Atrial Tachycardias are a common problem following the surgical repair of complex Congenital Heart Defects (CHD) and have a poor response to medication. Even patients with simple CHD, such as an atrial septal defect, can exhibit atrial tachycardias postoperatively, especially if the surgical repair is performed at an advanced age. The appearance of these tachycardias is associated with a deterioration of cardiac function, the occurrence of thromboembolic complications and sometimes sudden death. Management with medication is problematic, firstly because many patients have an unsatisfactory response to therapy, and secondly because of the frequent side effects of antiarrhythmic drugs. In addition, apart from the tachycardias, many patients also exhibit severe dysfunction of the sinus or atrioventricular node that can be exacerbated by pharmaceutical treatment, necessitating the implantation of a permanent pacemaker.

The successful treatment of other forms of tachycardia with radiofrequency (RF) current has led to the application of the same method in patients with postoperative atrial tachycardias especially 3D Mapping. The results in many cases have been very encouraging, although a number of problems still remain to be solved. We present an interesting of the treatment of atrial tachycardia in postoperative CHD patients using RF ablation with 3D Mapping in our hospital.

Case Report

A 17 year old girl from Iraq, was diagnosed as a case of congenital heart disease in her country at the age of 8 and operated for large perimembranous VSD after cardiac catheterization procedure which as per her records showed large VSD with severe PAH with Qp/Qs of 1.8:1. Post operatively she did well for a few months but gradually started having dyspnea on exertion. For last 2 years she also started having occasional palpitations at rest, which increased in frequency with episodes of pre syncope and perspiration from 5-6 months. She was evaluated at their local hospital and 2DEcho done their revealed PAH with RV dysfunction with ECG showing wide QRS with RBBB and RAD. She was referred to us for further management.
On examination her heart rate was 90/min, RR-22/min, bilateral basal crepts and pedal edema, JVP raised with prominent 'a' waves. CV5-S1 N, S2 wide split pansystolic murmur LL5B, RV S3 present. Liver was palpable 3 cm below subcostal margin. Baseline saturation was 88%, on O2 99%. The Chest X Ray showed prominent MPA segment and hilar pulmonary arteries with peripheral pruning.

Echo revealed intact ventricular septum, hugely dilated RA and RV, moderate TR, TR gradient 88 mmHg (SBP-100 mm Hg), RV dysfunction, with minimal pericardial effusion seen.

Her baseline ECG showed RBBB with RAD with atrial flutter with 2:1 conduction. QRS was 160 msec. She developed palpitations in CCU with hypotension, ECG showing wide QRS tachycardia with same morphology as baseline. Diagnoses of atrial flutter with 1:1 conduction and ventricular rhythm of 180bpm was made. She was stabilized on medical therapy. She underwent cardiac catheterization, which revealed systemic pulmonary pressure and no residual shunt at ventricular or great vessel level.

To improve the symptomatic status of the patient she was taken up for Electrophysiological Studies and Radiofrequency Ablation using Ensite Velocity 3D Mapping System. A decapolar catheter was placed in coronary sinus. A quadripolar catheter was placed at the high right atrium. Abalton catheter was used to map the huge right atrium. Activation and propagation maps were created. A diagnosis of Isthmus dependant clockwise atrial flutter was made. Activation map showed large areas of scar in the right atrium. Isthmus line was drawn using 4mm Therapy Coolflex irrigated catheter. Flutter broke during the lesion to sinus. Line was completed. Post ablation line of block was demonstrated at the isthmus. Patient became symptomatically better with sinus rhythm.

Three days post procedure her basal saturation on air was 92%. 2DEcho showed improved PA pressure (TR gradient 68 mm Hg, SBP-100 mm Hg), and improved RV systolic function. Patient came for follow up maintaining her good symptomatic status and no palpitations 6 months later.

CONCLUSION

Patients who undergo surgery for CHD often exhibit atrial tachycardias that are refractory to medication. RF ablation with 3D Mapping can offer significant benefit to most patients. The acquisition of experience and the use of new technology for mapping and ablation help to increase the efficacy of the procedure.

