smaller centers. This leads to undue concentration of neurosurgeons in a few centers, where facilities are available, leading to less and less of neurosurgical work for individual neurosurgeons and frustration among the younger neurosurgeons. This has to be tackled in two ways: i) The National body like the Neurological Society of India (NSI) must take charge in identifying the likely vacancies in a year in the Neurosurgery Posts in various Hospitals, both Teaching and non-teaching and suggest to various teaching Hospitals on the intake of candidates in a particular year based on the likely vacancies. The number may be based on likely vacancies in existing institutions and new opportunities likely to arise in new institutions with Neurosurgical facilities. This may sound utopian, but can be implemented with some effort. ii) The Teaching Hospitals must provide for a mandatory period of after-qualification training for all Neurosurgical Postgraduates trained by them. This may mitigate the unemployment and under-employment to some extent and also allow young Neurosurgeons gain more experience and confidence.

As for the second point, the NSI and the Medical Council of India must coordinate and see that the minimum standards are kept up in the Teaching Hospitals approved for Neurosurgical Training. They must do periodic surprise inspections in such Institutions and withdraw recognition of the Institutions if there is a shortcoming. The training must include opportunities to familiarize with operating microscope, endoscope, stereotaxy and training in one or more subspecialities. The minimum standards must be based on the standards available at the Central Institutes like AIIMS, PGI, etc. Private institutions offering Neurosurgical training (for DNB program), must be supervised by NSI and NBE and minimum standards must be same as Teaching Hospitals as mentioned. For this NSI must have a subcommittee to coordinate with MCI and NBE.

Regarding the third point, it is a real truth that the majority of Neurosurgical trainees in non-teaching Private Hospitals approved by the NBE do not get adequate practical experience because of the natural limitations of Private Medical Care environment. The trainees in such Institutions must be rotated through Teaching Hospitals preferably in the same city for a good period of their training. It must also be insisted by NBE that the candidates perform minimum number of independent operations under each category of Neurosurgery during their training.

Finally NSI, in concurrence with the various Universities Neurosurgery Examinations throughout the country.

I think we must act before it is too late. Otherwise the future may not forgive us.

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Non-traumatic atlantoaxial rotary subluxation

Sir,

A 17-year-old girl presented with complaints of sudden onset of torticollis associated with severe neck pain and restriction of neck movements. There was no history of trauma. On examination, she had “cock robin” deformity of the head, which was rotated to the left and the chin was directed to the right side. There was no neurological deficit.

X-ray of the craniovertebral junction (CVJ) (open mouth view) revealed overlapping of the lateral masses causing obscuration of the lateral atlantoaxial joints. Magnetic resonance imaging (MRI) scan of the cervical spine revealed rotatory subluxation of the atlas with the lateral mass lying adjacent to the cord (Figure 1). 3D-computed tomography (CT) scan revealed Fielding’s Type I atlantoaxial rotatory subluxation (Figure 2). She was treated with Gardner-Well’s skull traction and reduction

Figure 1: MRI scan of the craniovertebral junction both corona (A) and axial (B) sections shows rotatory atlantoaxial subluxation with lateral mass lying adjacent to the spinal cord.

Figure 2: 3D-computed tomography (CT) scan showing right rotatory atlanto-axial subluxation without any increase in the atlanto-dental distance confirming the diagnosis of Fielding’s type-I atlantoaxial rotatory subluxation.
was achieved. Dynamic CT scan revealed adequate reduction of the atlantoaxial rotatory subluxation. However, at the end of two weeks, she returned with torticollis despite wearing the cervical collar. She was again treated with Gardner-Well’s traction for two weeks followed by cervical collar for three months. At 42 months follow-up, she was asymptomatic.

Rotatory atlantoaxial subluxation is an uncommon and poorly understood clinical entity. Various conditions which can predispose to rotatory subluxation include inflammation, and surgical procedures in and around the throat, neck and cervical spine. The physiological laxity of the ligaments around the CV junction may be the underlying predisposing factor for rotatory subluxation. The presence of a torticollis in a child should arouse suspicion of rotatory atlantoaxial subluxation, particularly when there is a recent history of recent throat infection or trauma.

Fielding and Hawkins classified atlantoaxial rotatory fixation into 4 types depending on the degree of atlantodental distance. In Type I, the rotatory subluxation is associated with normal atlantodental interval. In Type II and Type III, the rotatory subluxations are associated with 3 to 5 mm and more than 5 mm of anterior displacement of the atlas respectively. Type IV rotatory subluxation is associated with posterior shift of the atlas due to failure of the dens. The Type I variety is the commonest and the most benign form of rotatory subluxation, probably because the transverse ligament is intact. The diagnosis of atlantoaxial rotatory subluxation requires a high degree of suspicion based on the signs and symptoms. A CT scan of the atlantoaxial complex is most valuable for demonstrating the anatomy and a 3D-CT scan provides more direct visualization of the abnormal anatomy. When diagnosed early, most patients respond well to a conservative treatment with cervical collar and bed-rest. Surgical intervention is advised when conservative treatment fails to achieve reduction or is followed by a recurrence of the deformity.

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Nadroparin in acute ischemic stroke

Sir,

I read with interest the recent article by Sarma GRK et al. Based on a randomized controlled trial, they conclude that the outcome of stroke is significantly better in the group receiving nadroparin and aspirin versus the group receiving aspirin alone. However, I would like to make certain comments.

Firstly, data regarding the incidence of recurrent or progressive infarction during the follow-up period has not been provided. It is well known that both aspirin and heparin are mainly useful in preventing either re-infarction or progressive infarction. Moreover, in previous studies, a similar reduction in the rate of re-infarction or progressive infarction was seen with low molecular weight heparin (LMWH) and aspirin. Therefore, anticoagulation with LMWH, in order to prevent new infarction or worsening of neurological status, is not recommended in the early management of stroke.

The second issue is regarding the risk of hemorrhagic complications. Sarma GRK et al noted upper gastrointestinal bleeding in one patient (5%) in the LMWH group (requiring withdrawal of nadroparin) as compared to none in the aspirin group. Other investigators too have observed a significantly higher risk of bleeding with LMWH as compared to aspirin.

Thirdly, Sarma GRK et al draw an analogy between nadroparin and recombinant tissue plasminogen activator (rtPA) and advocate an early initiation of the former. However, it should be noted that rtPA is the only proven therapy for restoring or improving cerebral perfusion till date and nadroparin does not have this ability. Therefore, it may be inappropriate to draw any comparisons between the two, as their roles in treatment of acute stroke are entirely different.

Finally, there are a few limitations as mentioned by Sarma GRK et al, such as small sample size (only 20 in each arm) and lack of follow-up beyond three months, which further limit the usefulness of this trial. Moreover, studies with a larger sample size (> 1000 patients) and longer follow-up (six months) have failed to demonstrate any significant benefit with LMWH over aspirin.

In conclusion, data provided in this study is inconclusive and does not instill enough confidence to make a change in the current practice of avoiding combination of LMWH and aspirin in the treatment of acute ischemic stroke.

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