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Message

I am very glad to know that Mymensingh Medical College-Doctors Association of North America (MMC-DANA), Nassau University Medical Center (NUMC), and Bangladesh Medical Association of North America (BMANA) jointly sponsoring a CME program on “Recent Developments in Medicine” on July 2, 2006 in New York city in the grand reunion ceremony of ex-students of MMC living in North America. As principal and ex-student (M-15) of Mymensingh Medical College, I feel proud of this occasion. It is really a great endeavor of the organization. The reunion ceremony will bring the opportunity to meet each other of our medical college ex-students. The CME programs will refresh and enrich the knowledge of the participating doctors. They will feel new incentive in their working places.

I invite all MMC-DANA members to visit MMC.

I wish MMC-DANA reunion and CME program a grand success.

Professor Dr. Md. Monwar Hossain
Principal
Mymensingh Medical College
Mymensingh, Bangladesh
Message

It gives me a great pleasure to congratulate the members of the Mymensingh Medical college alumni who have organized this grand unforgettable reunion here in New York. It needs deep devotion, sincere hard work and painful search to locate, identify and to organize such an organization so far away from its homeland Bangladesh. The organizer of this alumni reunion deserves a special thanks and appreciation.

Such a reunion will not only create an atmosphere of mutual professional acquaintance but also give a sense of family bondage amongst the members. I am sure this reunion will help each member to know each other and exchange ideas and experiences and sweet memories of the good old days in Mymensingh.

I feel deeply honored to be a part of this reunion and feel proud to be able to serve this distinguished institution from 1970 to 1978. I sincerely wish a grand success of the Mymensingh Medical College alumni reunion in USA and also all their future endeavors.

Professor Mohammed Fazlul Karim
Professor of Physiology &
Ex-Principal
Mymensingh Medical College.
I have been in the United States for over 32 years and been involved with various activities of Bangladeshi physician organizations. I always had the sense of lacking not having an alumni group of my medical college, especially since other physicians, every so often, were having the college alumni meetings. Some how I felt there were perhaps not enough physicians from Mymensingh Medical College in United States to create such entity.

Then I learned earlier this year about the initiative of few young Mymensingh Medical college alumni to organize MMC-DANA. Since then I have been happy and excited about the prospect of this organization. I am proud and happy to be part of this organization. This would provide a platform for all Mymensingh Medical College alumni to assist our medical college in every way, as we feel necessary and situation demands.

I sincerely congratulate the organizing alumni members to bring the dream to a reality. Let us take this opportunity and participate to make MMC-DANA a success, so we all can be proud of being alumni of Mymensingh Medical College.

Best wishes to you all. See you all at the Grand Reunion!

M. Abidur Rahman. MD. FACS.
M-3
Mymensingh Medical College.
President
MMC-DANA Ad Hoc Committee
# MMC-DANA Members List

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<th>Batches/Names</th>
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| Batch: M-01  | Dr. Mohammad A Motalib  
Dr. Rafiqlul Islam  
Dr. Ziaul Hasan  
Dr. Shariful Islam  
Dr. Maya  
Dr. Rowshan  

Dr. Chitta Ranjan Das |
| Batch: M-02  | Dr. Abidur Rahman  
Dr. G M Morsheed  
Dr. Abdul Haq  

Batch: M-03  
Dr. Abidur Rahman  
Dr. G M Morsheed  
Dr. Abdul Haq  

Batch: M-04  
Dr. Zahirul H Talukder Babul  
Dr. Chinmoyee Debnath  
Dr. Khodeza Akhter Shelly  
Dr. Shamsudin Ahmed  
Dr. Tasaddukul Karim  
Dr. Rezaul Karim  
Dr. Monayem Hossain  

Batch: M-05  
Dr. Mahmuda Majahar Rani  
Dr. Shila Sarker  

Batch: M-06  
Dr. Harun R Khan  

Batch: M-07  
Dr. Khairul Alam  
Dr. Rowshan Ara Begum  
Dr. Mira Rani Sharma  

Batch: M-08  
Dr. Shaukat Ali Khan  
Dr. Syed Nurul Huda  
Dr. Humayun Khalid  
Dr. Moinuddin Ahmed Selim  
Dr. Abdul Khaleque  

Batch: M-09  
Dr. Ferdous Ara Begum  

Batch: M-10  
Dr. Fazle N. Khan Tipu  
Dr. Fahmida Akhter Jolly  
Dr. Wazed Ali Khan  
Dr. Ghouse Mohammad Khan  
Dr. Eradul H. Khan Dulal  
Dr. Mahbubul Arefin Bhulu  
Dr. Shafi N. Khaled Sonny  

Batch: M-11  
Dr. Pariza Rahman Mia  
Dr. Selina Akhter Shelly  
Dr. Skdkar Mohd. Masud  
Dr. Ataur Rahman Masum  
Dr. Kajol Rekha Bhowmik  
Dr. Ataur Rahman Shaheen  
Dr. Harry B. Gomes  
Dr. Wahid Chy Shyamol  
Dr. Mahbub Rahman Selim  

Batch: M-12  
Dr. Md. Anisur Rahman  
Dr. Abdul Basit Khan  
Dr. Nazneen Ahmed Rina  
Dr. Md. Md. Kamal Mukul  
Dr. Nibraaz Parvin Ruma  
Dr. Ajoy Ghosh  
Dr. Panna  
Dr. Rosy  
Dr. Shafayet Zaman Nomir  

Batch: M-13  
Dr. Shaukat Ali Khan  
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Dr. Humayun Khalid  
Dr. Moinuddin Ahmed Selim  
Dr. Abdul Khaleque  

Batch: M-14  
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Dr. Shafi N. Khaled Sonny  

Batch: M-15  
Dr. Suraiya Sultana Ahmed  
Dr. Kakoli Hossain  
Dr. Ali Haider Rashidee  
Dr. Shahana Karim Shirleen  
Dr. Belayet Hossain John  
Dr. Shahana Huda Molly  
Dr. Nasreen Akhter Khan  
Dr. Lovely  
Dr. Sayeda Daisy Khan  
Dr. Yasmeen  

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A Review of Atypical Facial Pain

Dr. Shafi Khalid
M-14

Background: Atypical facial pain occurs in the territory of the trigeminal nerve, but the discomfort is not typical of trigeminal neuralgia. It may be as severe as trigeminal neuralgia, but its pattern and quality are different. Whereas trigeminal neuralgia is characterized by quick episodes of jabbing or lancinating pain, atypical facial pain is usually burning, aching, dull, or crushing. Moreover, an individual atypical facial pain attack always lasts longer than a few seconds and usually lasts minutes or hours (if not continuous). The distinction is important for making treatment decisions, because surgery, usually rhizotomy or vascular decompression, is highly effective for trigeminal neuralgia, whereas surgery is not appropriate for atypical facial pain.

