 years of age. A team from the Regional Medical Research Centre, Bhubaneswar visited households with affected cases. Patients were examined and blood samples collected. Serum was stored at –70 °C and transported in dry ice to the National Institute of Virology (NIV), Pune for molecular investigations. The enterovirus Coxsackie A-16 was isolated from 4 of 7 samples. The disease was self-limiting with spontaneous recovery.

Reports from the Asia Pacific region show occurrence of HFMD epidemics in 1997 (Sarawak), 1998 (Taiwan), 1999 (Perth) and 2000 (Singapore, Korea, Malaysia and Taiwan).[1,2] There are a few reports of HFMD from India. The first outbreak was reported in 2003 from Kerala[3] and subsequently from Nagpur and other western parts of the country (2005–06) and West Bengal (August–October 2007).[1,4] This report of HFMD, the first from Orissa, is important because of the large rural and especially tribal population (tribals constitute 24% of the total population of the state), with lack of general hygiene and water sanitation practices, which facilitate spread of the disease. Our report is meant to draw attention to HFMD and its emergence in an area previously free from this disease.

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India: The ESBL capital of the world?

The invention of penicillin was a landmark event but bacteria soon developed penicillinases to destroy penicillin. In the 1960s, extended-spectrum penicillins, resistant to penicillinases, were developed and were used extensively in the 1970s. The bacteria then developed beta-lactamases, enzymes which destroy beta-lactam (BL) antibiotics. Third-generation cephalosporins, stable to beta-lactamases, were developed in the 1980s. Bacteria especially the Enterobacteriaceae, then developed extended-spectrum beta-lactamases (ESBL), which inactivate penicillins, cephalosporins and aztreonam. In the presence of ESBLs, aminoglycosides, fluoroquinolones and trimethoprim–sulphamethoxazole also do not work. Beta-lactamase inhibitors (BLI), e.g. clavulanic acid, sulbactam and tazobactam block the beta-lactamase enzymes and allow BL antibiotics to work. The BL and BLI combi-nations work against ESBL-producing organisms in uncomplicated urinary tract infections, low-risk complicated intra-abdominal infections (cIAI), moderate respiratory tract infections, and skin and soft tissue infections. In a randomized study involving 306 patients across 17 centres in India, empirical use of cefoperazone and sulbactam was found to be useful in

the management of intra-abdominal infections.[1] Carbapenems are not affected by ESBLs and are the drugs of choice. Tigecycline is a new choice for empirical monotherapy of infections caused by ESBL-producing organisms; it is the drug of choice for carbapenem-resistant *Acinetobacter* but has no anti-pseudomonal activity.

The rates of resistance of bacteria to antibiotics, however, vary from region to region. While methicillin-resistant *Staphylococcus aureus* (MRSA) is a problem in the USA, ESBLs are a major issue in Latin America[2] and Asia. As many as 36% of *E. coli* and 15% of *Klebsiella* isolates from China were ESBL-positive.[3] Of the 3377 isolates from 20 centres across India, 80% were positive for beta-lactamase; production of beta-lactamase was as high as 91% in *Enterococcus* and 89% in *Klebsiella*.[4] As many as 60% of *E. coli* isolates in a 10-centre study were producing ESBLs.[5] In the intensive care unit (ICU) at a tertiary hospital in New Delhi, 95% of isolates were ESBL-producing.[6] In a recent study of 2870 blood samples from suspected cases of septicaemia in a tertiary-level hospital in New Delhi, 41 of 64 isolates (71%) of *Klebsiella* and 10 of 26 isolates (42%) of *E. coli* were ESBL producers.[7] The incidence of ESBLs has also been reported in community-acquired infections; in a report of 920 urine samples from Aligarh, 42% of the isolates were ESBL-positive.[8]

Clinicians should anticipate the presence of ESBL-producing bacteria in elderly patients, those in ICUs, on parenteral nutrition, with indwelling devices and undergoing interventions. In such patients, it is now recommended to start empirical but appropriate broad-spectrum antibiotics and then to de-escalate on the basis of culture and sensitivity reports.

Collateral damage in the form of increasing resistance of bacteria to antibiotics is a global health problem—more so in India which seems to have become the 'ESBL capital of the world' due to extensive but injudicious use of antibiotics. Clinicians, microbiologists and the industry need to play an important and responsible role to curb this menace of ESBLs. The industry should promote the use of antibiotics based on scientific evidence and not commercial profit. Hospitals should develop and follow protocols based on local microbiological data for judicious antibiotic use. A national policy for antibiotic resistance surveillance and control has been suggested.[9] There is a need to have an Indian Nosocomial Infection Surveillance (INIS) system on the patterns of the National Nosocomial Infections Surveillance (NNIS) in the USA.[10] Prevention of spread of nosocomial infections by implementing strict hand hygiene in hospitals is of utmost importance.

