THE ROLE OF INTERVENTIONAL TECHNIQUES IN CHRONIC CANCER PAIN MANAGEMENT

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The mortality rate of cancer patients have been at the top for 25 years in Japan. And one of the problems about advanced cancer patients is that about 70% of these patients are to have pain during their survival periods and one half of them are reported to be intractable. And what is worst only the one fifth of these patients have pain relief with proper treatments. So, pain relief is the most important issue for the patients with not only advanced and also with early stage cancer.
Concerning the average clinical course of 100 cancer patients,
It took about 80 days for them to undergo the systemic management of pain and they died in about 60 days. Treatments of pain in these 100 patients gave them the decrease of pain evaluated with a 10 steps visual analogue pain scale, VAS, from 7 in the first appearance to our pain clinic during survaival periods.
WHO started a global cancer pain relief program at 1986. We, of course, use many kinds of drugs for cancer pain relief and also used epidural opioids or other analgesics. We also use a continuous epidural and subcutaneous infusion technique with some PCA devices.
We have treated about one thousand and three hundred cancer patients so far with various procedures. Concerning the classification of the procedures that we applied to the patients, 405 patients were treated with a certain neurolytic nerve block. Those are splanchnic nerve block for 215 patients, subarachnoid phenol block for 155 patients. Trigeminal and / or Gasserian ganglion block for 21 and neuroadenolysis of the pituitary gland 8.
I will speak about neurolytic nerve blocks for cancer pain relief
focusing the next 4 techniques; splanchnic nerve block, subarachniod phenol block, Gasserian ganglion block and trans-distal superior hypogastric plexus block. Summary of the effects of these procedures are as follows; in the patients who underwent splanchnic nerve block, VAS value decreased from 7 to 0.7. In the subarachnoid phenol block group, that was from 7.7 to 1.3 and in the hypogastric plexus block group, that was from 8 to 3.3.
These results tell us that nerve blocking techniques are worth using in the treatment of cancer pain.
I will discuss the evaluation of pain management after neurolytic splanchnic nerve blocks comparing it with patients without the blocks whose pain were treated with the conventional WHO cancer pain relief program in another session in this Congress.

PALLIATIVE CARE IN BANGLADESH

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Palliative care is a positive care when there is no cure.

From the Qur' an
"May you be wrapped in tenderness, you my brother (sister), as if in a cloak." Symptoms, emotions and spiritual questions.
Two phases: one following receiving the bad news of diagnosis, two the dying process. Problems:
Symptoms, pain, distress, depression, fear
Beliefs (Spiritual) around death
Taboos and ignorance
The consequences of death will be different for each person
Palliative care is an approach to care. Patient centred rather than disease centred, accepting death but enhancing life. Concerned with healing rather than curing Ingredients
This is a team approach

Skilled communication, knowing how to communicate with body, actions and words. Convey hope, honesty and openness. Care for the physical (body) and emotions (psychological) and spiritual
Many people care and they are also part of the team: relatives, friends, professionals. Communication is often poor due to fear of what to say, lack of ability and poor training. Differences exist between developed and developing countries.
In a country with free health care most cancers present early and treatment will remove or reduce the tumour mass. Five years survival is high
The elderly die of dementia, and slow growing cancer in old age Developing country
People present late but still reduction in tumour mass can be helpful Cancer may not be the main killer as people do not live so long. Care problems
Patients travel long distances for treatment but return home to a lack of continuity, little information, less shared care and poor support services, and everything has to be paid for when income is reduced due to illness.
Common Clinical problems - Pain nausea, breathlessness, constipation Mood - fear and panic, depression
Dignity - loss of continence, loss of control and changed body image - frustration. "You matter because you are you
You matter to the last moment of your life
And we will do all that we can
Not only to help you die peacefully
But to live until you die."

Care can be provided at home, in day centres and in inpatient beds.

A building is needed for the office organization, education, store and possible drop in for day care
A bad hospice is due to not enough staff and untrained staff giving poor or wrong care. Start by education, assessing the need and costing the needs Money is important as the costs of nursing beds can be high - this is HDU nursing. Myths (all wrong)
People do not know that they are ill and dying
People cannot cope with knowing that they are dying and they will have a nervous break down if told.
Every one has a lot of pain
Death is slow and painful
Drugs are dangerous and will kill people
One big problem is fear - almost worse than pain. Pain is relatively easy to treat but fear is difficult.

Team working is essential.
Together
Everyone
Achieves
More

Every member of the team needs education and support.
What is the patient concerned about?
Effect of bad news : Disbelief, Denial, Depression, Seek explanations and bargain to find a way out, angry.

Solution - sharing of problems and feelings.
Occupy mind and body
Drugs have a place but use carefully, appropriately and stop if not effective. Consider local remedies.