A variation of atypical facial pain is discomfort similar to that of trigeminal neuralgia (e.g., lancinating) but atypical in location (e.g., far lateral on the face or in the occipital area). Some writers classify these as neuralgias of other nerves (in these examples, superficial temporal or occipital).

Frequency:
In the US: Although accurate figures are not possible because of the lack of agreement on criteria for classification, atypical facial pain is approximately one fifth as frequent as trigeminal neuralgia. The incidence of trigeminal neuralgia is 4-5 per 100,000 per year.

Race: No racial predilection is known.

Sex: Atypical facial pain affects both sexes with approximately equal frequency.

Age: The disorder mainly affects adults and is rare in the young.

History: Atypical facial pain must be distinguished from trigeminal neuralgia. It also must be distinguished from temporomandibular joint (TMJ) syndrome, migraine, and cluster headache.

- Trigeminal neuralgia is characterized by severe bursts of pain in one or more branches of the trigeminal nerve.
  - The bursts are quick, repetitive jabs of pain (lancinations). Each pain episode is only an instant in duration; the episodes recur irregularly many times a minute.
  - Trigeminal neuralgia episodes are not in synchrony with the heartbeat or pulse (usually slower).
  - The patient may wince, twitch, or cry out when a series of pain jabs occurs.
  - The pain is excruciating.
- Atypical facial pain may be as severe as trigeminal neuralgia, but its pattern and quality are different.
  - Pain episodes are always longer than a few seconds, lasting minutes to hours, and are sometimes continuous. The duration of the individual episode helps to make the distinction.
  - The pain is dull, aching, crushing, or burning.
  - Occasionally, patients use terms such as "sharp" or "knifelike" to describe the pain.
- TMJ syndrome is recognized by the following:
  - The location and quality of pain may be similar to those of atypical facial pain.
  - TMJ syndrome is characterized by focal tenderness of one or both TMJs and aggravation of Pharmacologic treatment is usually unsuccessful.
Treatment of TMJ syndrome (by an oral surgeon) often is directed at either the arthropathy of the joint itself or at fatigue and spasm of the pterygoid and temporalis muscles.

- The following signs and symptoms recognize migraine. Diagnosis of migraine may be acceptable if some of these features are absent, but generally at least 3 should be present. The essential feature is recurrent, severe headache with normal neurologic examination and blood pressure.
  - Female-to-male ratio is 3:1.
  - Pain is throbbing and synchronous with the heartbeat or pulse, usually on only one side of the head.
  - Headache may be accompanied by scalp tenderness.
  - Photophobia and sonophobia are common associated symptoms.
  - Nausea often accompanies migraine.
  - Patients often describe an aura at the onset.
  - Sleep (if it can be achieved) can abort a migraine.
  - Pharmacologic therapy, typically with ergotamine, isometheptene, or serotonin receptor agonists of the triptan family, is highly effective for migraine, almost to the point of diagnostic specificity. These agents are ineffective in atypical facial pain.

- Cluster headache, in terms of effective pharmacologic therapy, is closely related to migraine. Pain location and quality sometimes may be confused with atypical facial pain. Characteristics of cluster headaches include the following:
  - Male-to-female ratio is 6:1.
  - Pain awakens patient from sleep.
  - Onset of pain is sudden.
  - Episodes typically last minutes (but can last hours).
  - Pain is intense, crushing, or burning.
  - Pain recurs frequently up to several times a day in clusters of several days to weeks.
  - Strictly unilateral, the pain often is associated with ipsilateral conjunctival injection and nasal congestion.
  - Unlike migraine, cluster headache is very responsive to inhaled oxygen (i.e., abortive) therapy.

**Physical:**
- Neurological and physical examinations are usually normal.
- Trigger points, where pain is evoked by stimulation, are rare.
- The supraorbital or infraorbital foramen may be locally tender.
- Significant tenderness at one or both TMJs would favor the diagnosis of TMJ syndrome.

**Causes:**
- Atypical facial pain is usually without a specific cause. However, injury of any peripheral or proximal branch of the trigeminal nerve due to facial trauma or basal skull fracture can produce the disorder.

**Imaging Studies:**
- A brain MRI scans with gadolinium is the imaging modality of choice, although most studies will be normal. Examples of possible lesions include neuromas or cysts involving the trigeminal root or its distal branches outside the brain and malignancies of the skull base compressing or invading a branch of the nerve.
- CT scan of the head with contrast has a lower yield than MRI because of its poorer resolution of the brain stem and cranial nerves.
Medical Care:

- Medical treatment of atypical facial pain is less satisfactory than that of trigeminal neuralgia, reflecting the etiologic, symptomatic, and nosologic heterogeneity of the two conditions. Of the non narcotic drugs, tricyclic antidepressants give best results; phenytoin is of intermediate effectiveness, and carbamazepine is least effective. Any of these may be best in a particular patient.
- Of the new anticonvulsants, gabapentin and lamotrigine show promise, but their efficacy relative to the older drugs has not been established.
- In patients whose pain is typical of trigeminal neuralgia (e.g., lancinating) but atypical in location, carbamazepine is usually most effective, phenytoin of intermediate effectiveness, and tricyclics the least effective; gabapentin and lamotrigine retain their broad-spectrum positions.
- For neuropathic pain in any location, the same principle applies. For lancinating pain, the usual order of effectiveness is carbamazepine first, phenytoin intermediate, and tricyclics last. The reverse order is true for burning and other pain types.
- Aching joint pain and abdominal cramps are least likely to respond to these drugs. In all situations, the quality of pain is more important than its location.
- Narcotic treatment is sometimes appropriate as an adjunct but requires careful supervision, usually in collaboration with the patient’s pharmacist. The most effective program is to provide a monthly ration of narcotic that is strictly enforced and documented. Older patients fare better with narcotics since the risk of addiction is low in patients older than 70 years.