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Do journals acknowledge their past editors: An online survey of leading medicine journals

Medical journals have probably had the greatest impact on healthcare and yet their developmental history has been largely ignored.[1,2] Journals have reached the present stage as a result of dedicated efforts by their past editors. However, their contributions are forgotten to the extent that their names are not even mentioned in the history of the journal or in the web pages related to the editorial members. We assessed the extent to which leading online journals in the specialty of medicine provide the details of their past editors.

Among the journals started in the year 2002 or earlier, 7 internal medicine journals (*New England Journal of Medicine, The Lancet, Journal of American Medical Association, Annals of Internal Medicine, British Medical Journal, Archives of Internal Medicine, Canadian Medical Association Journal*) and 24 medicine-related subspecialty journals (*Circulation, Hypertension, Stroke, Neurology, Annals of Neurology, Lancet Neurology, Gastroenterology, Gut, American Journal of Gastroenterology, Hepatology, Journal of Hepatology, Lancet Oncology, Blood, Endocrine Reviews, Clinical Infectious Disease, Journal of Infectious Disease, Lancet Infectious Disease, American Journal of Respiratory and Critical Care Medicine, Thorax, Critical Care Medicine, Journal of American Society of Nephrology, Annals of Rheumatic Disease, Diabetes* and *Diabetes Care*) were selected by their hierarchy in the 2007 Journal Impact Factor.[3] Their websites were screened for a description of the editorial history with regard to a mention about the founder or members of the team which originally developed the journal and details of the subsequent editors. The websites were accessed between 25 December 2009 and 5 January 2010. Each author made an independent survey and the surveys were compared for consistency. A search was made in the main sections of the journals' web pages and their relevant links for the details.

Three of 31 (10%) study journals started publication before 1900 and 19 of 31 (61%) before 1975. A dedicated web page on 'About the journal' was found on all websites. Nine of 31 (29%) mentioned their first and subsequent editors, 2 (6%) mentioned only their first editor, 4 (13%) mentioned only their subsequent editors and 16 (52%) made no mention of their past editors. Among journals published after 1975 (n=12), 2 (17%) described their subsequent editors, while 3 (25%) mentioned their first and subsequent editors.

We observed a lack of interest among journals in documenting their editorial history on their web pages. Obviously each journal would have required a dedicated effort either from an individual or a team during their early days, followed by efforts by a series of editors to reach their current status. Members of most editorial boards would have given their effort out of pure passion for the job, often without any financial returns. This information may not be relevant to the current scientific community but is, in our opinion, an expected and essential gesture from the current editors. We observed that all the journals had a dedicated page which described the journal's policies, acceptable manuscripts, current impact factor, current editorial members, etc .We feel these pages should ideally also have information on the history of the journal, followed by a summary of the contributions of past editors. Unlike the print version of the journal, space is not a limitation in the online version, so past editors can easily be acknowledged.

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Glucose-6-phosphate dehydrogenase deficiency and haptoglobin polymorphism among Rajput and Brahmin children in Himachal Pradesh

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is a common inherited haemolytic disorder, affecting about 400 million people worldwide. G6PD is a housekeeping enzyme which catalyses the first step in the pentose phosphate pathway (PPP). This pathway produces NADPH molecules, which maintain reduced glutathione in the cell. Reduced glutathione is an antioxidant and protects cells against oxidative damage. Its chromosomal location is Xq28.[1] Haptoglobin is an α-2 glycoprotein which binds to free haemoglobin released from destroyed red blood cells. It helps in recycling the iron part of haemoglobin. Haptoglobin is inherited by 2 co-dominant autosomal alleles situated on chromosome 16 in humans—HP1 and HP2. Thus, there are 3 phenotypes HP1-1, HP2-1 and HP2-2. HP1-1 individuals have greater haemoglobin binding capacity than those with HP2-1 and HP2-2.[2]

We studied Rajput and Brahmin children between 12 and 18 years of age at Palampur in Himachal Pradesh to screen 2 genetic markers—haptoglobins and G6PD, to compare the results with those for other populations in India, and also to see if any association exists between the two markers.

Blood samples of 182 Rajput and 94 Brahmin children were collected and only male children were screened for G6PD deficiency. We found the frequency of the G6PD-deficient allele to be higher among Brahmins than Rajputs. The most frequent haptoglobin genotype in both groups was HP2-2 type, followed by HP2-1 and HP1-1. While the Rajput population followed the Hardy–Weinberg equilibrium, the Brahmin population deviated from it. The HP1 allele frequency was found to be higher among Brahmins than Rajputs (Table I).

Deviation of Brahmins from the Hardy–Weinberg equilibrium, with significantly higher homozygotes than expected with respect to HP polymorphism, can possibly be attributed to inbreeding among them. This is reflected in their surname endogamy. There is hardly much diversity of surnames among the Brahmins of Palampur, with 90% of the sample having the surname of Sharma. The Rajputs, on the other hand, had more subdivisions.

With respect to the selected genetic markers, the Brahmins differ significantly not only from the Rajputs of Palampur, but also from most of the studied populations, suggesting their distinctness and their different ancestry.[3]

Besides this, Brahmin children have higher G6PD-deficient allele frequency and all G6PD-deficient individuals have the HP2-2 genotype. Since G6PD is related to haemolytic anaemia and HP2 has higher haemoglobin-retaining capacity, HP2 may provide selective advantage to G6PD-deficient individuals. However, this association is still not statistically proven and our data are not sufficient to confirm the selection operating on this polymorphism.