REFERENCES

1) Arrhythmias in adult patients with congenital heart disease Edward P. Walsh and Frank Cecchin Circulation 2007;115:534-545


4) Three dimensional electroanatomical mapping of right periatrial tachycardias after interatrial defect correction. Frank Halimi, Manlio Marquez, Jareme Lacotte, Francois Hidden – Lucet, Joelci Tonet, Robert Frank Archives of cardiovascular disease (2008) 101,533-538

Quality Initiative by the Endocrinology Department for Management of Hypoglycemia

Dr. Sujeet Jha
Director – Max Institute of Endocrinology, Diabetes and Metabolism
Max Super Speciality Hospital, Saket

Hypoglycemia is one of the most feared complications of diabetes treatment. Traditionally hypoglycemia management in Indian setting is doctor centric rather than nurses centric. Inadequate awareness among nurses and lack of standardized protocol are also the major factors contributing to delay in treating hypoglycemia leading to increased morbidity and prolonged hospital stay. In view of this it was strongly felt; this clinical threat can be prevented and managed by empowering and participation of all nurses involved in direct patient care.

Based on this, we introduced nurses driven protocol for hypoglycemia in Max hospital; Hypoglycemia Boxes were placed in the wards for emergency cases and more than 1000 nurses were trained over a year for management of hypoglycemia, which is still an on-going process. This has proven to be very successful in managing hypoglycemia by nursing staff. After the implementation of our corrective measure it was observed that adherence to Hospital Hypoglycemia protocol increased from 75% to 99% (figure 1), which resulted in a significant reduction of hypoglycemia events in the hospital, approximately by 40%. Regular training of nurses reduced the reaction time from 6-12 minutes to 2-4 minutes and all this lead to uniformity of hypoglycemia management across wards. A Clear improvement in practice was observed. The service which was largely clinician led is now a nurses led service.

Nurses driven management, reporting, recording and documentation of hypoglycemia through a stream of speciality nurse: Diabetes Nurse Educator can add value and enhance the quality of nursing care delivered to the patients with diabetes. Empowering nurses could enhance clinical excellence in improving patient care.

Figure 1: Graphical representation of yearly incidences of Hypoglycemia

---

Wegener’s Granulomatosis Induced Diffuse Alveolar Hemorrhage with ARDS Treated Successfully by Extracorporeal Membrane Oxygenation

Wegener’s Granulomatosis (WG), renamed “granulomatosis with Polyangiitis” (GPA), is an Anti-Neutrophil Cytoplasmic Autoantibody (ANCA) - associated vasculitis, characterized by multifocal vascular necrotizing inflammation and granulomas. Genetic and microbial factors may play a role in induction and expression of the autoimmune process. GPA is a multi-system disease that can affect any organ/system, having a variable clinical presentation. Nevertheless, it is most frequently manifested as small vessel vasculitis affecting the upper and lower respiratory tract and the kidneys. In ANCA - associated vasculitis, including GPA, alveolar hemorrhage and concomitant glomerulonephritis causing renal insufficiency (Pulmonary-Renal Syndrome) are associated with high mortality.

Although many patients with WG have capillaritis seen on surgical lung biopsy specimens, diffuse alveolar hemorrhage (DAH) is a rare complication that carries an extremely high fatality rate. Support of the respiratory and renal systems is often required in conjunction with aggressive therapy for the underlying vasculitis.

Extra-Corporeal Membrane Oxygenation (ECMO) is an artificial lung support that has been used for acute respiratory distress syndrome (ARDS) with promising results. ECMO was developed as a supportive therapy for severe respiratory failure, and has been shown to be life-saving with respiratory failure. When conventional mechanical ventilation fails, ECMO can
support oxygenation and ventilation, thus enabling the ventilator settings to be markedly reduced. This can help to prevent further lung damage caused by high airway pressures and oxygen toxicity, while allowing more time for aggressive treatment of the underlying pathology. We report on a previously healthy 27-yr-old adult who presented with fulminant pulmonary hemorrhage leading to Hypoxemic Respiratory Failure causing Acute Respiratory Distress Syndrome due to Wegener’s Granulomatosis. He was successfully treated with ECMO and other supportive therapy.