Interventional Therapy:

- Maxillary, Infraorbital nerve, and sphenopalatine ganglion block is sometimes effective, which may be followed by a neurolytic procedure, usually pulse radiofrequency.
  - Because of the risk of anesthesia dolorosa, surgery should be reserved for cases of medical intractability with repeated demonstration of pain interruption by nerve block.
  - Nucleus Caudalis stimulation with a laminotomy lead may be effective if initial trial is successful in suitable patient.

Reference

We Are Prouds of You .....  
MMC-DANA congratulates these students of M-40 and their teachers and parents for their outstanding achievement in securing 7 position out of 10 in the 1st Professional MBBS Examination held under the University of Dhaka, in May 2005.  
We wish you even greater success in future!

Md. Mahmudul Alam (Hasan)  
First Position

ABM Shafiuzzaman  
First Position

AFM Radwanor Rahman  
Third Position

Arup Kumar Chakrabarty  
Fifth Position

Saila Sharmin  
Sixth Position

Md. Farhad Hossain Heera  
Seventh Position

Easmin Haque  
Eighth Position
Morphology and Innervation Patterns of Regenerating Otolith Afferents after Ototoxic Damage

Dr. Zakir Mridha
M-24

The early studies found two types of sensory cells in the vestibular canal cristae: type I hair cell that are characterized by a flask shape, and type II hair cell which are more elongated. More recently, it is described that 3 classes of afferents (calyx, bouton and dimorphic) are present in the cristae and otolith maculae (Fernandez et al. 1988, Zakir et al. 2003). Calyx units innervate type I hair cells. Dimorph afferents contain calyceal and bouton terminals to innervate both type I and type II hair cells. Bouton afferents exclusively innervate type II hair cells with bouton terminals. Trauma, disease, or toxicity can produce damage to the vestibular receptor system, with immediate functional disabilities resulting. For example, ototoxic antibiotics produce stereocilia loss, hair cell death and degeneration followed by severe dysfunction. It is now firmly established that cochlear and vestibular sensory cells spontaneously regenerate after damage in fish, amphibians, and birds. However, little is known about how regeneration progresses and whether regenerated afferents ultimately develop normal terminal field. In the present experiments we quantitatively examined afferent regeneration at discrete periods up to 1 year in otolith maculae and compared with that of normal adult pigeons.

The bony labyrinth of adult pigeons was exposed, portions of the mastoid removed and the vestibule opened. Streptomycin (2 mg) was applied directly into the vestibule. The wound was closed. The birds were given a regular diet once fully awake. All animals were allowed to recover for a period extending between 1 week to 12 months after streptomycin is delivered. Groups of four animals each were examined at 1, 2, 4, 6, 8, 12, 24, 36, and 52 weeks post-streptomycin treatment (PST). Ten days before the end of the specific recovery period for each bird, biotin dextran amine (BDA) was injected into the vestibular nuclei and allowed to retrogradely transport along vestibular afferents. After 10 days survival the bony labyrinths were exposed, perfused, decapitated. The maculae were processed for BDA using a modified Diaminobenzidine procedure. The reacted utricles and saccules were then embedded into plastic and serially sectioned. In several animals the maculae were prepared for examination by scanning electron microscopy (SEM). BDA tracer-filled afferents were reconstructed using a video microscope and 3D tracing program. Morphological parameters for each of the reconstructed afferents were quantified, including axonal length, branch order, number of terminals (the number of type I hair cells and bouton endings), number and volume of calyces and innervation area.

Results:
Stage 1 regeneration: During the first 4 weeks of regeneration immature fibers were first observed, and these gradually developed more elaborated branching patterns and terminal fields at 1 week PST, no visible stereocilia were observed. Parent axon enters to the neurepithelium, but there were no hair cells. Most of the innervation patterns observed were very simple with only a single segment (Fig. 1A). At 2 weeks PST, the number of visible hair cells was only a fraction of normal patterns, patterns. Some of the hair cells in the epithelium were beginning to mature, with complete and morphologically polarized stereocilia bundles (Fig. 1A1-i). However, many of the stereocilia were immature in appearance with only few cilia and no distinguishable kinocilum. At 4 weeks PST the stereocilia appeared in increasing number. In fact, the density of cells had doubled over the 2 weeks. However, only growth cones, en passant and boutons terminals were ever observed as terminal structures (Fig. 1A).