Pain can be considered as: nociceptive, neurogenic, sympathetic, emotional or dysfunctional in origin. Try to determine the cause.
Treatment of symptoms starts by explanation and the relief of fear, then drugs - if drugs are not available consider alternative therapies.
Fear often responds to the presence of another person, avoid the dark and mental activity. Breathlessness. Reduce exercise, sit rather than lie, keep stomach empty and avoid panic. Nausea and vomiting. Causes many - bowel obstruction, constipation, peptic ulcer, drugs and fear.
Every patient is an individual and their care will be different.
The patient knows that we do not have all the answers but they appreciate that we are trying to do our best

A NEW SIMPLE AND MODIFIED DEVICE FOR LOCATION OF THE EPIDURAL SPACE FOR TRAINEE

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Introduction

Despite years of use, controversy still surrounds the appropriate steps to ensure the safe initiation of epidural anaesthesia. The epidural space is entered after the tip of the needle passes through the ligamentum flavum, which up to that point had been only a potential space. Identification of the precise moment when the needle is advanced into this space decreases the likelihood that the needle will puncture the dura1. Methods for identifying this space fall into two broad categories 2 the loss of resistance technique and those depending on the advantage of the sub-atmospheric pressure in the epidural space such as the hanging drop method (Soresi 1932) and the balloon method (Macintosh 1950)

The risks associated with epidural blockade include unintentional subdural, subarachnoid and intravenous drug injection. Subdural injection is the least common misadventure. entry into this space, may not be recognized because CSF will not flow freely either spontaneously or with aspiration. Injection of local anaesthetic solution into this small space produces a high level of sensory and motor anaesthesia resembling epidural anaesthesia in it's speed of onset although the duration is usually shorter and may lead to either an unexpected high epidural anaesthesia or a failure of anaesthesia[1-3].
Intrathecal injection is a more common complication, but intravascular injection is the most significant hazard of epidural blockade for two reasons first, vessel entry is sometimes difficult to diagnose, second, bolus injection of large volumes of local anaesthetics can rapidly produce toxic drug concentrations, seizures, respiratory arrest, and with bupivacaine especially, cardiac arrest and death can follow[4].
The purpose of this work was to evaluate the use of El mulla's balloon device and technique (modified Macintosh's technique) in comparison with the traditional syringe technique (loss of resistance technique) for location of the epidural space by beginners in epidural analgesia.
Methods: Clinical trials were performed on 90 patients for evaluation of this modified device and technique (El Mulla's device and technique which is a modification of Macintosh's balloon device) and the remaining 60 cases were performed by the beginners in epidural analgesia using this new device and technique. Thirty cases were performed by experienced anaesthetists and the remaining 60 cases were performed by the beginners in epidural analgesia using this new device and technique. The former and the later cases were compared with 30 and 60 cases which were performed with the traditional syringe technique both by the experts and the beginners respectively.

Dr. El Mulla's Invention (Technique)

The following is a brief description of El Mulla's Device for the safe location of the epidural space (the prototype for this device is the balloon of Foly's cathter) : "The invention is based on detecting the slight vacuum that exists inside the epidural space. This is detected by a small inflated balloon, which collapses the moment the needle reaches the epidural space." The device consists of the following 2 main parts: 1. Solid rubber tube this has an inflatable balloon at one end and a one-way valve at the other. 2. 3-way connector has a built-in 3-way valve. The openings of the connector fit the epidural needle, the solid rubber tube and the inflating syringe respectively. In practice, the device is used as follows: Following the preparation and disinfection of the patient's back, the epidural needle is inserted and advanced some 2-3 cms. Using an ordinary medical syringe and the appropriate settings of the 3-way valve, the rubber balloon is inflated with approximately 5 cm3 of air. The rubber tube with the inflated balloon at one end is then inserted into the end of the epidural needle and the 3-way valve is turned into a position where the inflated balloon is in direct communication with the needle cavity. At this stage, the anesthetist continues to slowly advance the epidural needle into the patient's back, until the rubber balloon suddenly deflates, indicating the presence of the epidural space.In order to double-check whether the needle tip is accurately within this space, the 3-way valve is rotated to allow direct communication between the needle cavity and the atmosphere. In this position, the exit of biological fluids can be observed (either blood or cerebrospinal fluid). The absence of any fluid usually confirms the precise location of the tip of the needle within the epidural space, indicating that it is safe to carry out the injection procedure.

The clinical results showed that the failure rate of the new technique done by the beginners was 6.67% compared with 33.33% of the traditional technique. While the procedure sequalae differed significantly, ($P<0.05$) where headache, possible subdural injection and inadvertent dual puncture were encountered among 26.67%, 20% and 6.67%, respectively for the traditional technique compared with none for the new technique. Also the duration ,the number of trials and the amount of air used for identification by the beginners with the traditional technique were significantly greater ($P<0.05$) than those with new technique. The beginners observed the easiness, simplicity and safety of the new device and technique.