A 27 year male was admitted with respiratory failure and hypotensive shock. Patient was put on veno-venous ECMO because saturation was 60% on FiO2 100% on ventilator. He had bilateral subcutaneous emphysema. He also had multi-organ dysfunctions including kidney and central nervous system. On investigations he was diagnosed with Wegener’s Granulomatosis. The diagnosis was based on clinical, radiologic, serologic and histologic manifestations. His WG was aggressively treated with high-dose cyclophosphamide (300 mg IV daily) and dexamethasone (60 mg IV three times per day). He underwent plasmapheresis for 6 days followed by therapy with IV Ig (400 mg/kg/d) for an additional 5 days. Because the use of ECMO and aggressive immunosuppression interfered with our ability to monitor for signs and symptoms of infection, the patient received empiric therapy with broad-spectrum antibiotics, and daily surveillance cultures were collected. He also underwent hemodialysis followed by continuous venovenous hemofiltration through the ECMO circuit. All modalities of support to sustain life continued until clinical remission could be achieved.

ECMO sustained life and allowed disease control, together with plasmapheresis, cyclophosphamide, corticoids, and renal replacement therapy. The patient was successfully weaned from ECMO, extubated, and discharged home on day 23.

ECMO can be successfully used to manage a fulminant variant of WG with severe pulmonary hemorrhage with ARDS when maximal conventional ventilatory support fails to respond.

Management of Facial Synkinesis

Facial Synkinesis is common sequelae to Idiopathic Facial Nerve Paralysis, also called Bell’s palsy or Facial Palsy. Bell’s Palsy, which is thought to occur due to a viral reactivation which can lead (through unknown mechanisms) to diffuse axon demyelination and degeneration of the seventh cranial nerve, results in a hemifacial paralysis due to non-functionality of the nerve (figure 1)

As the nerve attempts to recover, nerve miswiring results. In patients with severe facial nerve paralysis, facial synkinesis will inevitably develop. Yamamoto et al26 found that synkinesis occurs most frequently in the 24–39th weeks of facial palsy. Celik et al27 noted that synkinesis develops at least 4 months after the onset of facial palsy

The most common symptoms of facial synkinesis include:

- Eye closure with volitional contraction of mouth muscles
- Midfacial movements with volitional eye closure (figure 2)
ABERRANT NERVE REGENERATION

The aberrant nerve regeneration hypothesis is the most widely accepted mechanism for synkinesis. The hypothesis states that, after trauma, axons project from the facial nucleus to incorrect peripheral muscle groups. These aberrant branches can simultaneously innervate different subdivisions of the facial nerve.

For example: compression to the facial nerve causes a lesion and the set of axons that innervates the orbicularis oris (mouth muscle) degenerate. Once the compression has relieved, regeneration of axons from the lesion site begins. This time though, only 50% of the set of axons that innervate the orbicularis oris successfully reinnervate the original site. The other half aberrantly branched off and innervated the orbicularis oculi (eye muscle). Thus, when the patient purses their lips, the ipsilateral eye will squint.

The hypothesis assumes that disorganized regeneration occurs at the site of the lesion. On the contrary, recent research by Choi and Raisman has provided a more thorough understanding of synkinesis through aberrant axonal regeneration. Their study has shown that regenerating axons become disorganized throughout the length of the nerve and not only at the site of the lesion. Previously, many developed treatment strategies (that inevitably failed) were invented based on the original hypothesis by only focussing on the lesion site for improving the organization of regeneration. The new modification to the hypothesis could allow for better success in developing treatments.

NUCLEAR HYPEREXCITABILITY

The basis of this hypothesis is as follows: after a lesion, axonal degeneration (via Wallerian degeneration) occurs. The postsynaptic cell consequently becomes deprived of input and becomes more sensitive to neurotransmitters (e.g. creating additional receptors). Subsequently, residual undamaged axons in the same premise of the lesion can provide a source of neurotransmitter to the deprived postsynaptic cell. Since the post-synaptic cell is hypersensitive, the neurotransmitters that reach it from an axon of another nerve will successfully provide stimulation. This consequentially creates undesired peripheral movement (i.e. synkinesis).