Figure 1: Photomicrographs and anatomical reconstructions of stage 1 (Fig. 1A), stage 2 (Fig. 1B), and stage 3 (Fig. 1C) regenerating afferents. Fig. 1A a-h, afferents innervating the macula at 1 week PST were primarily composed of a simple initial fiber segment, prominent swellings, and a large terminal growth cone. A i-o, for 2 week PST regenerating afferents. 1. SEM image of the apical surface of the utricular macula. A 1-o, simple branched afferent structures with terminal growth cone processes directed toward hair cells were observed for most afferents. A p-v, afferents at 4 weeks PST exhibited more numerous short-branched segments, most with growth cones and bouton terminals (red circles) was observed. Asterisks denote penetration point into the sensory epithelium. Fig. 1B a-i, 6 weeks and 1B (j-r) 12 weeks PST. B a, SEM of striola region near the polarization reversal line (dotted). B b-f, developing dimorph afferents with two prototypic calyceal terminals in the striola region. B d, g-i, bouton afferents exhibited increased structural complexity, many with both terminal boutons and growth cones at 6 weeks PST. B j, medial striola region at 12 weeks PST, with reversal line (arrow). B k, n and o, calyceal terminals were present for the first time in calyx afferent at 12 weeks PST; but these fibers were few in number and most were immature. The majority of calyceal terminals were contained in dimorph afferents, which appeared more structurally complex (B i, p). Bouton afferents increased in complexity with dense arborizations and numerous terminals (B q). Fig. 1Ca, SEM of central striolar region. C b, DIC photomicrograph of central striolar region in 24 week PST maculae. C c-u, reconstructions for utricular C(c–k) and saccular C(n–u) afferents for stage 3 regeneration. Calyx, dimorph and bouton afferents arranged in increasing order of complexity. Insets, Composite surface maps of stage 3 regeneration utricular (left) and saccular (right) maculas. Distributions of reconstructed calyx (diamonds), dimorph (squares), and bouton (circles) afferents are shown. Scale bars, 10 μm.
No calyceal terminals were present. As shown in Figure 1A, simple afferents contained few short branching processes with large growth cones (Fig. 1Ar). More complex fibers exhibited a number of branches, some terminating growth cones and others ending with terminal boutons (Fig. 1As–u). Quantitative evaluations for all stage 1 reconstructed afferents were plotted for comparison in Figure 2. No calyceal terminals were ever observed during 1st stage; thus only bouton afferents existed in the receptor epithelium. The afferent terminal structure complexity increased as regeneration progressed. The overall length of the fiber, the number of branching segments, the branch order, the number of growth cones, and the area of epithelial innervation all increased through the 4 weeks of stage 1 regeneration (Fig. 2).

Stage 2 regeneration: The second stage of regenerative recovery extends from 6 to 12 weeks PST, several significant changes in afferent innervation were observed. At 6 weeks PST, many receptor cells were mature as exhibited by fully developed stereocilia bundles that were morphologically polarized (Fig. 1Ba). The polarity orientations were organized relative to the developing reversal line called striola. Most of the bouton afferents exhibited more branching pattern with a mixture of terminal boutons and growth cones. Dimorph afferents begin to develop next, with calyx following last. At 12 weeks PST, the density of cells continued to increase such that the epithelium was approaching a more normal appearance. Calyx afferents were observed for the first time (Fig. 1Bk), confined to the striolar region. At this stage, the majority of the calyx units had immature partial calyceal terminals. Both the dimorph and bouton afferents continued to increase in structural complexity (Fig. 1B, i, q and r).

Stage 3 regeneration: The final stage of regeneration included the period extending between 24 weeks and 1 year PST. After 6 months regeneration, the hair cell density had returned to normal values. Figure 1Ca shows a patch of regenerating sacculum epithelium in the central striolar region with densely packed hair cells. The stereocilia bundles are complete with well-formed polarizations. At 24 weeks PST, no new developing afferents to enter the receptor epithelium and no growth cones on existing afferents were observed. For these reasons, we concluded that the regeneration was essentially complete at 24 weeks PST. Even so, we continued to collect data from regeneration birds at 36 and 52 weeks PST, in the event that some afferent terminal recovery continued. Close inspection of the afferent innervation patterns for individual fibers suggested that significant differences between the complete terminal fields of regenerated afferents and those developed normally existed. The size, structure, and complexity of the afferent innervation patterns varied widely. Figure 1C illustrates the relative diversity in calyx, dimorph, and bouton innervations, from simple to the more complex for all regenerated afferents. For all reconstructed calyx afferents in regeneration maculae, the calyceal terminals varied between simple structures, which innervated only one hair cell, to the more complex calyces, which contained up to 17 hair cells (Fig. 1Cf). In normal birds, calyx afferents had between 1 and 15 hair cells per calyceal terminal (Zakir et al., 2003). Simple dimorph afferents had small calyceal terminals that contained a single hair cell and one or two branch segments that contained a few terminal boutons. Bouton afferents also varied from small to large, the arborization complexity, number of branches, and quantity of bouton terminals again appeared less for fibers innervating the stage 3 regeneration maculae compared with the normal maculae (Fig. 1C).

It was decided to pool the morphological data for statistical comparisons between regeneration and normally developed afferents (Fig. 3). Regeneration calyx fibers had significantly lower fiber volumes, fewer calyceal terminals, fewer type I hair cells per fiber, and lower innervation densities than afferents innervating normally developed maculae. As also shown in Figure 3 (asterisks), both dimorph and bouton afferents had significantly lower fiber diameters, fewer branch segments, lower volume, and smaller innervation areas than normally developed afferents. The number of bouton terminals per afferent (dimorph and bouton) for treated and normal animals was not different. However, the mean innervation density for both dimorph and bouton afferents was actually significantly higher in the regenerated epithelium, whereas the calyx density was smaller (Fig. 3). For calyx afferents, there were fewer hair cells per fiber in regenerated afferents, yet the innervation areas were not that different from normal fibers, which produced a lower terminal density. In contrast, regenerated dimorph and bouton afferents had equivalent numbers of bouton terminals per fiber, yet smaller branching arborizations and innervation areas, thus significantly higher densities than normally developed afferents. Thus, these findings

![Figure 2: Mean values for regenerating afferent structural characteristics as a function of recovery time (PST). The innervation area (Area; in square micrometers) and total fiber length (Length; in micrometers) values were reduced by a factor of 10 for illustration purposes. Other structural features illustrated include the number of branching segments (Branches), branch order (Br. Order), number of terminal growth cones (Cone), number of boutons per fiber (Boutons), number of type I hair cells/calyceal terminal (Calyx HC), and the fiber diameter (Diameter; in micrometers).]
suggest regenerated fibers innervate fewer hair cells in the maculae, but have more bouton terminal contacts per receptor hair cell compared with normal maculae.