However, the high G6PD deficiency (16%) among male children is a matter of concern. This population needs to be screened before administering drugs that cause drug-induced haemolytic anaemia so as to minimize the morbidity and mortality.

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TABLE I. Distribution of haptoglobin and G6PD polymorphism

Population	*n*	Haptoglobin polymorphism					G6PD polymorphism		
		Genotypes			Allele frequencies		*n*	Normal	Deficient
		HP1-1	HP2-1	HP2-2	HP1	HP2			
Rajput	182	3 (1.6)	28 (15.3)	151 (82.9)	0.093	0.907	79	75 (94.9)	4 (5.06)
Brahmin	94	7 (7.4)	11 (11.7)	76 (80.9)	0.130	0.870	48	40 (83.3)	8 (16.67)

Values in parentheses are percentages

Nurse tutors to teach basic clinical skills: An experience from Selcuk University, Turkey

There are difficulties in providing clinical training for undergraduate medical students especially when there are more students and the clinical staff has limited time available.[1] Nurses and doctors have some overlapping skills, which allows for one to substitute for another in some areas.[2,3] The use of nurses as educational resource personnel in the undergraduate medical curriculum is not new.[4,5] Nurse tutors have played a valuable part in learning clinical skills and this role has a beneficial impact on inter-professional educational collaboration.[6]

Using a retrospective study design, we compared the feedback of medical students tutored by nurses in 2008 with those of students tutored by physicians in 2007 for the same 13 basic clinical skills. We had 1238 feedback forms for physicians and 1846 for nurse tutors. We randomly selected 30 forms for each tutor for every skill to get a confidence interval of 95% with 5% margin of error. A total of 780 forms (390 for physicians and 390 for nurse tutors) were compared. The standardized 9-item feedback form was designed to assess students' perceptions and 6 items were about tutoring abilities. Students scored these items as agree, not sure and disagree.

We found that both nurse and physician tutors were highly valued by students with respect to training of basic clinical skills. While nurse tutors were assessed to be significantly better than physician tutors on 4 of the items ($p<0.05$), the items 'I felt adequate after this training' and 'I am satisfied about learning this skill' showed no statistical difference between nurse and physician tutors ($p>0.05$; Table I). While physician tutors got a mean (SD) 10.7 (2.36) points in the total feedback score, nurse tutors got 11.2 (1.76) points ($p<0.001$).

Kilminster *et al*. reported that nurses helped medical students to develop their skills in history-taking, physical examination and technical skills.[1] Henley *et al*. acknowledged the general teaching skills of family nurse practitioners.[4] Howe *et al*. highlighted the benefits of nurse educators for medical students during a course in primary care.[5] Xanthos *et al*. reported that nurses proved to be more efficient than doctors in teaching cardiopulmonary resuscitation by providing a sufficient theoretical background to the trainees and infusing them with competent practice skills.[7]

In conclusion, medical education units have some shortcomings which need to be addressed.[1,8] Our study reflects students' opinion that 'nursing tutors' are important contributors to undergraduate medical education.[1] They met the needs of students adequately.

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TABLE I. Comparison of students' feedback for physician (n=390) and nurse (n=390) tutors' abilities in teaching 13 basic clinical skills

Feedback item	Physician tutors			Nurse tutors			p value	Mean score		p value
	Disagree	Not sure	Agree	Disagree	Not sure	Agree		Physician	Nurse	
The demonstration was good enough to learn this skill	13 (3.3)	96 (24.6)	281 (72.1)	11 (2.8)	66 (16.9)	313 (80.3)	0.02	1.68 (0.53)	1.77 (0.48)	0.01
I could perform the skill alone and I learned better	3 (0.8)	59 (15.1)	328 (84.1)	0 (0)	28 (7.2)	362 (92.8)	<0.001	1.83 (0.39)	1.92 (0.25)	<0.001
I felt more adequate after this training	8 (2.1)	90 (23.1)	292 (74.9)	15 (3.8)	90 (23.1)	285 (73.1)	0.48	1.72 (0.48)	1.69 (0.53)	0.33
Tutor helped me to take the steps properly	9 (2.3)	43 (11.0)	338 (86.7)	6 (1.5)	17 (4.4)	367 (94.1)	<0.001	1.84 (0.42)	1.92 (0.31)	<0.001
Tutors' approach motivated me to learn	16 (4.1)	78 (20.0)	296 (75.9)	6 (1.5)	16 (4.1)	388 (94.4)	<0.001	1.71 (0.53)	1.92 (0.31)	<0.001
I am satisfied about learning this skill	1 (0.3)	41 (10.5)	348 (89.2)	3 (0.8)	22 (5.6)	365 (93.6)	0.03	1.88 (0.32)	1.92 (0.28)	0.07

Values are n (%) unless specified