Since synkinesis has been reported in patients within 1–2 months, the nuclear hyper-excitability hypothesis is being supported by more researchers. Furthermore, axonal regeneration is a slow process (~1 mm/day growth) and regeneration at this rate of the facial nerve would roughly take 4–8 months. Since synkinesis is observed much earlier, aberrant regeneration and ephaptic communication fails to explain for this observation thus providing evidence that nuclear hyper-excitability is an important factor in the mechanism of synkinesis development.

Although these three mechanisms have been argued for and against in various ways, it has become more accepted that synkinesis develops through a combination of these mechanisms.

MEASURING SYNKINESIS

Until May 2007, there was no clinical scale to measure synkinesis. A study led by Mehta et al. has validated the use of a newly designed instrument to evaluate facial synkinesis called the Synkinesis Assessment Questionnaire (SAQ) (figure 4). The instrument, consisting of nine questions, was found to be both reliable and valid. In addition, it is simple, easy to administer, and inexpensive. Its analyses can allow for treatment options to be evaluated.

TREATMENT

Facial synkinesis has the benefit of less invasive treatments such as facial retraining, biofeedback, mime therapy, and botox.

Physiotherapy Treatment:

Additionally, a common treatment option for facial palsy is to use electrical stimulation. Unfortunately, this has been shown to be disruptive to normal reinnervation and can promote the development of synkinesis. Physiotherapy
treatment for facial synkinesis includes facial retraining, biofeedback and mime therapy.

**FACIAL RETRAINING**

Facial neuromuscular retraining is a highly specialized field in physiotherapy. Facial retraining therapy builds upon the idea that neurons are constantly in a dynamic state. Facial neuromuscular retraining is a highly specialized field. In other words; there is constant growth and regression of neuronal projections dependent on the stimuli produced.

Before start of exercise, it is very important to educate the patient to get familiar with the facial muscles. Diagram of the facial muscles and its actions are grossly explained to the patient (Figure 5). It is important for the patient to learn to isolate muscle actions as well as coordinate multiple muscle movement. Regaining balanced, symmetrical movements is the key to restoring the face as it was before. To reduce synkinesis, facial retraining teaches the patient techniques for increasing wanted movements while focussing on restricting unwanted movement. If, for example, the mouth moves whenever the eyes blink voluntarily, facial retraining techniques will teach the patient to slowly close the eyes while actively focussing on keeping the mouth muscles still. Facial retraining has shown to be very successful with almost a 60-70% average decrease in synkinesis reported after 7 months.

**BIOFEEDBACK**

Biofeedback therapy for facial synkinesis aims to increase the patient’s awareness of the facial muscle posture and movement. Facial muscles contain few to none intrinsic muscle sensory receptors (used for proprioceptive feedback) and additionally they do not span movable joints and so lack joint receptors (another source for proprioceptive feedback). Thus, biofeedback allows the patient to actively sense the motion of their muscles. The two common forms of biofeedback used are electromyography feedback and mirror feedback. Electromyographic feedback includes visual EMG signals (coming from facial muscle sites displayed to the patient from a computer in the form of waveform traces (figure 6) or auditory signals that indicate strength of muscle contraction. The subsequent role of the patient is to control the movement of undesired muscle during volitional movement by incorporating the information perceived through the EMG. While mirror feedback is a much more basic way of providing the patient feedback on muscle movement, studies have shown that both are very effective options for synkinesis / paresis reduction. Biofeedback is commonly coupled to facial retraining techniques to achieve maximal effectiveness. A study by Nakamura et al has shown that biofeedback works better for prevention of synkinesis as opposed to treatment of synkinesis. Due to the extreme efforts needed to achieve improvements during synkinesis, Nakamura et al. observed that patients will often fail to reach their desired goal because of the difficulty of maintaining motivation during training. The desired course of action is to catch the patient shortly after facial nerve trauma and teach the patient biofeedback techniques. This course of action has been experimentally proven to significantly reduce the development of synkinesis.