### Discussion and Summary:

Our findings present the first comprehensive treatment of vestibular afferent regeneration in any species. We found that regenerated receptor cell distributions, planar polarities, and location of the reversal line were all morphologically normal. The calyx, dimorph, and bouton afferents all regenerate and were similarly distributed to topographic norms, but do so much slower than previously thought. We observed a staged temporal sequence of afferent regeneration, in which bouton fibers recover first, followed by dimorph, and then calyx afferents. However, we also found that the completed innervation patterns for individual regenerated afferents differed significantly from those in normal pigeon. Both the hair cell and their innervating afferents differentiate from an early form into more advanced forms during regeneration. First, we found that bouton afferents initially innervate the striolar regions, and then gradually get replaced by dimorph afferents. Second, partial calyces appeared to form on regenerating fibers that already contained branches with bouton terminals. Third, calyx afferents regenerated last, with their calyceal terminals being larger and containing more hair cells than their neighboring dimorph afferents. Thus, we propose that bouton afferents in the striolar regions develop a calyceal terminal to become dimorphs, as a result of an as-yet unknown cue. Similar transdifferentiation of simple regenerated dimorph afferents then occurs, with loss of their few bouton branch segments and enlargement of their calyceal terminal. Similar to our findings for vestibular afferents, a progression from early bouton terminals to the more mature cup-like terminals on regenerating chick cochlear afferents has also been described (Duckert and Rubel, 1993; Hennig and Cotanche, 1998). Finally, we show that regenerated terminal morphologies were significantly less complex than normal fibers. Spontaneous regeneration of mammalian hair cells does not occur. However, recent developments in mammalian regeneration provide renewed hope for restoring hearing and balance after loss (Izumikawa et al., 2005; Sage et al., 2005). The adaptability of the neural processing of motion information in regenerative systems should be expected during functional recovery and deserves additional investigation.

### References:


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![Figure 3: Comparison of regeneration and normal afferent innervation morphological parameters. Mean values are shown for each parameter for normal (gray) and regeneration (hatched) afferents. Diameter, Average fiber diameter (in micrometers); Branches, number of branch segments per fiber; Length, sum of all branch segments (in micrometers); Volume, sum of all fiber branch segment volumes (in cubic micrometers); Area, innervation area of receptor epithelium (in square micrometers); Cal Term, number of calyceal terminals per fiber; HCL, number of type 1 hair cells per fiber; Bouton, number of bouton terminals per fiber. Density, terminals/innervation area (number per square micrometer). For clarity, quantities for fiber length, innervation area, and volume are plotted as a factor of 10 reduction. All quantities are plotted relative to the left ordinate, except density, which was scaled on the right ordinate. Error bars indicate SE. *p < 0.05 level.](image-url)

Zakir H. Mridha was born in Munshigonj, Bangladesh. He received the Bachelors degree of Medicine and Surgery (M.B.B.S.) from the Mymenasing Medical College under the Faculty of Medicine, University of Dhaka, Bangladesh in 1994 and the Ph. D. degree in Medical Sciences from the Tokyo Medical University, Tokyo, Japan, in 2001. Currently, he is working as a Research Associate, at Washington University School of Medicine in St. Louis, Missouri, USA. His primary interests of research are vestibular neurophysiology, hair cell regeneration processes and ensuing vestibular function, effect of gravity in development of receptors in vestibular system.
LOOKING BACK TO THE GOLDEN PAST

Dr. Fazle Nasim Khan
(M-14)

The alumni of Mymensingh Medical College (MMC) in North America are holding its ‘Grand Reunion’ on July 2, 2006 in New York. It is a historic occasion and should be a very memorable event for those who attend. It’d be a time to look back at the past, revitalize the old friendship and bonds in the present and plan for the future.

The fourteenth batch of MMC started its journey in November 1976. I still vividly remember the first day in college. On the very first morning we were taken straight to the dissection room and assigned to “bodies” by the groups. There were bodies lying on the dissection tables on both the floors. Quader bhai was moving back and forth between tables. Nasiruddin sir was the teacher for our Batch A. He gathered us round a table and gave a brief introduction to dissection. Some of the good ones amongst us had their scalpels and forceps in pocket; some also had their “Cunningham’s” in hand. We then proceeded to do dissection. Most of us were hesitant, if not scared, to touch the bodies, lest we make a ‘mistake’ in trying to dissect and ruin the body! That’s how all-future doctors start their long journeys in medical colleges.

Days, months and years passed by. The first two years for our batch were perhaps the best. The country was virtually under martial law and there was no student politics. The environment was very peaceful indeed. In the six years we spent to cover the five-years MBBS course, we saw lots of changes take place, some good, some not so good ones. We held many class and college programs on different occasions (sometimes without any occasion). I still clearly remember the highly successful musical parody program “Medico Cartoon” in December 1977, the drama, the musical and variety programs we held, the picnics, the lectures and practical classes, wards, the teachers, the exams, the elections, the political fighting’s, violence and all that. Just too many to list and recall. Those years were also memorable for many of us who found their soul mates there.

At the completion of our internship in December 1983, our batch got scattered all over. Some went to work in the rural health centers, some in the bigger hospitals, some went overseas. Now after 22 long years, it’s really exciting to get back together with many of them. Just like the great poet Rabindranath Thakur sang: “Hae majhe holo chhara chhari, gelem ke kothae; abar dekha jodi holo shokha, praner majhe aye”.

We need to unite not only to enjoy the fellowship with each other but also to put our individual talents, abilities and resources collectively for the good of our alma mater and for the poor people of the country to whom we owe a lot. At this moment of reconnecting to our past and enjoying the present, let’s focus on our future and make this organization an effective vehicle for serving the fellow humanity. I sincerely thank all those who worked day in and day out to bring us to this stage. May Allah bless them all and help us guide our talents and resources not towards competition for positions, but for serving His creations through this organization.
**Daptomycin for Treatment of Persistent Gram-positive Bacteremia and/or Infective Endocarditis (IE) that failed to respond to currently available antimicrobial therapy**

Dr. Mohammud Alam

Batch M-19

Mohammud Alam¹, Brijesh Goyal¹, Vinod Gulati¹, Victor Jimenez², Richard Go², ¹Brunswick General Hospital Amityville, NY and New Island Hospital, Bethpage, NY, ²Stony Brook University Hospital, Stony Brook, NY.

**Background:** Mortality associated with bacteremia and/or IE remains high particularly those associated with drug-resistant gram-positive bacteremia such as MRSA and VRE. The treatment of these life threatening infections can be difficult and challenging when unresponsive to other antimicrobial agents. Daptomycin, a novel lipopeptide with rapid bactericidal activity against drug-resistant gram-positive strains recently approved for complicated skin and soft tissue infections, may provide an effective option particularly when difficult to treat infections.

**Methodology:** Retrospective analysis of medical records from three medical centers of all patients diagnosed with persistent bacteremia (5 days or more) and/or IE and treated initially with conventional therapy (Oxacillin, or Vancomycin or Linezolid or in combination) that required a change to Daptomycin at with 6mg/kg Q daily.

**Results:** A total of 23 patients were identified with bacteremia and 13 patients with Endocarditis, 4 with Para spinal abscess, 1 with Para spinal and epidural abscess, 2 with septic arthritis, 1 with prosthetic joint infection and 2 with no obvious source. 14 with MRSA, 4 with MSSA, 4 with VRE, 1 with Streptococcus and. Prior antibiotics therapy was used in all patients either with inadequate response or with adverse events that require a change in therapy. Overall of 20 of 22 patients had a successful clinical outcome with Daptomycin.

<table>
<thead>
<tr>
<th>Age &amp; Sex</th>
<th>Co morbidities</th>
<th>Strain</th>
<th>Length of Bacteremia</th>
<th>TTE/TEE</th>
<th>Initial Antibiotic Therapy(s)</th>
<th>Neg. BC once Dapto Initiated</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>F/95</td>
<td>DM/HTN/ Dementia</td>
<td>MRSA</td>
<td>19</td>
<td>TTE (-) TEE refused</td>
<td>Vancomycin/ Linezolid</td>
<td>1</td>
<td>Pt disc on IV Dapto</td>
</tr>
<tr>
<td>M/87</td>
<td>HT/V-HD/ Gl Bleed/PVD</td>
<td>MRSA</td>
<td>14</td>
<td>TEE (+)</td>
<td>Vancomycin/ Linezolid</td>
<td>3</td>
<td>Pt disc on IV Dapto</td>
</tr>
<tr>
<td>F/76</td>
<td>UTI/ Seps/ Dementia/ DIV</td>
<td>MRSA</td>
<td>7</td>
<td>TEE (+)</td>
<td>Vancomycin/ Linezolid</td>
<td>2</td>
<td>Deceased aft 11 days</td>
</tr>
<tr>
<td>M/68</td>
<td>DM/A-Fib/ CHF/ CAD</td>
<td>MSSA</td>
<td>5</td>
<td>TEE (+)</td>
<td>Vancomycin/ Oxacillin</td>
<td>1</td>
<td>Pt disc on IV Dapto</td>
</tr>
<tr>
<td>M/74</td>
<td>DM/ PVD/ Rt. Foot Ulcer</td>
<td>MRSA/VRE</td>
<td>11</td>
<td>TTE (-) TEE (+)</td>
<td>Vancomycin/ Linezolid</td>
<td>2</td>
<td>Pt disc on IV Dapto</td>
</tr>
<tr>
<td>M/86</td>
<td>HT/CAD/ Diverticulosis</td>
<td>Streptococcus</td>
<td>6</td>
<td>TEE (+)</td>
<td>Vancomycin</td>
<td>2</td>
<td>Inpt. On Dapto</td>
</tr>
<tr>
<td>M/77</td>
<td>HT/Rt. Hip Prosthesis</td>
<td>MSSA</td>
<td>8</td>
<td>TEE (-) TEE (-)</td>
<td>Oxacillin/Vancomycin/ Linezolid</td>
<td>2</td>
<td>Pt disc on IV Dapto</td>
</tr>
</tbody>
</table>
Conclusion: Our experience suggests Daptomycin maybe an effective alternative treatment for patients with persistent gram-positive bacteremia and/or IE. The safe and rapid resolution of these resistant gram positive infections may provide decrease mortality and length of hospital stay with the benefit of potential cost reduction which need further studies.

References:
Robert D. Arbeit, Dannis Maki, Francis P. Tally, The safety and efficacy of Daptomycin for the treatment of complicated skin and skin-structure infections Clinical Infectious Diseases, 2004; 38: 1673-81


Mohan S. McDermott BP, Cunha BA. Methicillin-resitant Staphylococcus aureus (MRSA) prosthetic valve endocarditis (PVE) with paravalvular abscess treated with Daptomycin Heart Lung 2005; 34:69-71

Apology for the Death of Your Father

AMRITA KHALID

I
You were the kind of
Girl that detached herself
Early. You were born unlinked,
Free, nature-tossed. He named
You in Sanskrit, named you after
Flowers.

They told me your father died
On a soft, blue Tuesday
Morning. The night was warm
Before, they said he died in his
Sleep, passing slowly, exhaling.

II
On coming to see you, I
Felt as if I could never
Really see you. On parting,
I felt like I could never part
Sooner.

If I could tell you how
Sorry I was, I would, if I knew
It would help, I would.

III
The mourning in my voice
Lilts, it forms like a
Wine-glass, transparent
And hollow.

You look up, your
Eyes, hot, blue, and mellow.
With your eyes, you break it.

IV
The sick politeness of death,
You’re not the one for it.

I remember when we were too
Young to know any better,
Our families went swimming
At the lake, and we went so far.

We got lost in depths of the water,
Everything was razor-wet and blue,
And you got lost so hard all you
Could do was drink it.

I held onto a rock and watched them
Save you, your body slick and robotic,
Your chest still. I thought you were killed.
Face cut from the rocks,
Lightly bleeding that ugly, alien liquid.
Ask me to recall, and
I’ll tell you, how sick that was, how
Polite that was.

V
I unearth you. Your arms
Are starved like tree branches.