**DURATION OF PHYSIOTHERAPY**

The entire course typically lasts from 12 months to 3 years. Videotape and

**MIME THERAPY**

Mime therapy was introduced in the Netherlands in 1980. It was initially designed to treat facial palsy by improving symmetry of the face both at rest and during movement. It was then later observed that people who had post-facial palsy synkinesis also benefited from this therapy. It wasn’t until 2003 that Beurskens and Heymans were able to experimentally conclude that mime therapy was indeed a good treatment choice for synkinesis. Furthermore, later studies by Beurskens et al. have shown that benefits obtained from mime therapy are stable 1 year after therapy. Current mime therapy consists of a combination of procedures designed to promote symmetry of the face at rest and during movement and control synkinesis (Figure 7). The components include: massage, stretching exercises, exercises to coordinate both halves of the face, etc. The overall aim of mime therapy is to develop a conscious connection between the use of facial muscles and emotional expression. So while facial retraining therapy is much more focused on treating slight synkinetic movements, mime therapy aims to increase the overall vigor of the muscles through active exercises, while in the process of doing so, teaching the face to decrease unwanted synkinetic movements.
photographic re evaluations are completed approximately every six months or as significant functional change is noted. Patients actively participate in comparing the initial to subsequent evaluations, identifying new problem areas and establishing new goals. Figure 8) Exercises are progressed as successive short term goals are attained during this relatively slow process. Long term goals may take years to achieve, therefore it is unrealistic to expect this process to occur quickly.\textsuperscript{21, 26}

**BOTOX**

Botox is a new and versatile tool for the treatment for synkinesis. Initially used for reducing hyperkinesis after facial palsy,\textsuperscript{24} botox was later attempted on patients with post-facial palsy synkinesis to reduce unwanted movements (Figure 9).

The effects of botox have shown to be remarkable with synkineitic symptoms disappearing within 2 or 3 days. Due to the short span of botox effects though, patients must come back to the doctor for re-injection approximately every 3 months. More notable though is that in a majority of patients, various synkinetic movements completely disappeared after 2-3 sessions of botox injections (each session administered after ~3 months).\textsuperscript{24} A more specific synkinesis, crocodile tears syndrome (hyperlacrimation upon eating), has been shown to respond exceedingly well to botox injection. Botox is injected directly into the lacrimal gland and has shown to reduce hyperlacrimation within 24–48 hours. The procedure was shown to be simple and safe with very little chance of side-effects (although rarely ptosis can occur due to botulinum toxin diffusion).\textsuperscript{5} Furthermore, reduction in hyper-lacrimation was shown to last longer than the expected 3 months (about 12 months).\textsuperscript{24} Since botox can mimic facial paralysis, an optimized amount of botox has been determined that reduces involuntary synkinesis of the muscle while not affecting muscle tone.\textsuperscript{24}

Patients who are using muscle relaxants such as baclofen, dantrolene or diazepam can continue taking these meds for further assistance with muscle spasms or tightness if needed; they will not interact with Botox.

**Botox can be combined with**

neuromuscular retraining to try to maintain improved motion patterns and appearance without continuing injections. While Botox is preventing inappropriate muscle movements, exercises are performed daily to reinforce correct, isolated movements. The exercises are continued after the Botox wears off to further reinforce the new pattern.

**SURGERY**

Practical surgical procedures used for treating synkinesis are neurolysis and selective myectomy. Neurolysis has been shown to be effective in relieving synkinesis but only temporarily and unfortunately symptoms return much worse than originally.\textsuperscript{17} Selective myectomy, in which a synkinetic muscle is selectively resected, is a much more effective technique that can provide permanent relief and results in a low recurrence rate; unfortunately, it also has many post-operative complications that can accompany including edema, hematoma, and ecchymosis.\textsuperscript{4} Therefore, surgical procedures are very minimally used by doctors and are stored as last-resort options for patients who do not respond well to non-invasive treatments.

**REFERENCES**


Young male 30 years old reported in Emergency with history of road traffic accident. He was in the front seat of car and took the brunt of broken window pane glass on whole face, as he was not wearing seat belt. At the time of admission, he was semi conscious, with multiple injuries on face and eye.