You’re too limp to move, so I
Look at you, pull you apart, quietly,
Carefully. How cold you are, how
Exhausted and tiny and cold.

I look at you.
Look at
What a mess
Life has made of you.

Days will pass, years, even,
When you’re walking over stones
To avoid him, or to confront him.
People will hush out his name, you’ll
Ache to forget him, you’ll cross him
Out savagely, and then reach out,
Arms flailing, screaming out in
Agony, the instinctual bond
Between daughter and father,
Long buried, and you, the half-
Orphan, the incomplete growing
Animal, your father’s face blurring.

You’ll fall asleep.

One day, you’ll wake and you’ll
Realize that you’re without him,
You’ll know that you’ll be fine.

Because that’s all we can ask, in the end.

To be fine.

Amrita Khalid, daughter of Dr. Shafi Khalid and Dr. Pariza Rahman (M-14). This Piece earned “The Patricia Grodd Poetry Prize for Young Writers”.
AVIAN INFLUENZA (BIRD FLU) AND THE NEXT PANDEMIC
Dr. Reza Chowdhury
M-17

It always amazed me how tiny microbes outplayed human beings time after time. We know about the devastating damage done by small pox and plague epidemic to mankind in the past centuries, taking away thousands of lives. Over time mankind were able to stop those outbreaks by increasing their knowledge and understanding about bacteria and viruses, developing antibiotic to kill the bacteria and viruses, improving sanitation and environmental hygiene to break the vectors that help spreading the germs.

Then came the HIV/ AIDS epidemic in the early eighties, when scientists and doctors around the world have no clues about the virus and the disease, only they were hopelessly watching thousands of people dying of AIDS, Acquired Immunodeficiency Syndrome. But within a span of a decade scientist were able to identify the virus, HIV, its molecular genetics, its pathogenesis and antiviral drugs that efficiently kill the virus and stop its replication and spread. Once HIV was called “passport to death”, that means once you have HIV, you will die, today or tomorrow, regardless of treatment. Today in the developed countries HIV infected patients are living a healthy and productive life. In 2006 with so many effective Antiretroviral drugs available to kill the virus, HIV is able to put its name in the list of other chronic diseases, like diabetes, where once you have the disease you have to take medication rest of your life to be healthy and alive.

Since the beginning of twenty first century the microbe that is making the headline news again and again in the Bird Flu virus or Avian Influenza Virus. Why it is making the headline? Because scientist are predicting that in the near future, it will acquire the ability to spread from human to human more efficiently and since human has no immunity to this new strain of Avian influenza virus, it has the potential to cause the next pandemic, killing millions of people worldwide.

Avian Influenza (Bird Flu)

Avian influenza is an infection caused by avian (bird) influenza (flu) viruses. These influenza viruses occur naturally among birds. Wild birds worldwide carry the viruses in their intestines, but usually do not get sick from them. However, avian influenza virus is very contagious among birds and can make some domesticated birds, including chickens, ducks, and turkeys, very sick and kill them.

Infected birds shed influenza virus in their saliva, nasal secretions, and feces. Susceptible birds become infected when they have contact with contaminated secretions or excretions or with surfaces that are contaminated with secretions or excretions from infected birds. Infection with avian influenza viruses in domestic poultry causes two main forms of disease that are distinguished by low and high extremes of virulence. The low pathogenic form may go undetected and usually causes only mild symptoms. However, the highly pathogenic form spreads more rapidly through flocks of poultry. This form may cause disease that affects multiple internal organs and has a mortality rate that can reach 90-100% often within 48 hours.
Human infection with avian influenza viruses

There are many different subtypes of type A influenza viruses. These subtypes differ because of changes in certain proteins on the surface of the influenza A virus (hemagglutinin [HA] and neuraminidase [NA] proteins). Usually avian influenza virus refers to influenza A viruses found chiefly in birds, but infections with these viruses can occur in humans. The risk from avian influenza is generally low to most people, because the viruses do not usually infect humans. However, confirmed cases of human infection from several subtypes of avian influenza infection have been reported since 1997. Most cases of avian influenza infection in humans have resulted from contact with infected poultry (e.g., domesticated chicken, ducks, and turkeys) or surfaces contaminated with secretion/excretions from infected birds. The spread of avian influenza viruses from one ill person to another has been reported very rarely, and transmission has not been observed to continue beyond one person.

Symptoms of avian influenza in humans have ranged from typical human influenza-like symptoms (e.g., fever, cough, sore throat, and muscle aches) to eye infections, pneumonia, severe respiratory diseases (such as acute respiratory distress), and other severe and life-threatening complications. The symptoms of avian influenza may depend on which virus caused the infection.

The avian influenza virus that cause outbreak in poultry and infection from bird to human and human to human is Influenza A (H5N1), (where H stands for hemagglutinin and N- neuraminidase). Most of these cases have resulted from people having direct or close contact with H5N1-infected poultry or H5N1-contaminated surfaces.

Let's look at the devastation already done by the Avian Influenza H5N1 virus: - Although avian influenza virus generally replicate inefficiently in humans, some subtype of avian flu can replicate within the human respiratory tract and cause disease. The first association of avian influenza H1N1 with clinical respiratory disease occurred in Hong Kong in 1997, when 18 human cases occurred during a poultry outbreak of highly pathogenic H5N1 influenza in live bird markets. This outbreak was associated with high mortality rate (33 percent), a high incidence of pneumonia (61%), and a high rate of intensive care (51%).

In 2003, H5N1 reemerged in humans when two culture-confirmed cases occurred in a family group returning to Hong Kong from China. In late 2003 and continuing through 2004 and 2005, H5N1 virus have caused multiple outbreaks among poultry across Asia, including Cambodia, China, Indonesia, Thailand, Vietnam, and Turkey. A total of 130 human cases have been reported with a mortality rate of greater than 50 percent, in these outbreaks.

Also a major concern is that migratory birds are spreading avian flu virus, after isolation of H5N1 strains, related to the 2005 Chinese viruses in wild birds in Romania, Turkey, Croatia and Greece. Avian influenza H9N2- In Hong Kong in 1999 and 2003 influenza H2N2 viruses were isolated from children.