**OCULAR EXAMINATION**

<table>
<thead>
<tr>
<th></th>
<th>Right Eye (Figure 1)</th>
<th>Left Eye (Figure 2 a &amp; b)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Visual Acuity</td>
<td>PL only, PR doubtful</td>
<td>PL, PR accurate, HM close to face</td>
</tr>
<tr>
<td>Eyelids</td>
<td>Edema++, ecchymosis</td>
<td>Edema, Lower lid full thickness laceration</td>
</tr>
<tr>
<td>Globe</td>
<td>Full thickness scleral perforation with uveal + vitreous prolapse</td>
<td>Full thickness scleral perforation with uveal ++ vitreous prolapse</td>
</tr>
<tr>
<td>Cornea</td>
<td>Nasal Perforation, Oedema</td>
<td>Clear</td>
</tr>
<tr>
<td>Ant chamber</td>
<td>Shallow, Hyphaema</td>
<td>Formed / Pupil sluggish reaction</td>
</tr>
<tr>
<td>Lens</td>
<td>No View</td>
<td>Clear</td>
</tr>
<tr>
<td>Fundus</td>
<td>No View</td>
<td>Disc Normal, Macular Edema</td>
</tr>
</tbody>
</table>

**CT SCAN – ORBIT (FIGURE 3)**

Multiple foreign bodies in superficial adenaxa in right eye. Two large foreign bodies (glass) in lateral orbit of left eye.

**PLAN**
- A combined reconstructive plastic and ophthalmic surgery was planned under general anaesthesia.
- Tetanus prophylaxis + topical and systemic antibiotics
- IV steroids for suspected traumatic optic neuropathy in left eye
- Detailed documentation with informed high risk consent
- Ultrasound B Scan of eyes after primary reconstructive surgery to evaluate posterior segment and decide further course of action
- Ocular trauma was done to see the extent of corneoscleral perforation. Superficial vitrectomy + repositioning of choroidal tissue + cornea scleral wound repair were done. Anterior chamber was washed and formed with saline. Multiple tiny glass foreign bodies removed from lids, conjunctiva and tenon's.

**PRE-OPERATIVE**

**Right Eye (Figure 4):** Exploration of the wound was done to see the extent of corneoscleral perforation. Superficial vitrectomy + repositioning of choroidal tissue + cornea scleral wound repair were done. Anterior chamber was washed and formed with saline. Multiple tiny glass foreign bodies removed from lids, conjunctiva and tenon's.
Left Eye (Figure 5 a & b): Removal of glass foreign bodies from lateral orbit followed by superficial vitrectomy + repositioning of choroidal tissue + scleral wound repair was done. Lower lid full thickness repair was done.

Postoperative Ultrasound (Figure 6): After primary repair, patient was undertaken for B Scan Ultrasound to evaluate posterior segment. There was vitreous haemorrhage but no retinal detachment.

Left Eye (Figure 8): The lower lid was well apposed to globe with no ectropion. Scleral wound was also healing well. Posterior segment evaluation was normal.

Right Eye (Figure 9): Phacoemulsification with foldable intraocular lens with capsular tension ring support was done under local anesthesia.

Postoperative - 1 Month; Right Eye (Figure 7): The corneoscleral wound had healed well but there was no fundus glow due to Traumatic Cataract formation by this time. At the site of nasal corneal perforation, there was iris tissue loss with suspected zonular dialysis.

Postoperative-2 months (Figure 10): Right Eye: Vision restored to 6/18p with glasses, Pseudophakia, IOL stable c/o glare due to mid dilated atonic pupil. Resolving vitreous haemorrhage-PVD, opacities / strands, No retinal breaks. Tinted soft contact lens trial given to avoid glare.

DISCUSSION
Severe perforating injuries often have a poor prognosis for both recovery of visual function and salvage of the eye. Initial surgery should be directed towards optimal repair of the perforating wound, correction of damage caused by the injury and prevention of secondary complications. Even severe injuries should undergo atleast one full hearted attempt at repair as they may have a chance to restore some visual function.

Evaluation and surgical repair are best performed under general anesthesia due to advantages of akinesia, anesthesia, and reduced intraocular and orbital pressure.

Few key points of surgical repair are listed as following:

CORNEAL / SCLERAL PERFORATION REPAIR
- Meticulous exploration of the wound to assess the extent of injury.
- Tissue adhesive with bandage contact lens can be used if corneal perforation is very small.
- Prolapsed iris may be preserved and reposited back if the look is viable. In case of old injuries or infection, it should be excised.
- Vitreous incarceration should be relieved by anterior vitrectomy to avoid risk of endophthamitis, chronic inflammation, cystoid macular edema and retinal detachment.
- 10-monofilament is used to repair corneal perforations and the bite should pass through 2/3rd of corneal thickness. Large bites are taken (1.5 to 2mm) from edges so that the edematous edges are well approximated. The knots should be rotated and buried.
- In a corneoscleral perforation, the first sutures placement is at limbus to realign.
- Donor scleral graft should be arranged in OT in case the wound gape is large with loss of tissue
- Scleral perforations are sutured with spatulated needle 8-0 silk or ethibond. Some use 6-0 vicryl also. Scleral sutures should be 75 - 90% deep, with entry and exit at least 1 mm from the wound edge. Start anterior and progress posterior, so the wound is more stable when you need...
to rotate the globe for posterior access.
• Posterior scleral perforations should be handled by posterior segment surgeon as they are mostly accompanied by retinal trauma.
• Avoid ointment at end of surgery as it can get entrapped in repaired lacerated corneal tissue and delay healing.

FULL THICKNESS EYELID LACERATION REPAIR
• If the margins are smooth they can be simple approximated. If the vertical cut is ragged or there is loss of tissue, excise in a pentagonal fashion.
• 6-0 vicryl or silk on spatulated needle is used to reapproximate the gray line to achieve correct approximation of the margin edges. Place two additional 6-0 silk suture anterior and posterior to initial grey line suture. This prevents any notch formation in the eyelid
• Keep the knot away from the eyeball because irritation or potentially ulceration may result if the knot rubs on the conjunctiva or cornea.
• Use two 6-0 vicryl suture bites to reapproximate the tarsal plate and or orbicularis muscle in one or two layer.
• Close the skin with 6-0 silk or nylon

OVERVIEW
• No matter how serious the ocular injuries look at presentation, always give a full hearted attempt for meticulous repair
• CT Scan vs MRI: CT Scan is the choice of radiography in ocular trauma-detailing of bony margins, globe integrity, muscles, optic nerve status, presence and location of foreign bodies, can all be detected on fine 1-2 mm axial and coronal cuts.
• Always explore under GA- advantages of akinesia, anesthesia, reduced intraocular and orbital pressure
• Role of steroids in traumatic optic neuropathy- IV steroids-methyl prednisolone-mega pulse therapy for 3 days- effectiveness is unproven, but may be given in the absence of contra-indications.
• Visual & Psychological rehabilitation – Loss of vision, disfigurement, or opting for prosthesis can all be psychologically traumatic for the patient. Surgeon should not only help in visual rehabilitation but also give positive counseling and encouragement for him to cope up in his life.

NOTE
The patient was not wearing seat belt and hence sustained grievous injuries due to the sudden impact. Let this also be a reminder to all our colleagues that the use of seat belt in car is a must for everybody. Most of the injuries can be prevented by following the safe driving guidelines... The adage ‘An ounce of prevention is worth a pound of cure’ is most apt for ocular trauma.

Watch Out For Stroke

Dr. JD Mukherjee
Director & Head of Unit– Neurology
Max Super Speciality Hospital, Saket

WOMEN HAVE ADDED STROKE RISKS THAT COME WITH PREGNANCY COMPLICATIONS, HORMONE REPLACEMENT THERAPY AND SMOKING

A healthy lifestyle can halve the risk of stroke in women, a new study has found. Particularly after menopause, it is crucial to eat right, exercise, maintain a healthy weight — and watch out for warning signs.
our years ago, while dining
to her father’s house, Namita
Dhur* developed sudden numb-
ness in her right arm and leg.
Soon after, her speech started
to slurr.
"When I got to my dad’s home, I
saw him I was feeling uneasy," says
Dhur, a 37-year-old Delhi
resident. "He said I looked fine,
but I was feeling really unwell."

So Dhur decided to head back home. By
the time she finished her ten-minute drive
to her home, she was completely disoriented.
She quickly got out, hailed a cab and
went to a homeopath in her neighbour-
hood. "The homeopath assessed the situa-
tion immediately and told me to rush to a
neurologist," says Dhur.