Avian influenza H7 – extensive outbreak of H7N7 occurred among poultry in the Netherlands. Among the workers who were involved in the slaugthering of the poultry, 83 person were infected with the virus. Needless to say that in each of these outbreaks millions of poultry were slauhtered or killed to prevent the outbreak, costing millions of dollars.
What changes are needed for H5N1 or another avian influenza virus to cause a pandemic?

Three conditions must be met for a pandemic to start: 1) a new influenza virus subtype must emerge for which there is little or no human immunity; 2) it must infect humans and causes illness; and 3) it must spread easily and sustainably (continue without interruption) among humans. The H5N1 virus in Asia and Europe meets the first two conditions: it is a new virus for humans (H5N1 viruses have never circulated widely among people), and it has infected more than 190 humans, killing over half of them.

However, the third condition, the establishment of efficient and sustained human-to-human transmission of the virus, has not occurred. For this to take place, the H5N1 virus would need to improve its transmissibility among humans. This could occur either by "reassortment" or adaptive mutation.

Reassortment occurs when genetic material is exchanged between human and avian viruses during co-infection (infection with both viruses at the same time) of a human or another mammal. The result could be a fully transmissible pandemic virus—that is, a virus that can spread easily and directly between humans.

No one can predict when the next pandemic might occur. However, expert from around the world is watching the H5N1 situation in Asia and Europe very closely, for the emergence of a novel virus that will result from the reassortment of avian H5N1 and human influenza virus, that will transmit from human to human and cause a new catastrophe.

Do we have a vaccine against the new virus? Not yet.

Do we have a drug to treat the virus? Currently available oseltamivir, may be of some benefit.

Can we win the the battle against the new avian flu virus? Only time can say.

Once an Infectious Diseases Specialist told in his lecture, how can we win against microbe, they came before us in this planet and a healthy human body contains two millions bacteria at any given time!

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MMC-DANA: How We Got Here, and What’s Next

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“Never doubt that a small group of thoughtful, committed citizens can change the world. Indeed, it is the only thing that ever has.”
-Margaret Meads

It indeed began with a dream!

The idea of MMC-DANA conceptualized when a group of friends met here in New York City on an October morning in 2004. The need was felt repetitively from time to time, constraint of time and stress came between many who felt that such an organization should have long existed. To start, a few email addresses and telephone numbers were gathered, and in December, a group-email was generated to about twenty-five people.

Enormous response followed! Soon a database was in place with a few more names, addresses and numbers, and the process of snowballing started. In September 2005, our website (www.mmc-dana.org) was launched with the idea of using it as a platform for the organization activities. The first reunion took place in Columbia, Missouri in December 2005, mainly to create a thrust on the organization’s activity and future infrastructure. By then we learned that about three hundred MMC doctors currently reside in USA and Canada, however, MMC-DANA database had only about fifty names. A substantial task was planned to collect more names. Any and every efforts were being undertaken to enrich the members’ database. Countless hours were spent by a small group of enthusiastic people on collecting members and spreading the news.

The possibility and feasibility of a grand reunion was being debated, especially on choosing the place. Finally New York was being selected considering the vast number of MMCians’ presence in and around. More people were on board by the beginning of 2006. Application was being sent for licensure as a non-profit voluntary organization in March, which got approved later. A massive plan was set to successfully arrange and implement the reunion activity. A new link had been added in our website just for the 2006 New York Reunion, which actually added to the momentum as a conduit to effectively convey our message. Members’ response skyrocketed ever since that launch. Continued Medical Education (CME) had been incorporated with the reunion activities, plus plans to publish this souvenir, cultural function, selection of venue, etc. followed in a very short scope of time.

The members’ list is in print elsewhere in this publication. Innumerable hours, patience and tirelessness are behind this innocent looking list! Ninety percent of the members were at any point communicated individually over telephone, and group emails have been sent every time there was something important to notify. No member information has been ever published other than the names and batch. Careful attention has been practiced strictly not to disclose personal information that compromises privacy issues.
Future plans include scholarships for meritorious, but financially challenged current students of Mymensingh Medical College, cooperation with Dhaka University and college administration regarding physicians in Bangladesh to attend and avail scholarships, fellowships, trainings, and opportunities in North America, sponsor a MMC-DANA corner in MMC library equipped with computer, books and medical journals, etc. to mention a few. Every member is encouraged to come up with ideas and suggestions, and all are welcome to contribute positively on these issues. Our website will serve as the portal for all communications, and contact information is listed there.

No names are being mentioned in this piece; it is not important who are the people behind the scenes or who made it possible, what is important is let us all face the challenge to keep it moving forward. Let’s make some impact! We are a group of doctors, we belong to a privileged group, and we have the ability to do better. We must not forget our past, and the vast population back in Bangladesh who most deserves our contribution. We must not forget our beloved institution and hospital, where we got trained to be who we are today. Let’s do something for those who really deserve our services!

Many of us here are successful and well established, while others are still struggling. Let us extend cooperation, compassion and respect to other MMCians who are yet to prosper, in terms of jobs, residency positions, and any assistance! The least we can do is not to offend anyone, based on status. We are all MMC-doctors, we all belong to the same past, and we must not discriminate within MMC-DANA, based on licensing status or so.

An organization that is still in its infancy is vulnerable to make mistakes, let us learn from it. We all must understand that MMC-DANA activities are totally voluntary and it is expected that every members and affiliates understand that there are no financial or worldly incentives for contribution. This is a doctor’s organization, and we are bound to maintain a certain degree of sophistication at any cost.

Please step forward to make MMC-DANA a successful organization. Everything that is being done so far, got done with a handful of people’s efforts and hard work on one hand, and was not done easily, as we still deal with irresponsibility and attitudes motivated by ego, in another group of people. We can at least not complicate things while a good number of devoted people are dedicating their time and talent to make some impact. We know we cannot change the world, but we can at least make the best use of our aptitude and efforts to make changes.

Let us keep this dream alive!!