At the emergency unit of Max Hospital in
Saket, Dhur was told that she was
among the fortunate 25% of stroke
patients who make it to a hospital within
that critical first hour.

Typically, stroke strikes when either
the blood supply to the brain is blocked
or a blood vessel in the brain ruptures,
causing brain tissue to die. In Dhur’s
case, she had a clot in the blood vessels
of her brain, stopping blood flow.

"Namita was given clot-busting
medicines within an hour of ‘having the
stroke’," says Dr JD Mukherji, director
and head of neurology at Max Hospital.
"She has not experienced any more sym-
toms of strokes since then."

Dhur, who retains a slight limp from
the brain attack, still goes to the hospital
for a follow-up check every three months.
"I am very careful with what I eat. I have
been told to include lots of fruits and
vegetables in my diet. To ensure my body
and brain stay active I go for regular
walks and also spend time solving puz-
zel," she says.

While India-specific data is unavailable,
globally, stroke is the third biggest cause
of death for women, after heart disease
and cancer. In men, it’s the fourth-biggest
cause of death.

In India, say medical experts, post-
menopausal women, especially those
who consume excessive tobacco and
alcohol, are at a higher risk of suffering
a stroke. Women also share the same
risk factors for stroke as men, but have
added risks that come with pregnancy
complications, hormone replacement
therapy and smoking.

According to Dr Mukherji, the combi-
nation of smoking and use of birth
control pills puts women at a 50% higher
risk of stroke. "Add to this unhealthy eat-
ing habits, hypertension, increased levels
of fat in the blood, obesity and diabetes,
and the odds are very heavily stacked," he
adds.

A healthy lifestyle, however, can halve
the risk of stroke in women, according
to a study whose results were published
in the journal Neurology, and in which
31,886 Swedish women were followed for
19 years.

The study recognises five contribu-
tory factors to a healthy lifestyle—a
balanced diet, moderate consumption
of alcohol (7 to 9 drinks per week), no
smoking, physical activity (walking
or biking for 40 minutes a day, vigor-
ous exercise for one hour a week), and
maintaining a healthy weight.

Compared with women who didn’t
pay attention to these five factors,
women who did had a 54% lower risk
of cerebral infarction, a type of stroke
triggered by a blockage in the blood
vessels supplying blood and oxygen to
the brain, which accounts for 90% to 95% of
all strokes.

A healthy lifestyle had no effect on
the risk of haemorrhagic stroke, which
is caused by bleeding in and around the
brain, according to the same study.

By and large, the signs of stroke in
women are similar to those in men—
dropping of the face, sudden numbness or
weakness in one arm, sudden difficulty
speaking, etc.

"In women, however, the symptoms
may be more subtle and diffused because
of smaller blood vessels, which is why
one needs to pay attention to any sudden
changes in movement and behaviour," says
Dr Vipul Gupta, head of interven-
tional neuroradiology at Gurgaon’s
Medanta Hospital.

The American Stroke Association’s latest
guidelines, released on Wednesday, recom-
pend checking blood pressure regularly
and eating a diet high in fruits, vegetables,
whole grains and nuts to lower first time
stroke risk.

"We have a huge opportunity to im-
prove how we prevent new strokes, because
risk factors that can be changed or controlled
— especially high blood pressure — account
for 50% of strokes," James Meschia, lead
study author and chairman of neurology at
The Mayo Clinic in Jacksonville, Florida,
said in the guidelines.

(* Name changed on request)
Funny Bone

Dear stomach, you’re bored, not hungry. So shut up.

What is your favourite exercise?

Chewing

‘If I gain 20 pounds, it will give me the motivation I need to stick to my diet!’

An Alternative Medical Dictionary:

1. Bacteria - Backdoor to cafeteria
2. Bowel - Letter like A E I O or U
3. Cauterize - Made eye contact with her
4. Coma - Punctuation Mark
5. Enema - Not a friend
6. Genital - Non-Jewish
7. Impotent - Distinguished, well known
8. Post Operative - A Letter Carrier
9. Rheumatic - Amorous
10. Tibia - Country in North Africa