Discussion

Although the traditional loss of resistance technique to air remains the choice of many experienced anaesthetists. One cause of failed epidural anaesthesia is false loss of resistance. In some young adults the spinal ligaments are quite, soft and resistance to injection is not as distinct as the practitioner has become used to. The practitioners may believe the epidural space has been entered when the needle is in fact within the interspinous ligaments. On the other hand an occasional patient may have some cystic degeneration within the substance of the ligament and entry into this area will be incorrectly perceived a loss of resistance[1].

The present study described a simple, easy, and safe method for identification and location of the epidural space for use by the inexperienced anaesthetists in epidural analgesia, till they can easily sense the puncture of the ligamentum flavum. In loss of resistance technique five cases have been failed, and the explanation for that failure was mainly attributed to lack of experience, although other factors could be consider e.g. subdural injection. In balloon method only one case has been failed.

Unintentional dural perforation after epidural block results in headache due to CSF leakage[5]. The use of air for loss of resistance testing was associated with the incidence of post-meningeal puncture headache (PMPH), which might be attributable to subarachonoid air injection and CSF leakage. The dura can be unintentionally performed by a needle tip and if the air in the syringe is introduced into the intrathecal space during the block procedure without obvious CSF backflow, air may stimulate CNS structures and produce headache. Sumihisa A and his colleagues[6] concluded tat the use of air for loss of resistance testing during epidural block was associated with a higher incidence of PMPH. In Sumihia's study[5] intrathecal air had been demonstrated by brain CT examination.

The present study demonstrated four cases in loss of resistance group with headache that was of rapid onset and short duration (2-10 min). That headache could be explained by either 1- increased pressure in the epidural space due the excessive volume of air used by the beginners for correct location of the epidural space (>5 cc air), as two cases developed nape and shoulder pain in accompany with headache, or 2- CSF leakage caused by the tip of the needle and/or potentially, to subarachonoid injection of air used as a part of loss of resistance testing which results in intrathecal air.

The last explanation agreed with Sumihia's study[6]. However, another possibility for intrathecal air, which had been demonstrated by Ellis and his colleagues[7] was that valveless venous plexuses of Bateson has been introduced without obvious blood leakage. So, air injected into the epidural veins may tack up to the brain[7]. This was not the case in this study as no intravascular injection was detected.

In the present study the described balloon technique limited the volume of air used as a part of the balloon technique (=5 cc air), thus no cases developed neither headache nor nape or shoulde pain as in loss of resistance group. However, pre-existing headaches were excluded in the studied groups.

Subdural placement of an epidural catheter has been confirmed on four occasions[8-11] The possibility of a subdural injection has been discussed as a cause of failed spinal and demonstrated a potential space between the dura and the arachnoid[3]. On the other hand, several case reports have described a massive epidural anaesthesia following injection of small volumes of local anaesthetic into the presumed epidural space. These findings were attributed to accidental injection into the subdural space[3].

Lee A, and his colleagues[12] has been demonstrated radiographically a case of subdural catheterisation, presented with a massive sensory loss, weak motor block, hypotention and Horner's syndrome. The present three cases in this study (loss of resistance technique) corresponds with Lee's case, although they were not documented radiographically, but most probably due to subdural injection. As the three cases have been presented with massive sensory loss to D4, weak motor loss (Bromage I), hypotension and vomiting but no Horner's Syndrome. No cases with such observations have been demonstrated in balloon technique) although failed cases in both studied groups might be explained on the basis of subdural injection.

The patients of both studied groups did not demonstrate complications such as inadvertent dural perforation (except one case in loss of resistance group) or intravascular injection by the inexperienced anaesthetists which might be due to the strict supervision of an experienced anaesthetist.

The described balloon technique in the present study has been incorporated readily available cheep materials and monitoring devices, limited the use of excessive volume of air (no nape and shoulder pains or headache) and avoided the further advancement of the tip of the epidural needle and the possibility of either subdural injection or unrecognized dural perforation especially for the inexperienced anaesthetists.

In Conclusion, the indentification of the epidural space by the prevously described balloon technique should be widely practiced and taught by the trainee and other pain practitioners for its easiness and safety for correct location of the epidural space until they have the experience of sensing the puncture of the ligamaentum flavum.

The inventor has recently received the Inventors' Net award "Best Invention 1999/2000" This device is patented in Sydney-Australia and also the scientific research academy-Cairo-Egypt.This topic was represented in:- 12th world congress of Anaesthesiologists-Montreal-Canada.- First African Congress of Pain-Alexandria-Egypt.- 11th world congress of Pain-Sydney Australia.- Correspondence: Prof Dr Ahmed El Molla aelmulla@yahoo.co.ukFor more detailswww.elmulla.cjb.net

References

1. Edward M, Maged SK. Spinal,epidural and caudal blocks:In clinical anaesthesiology(1st ed.) USA, chapter 16,1992;1:211-220.
2. Wildsmith JAW, Armitage EN. Lumbar and thoracic epidural anaesthesia. In: Princibles practice of regional anaesthesia(5th ed.) USA,Churchill Livingstone,chapter 8,1987;1:81-101.
3. Maaten JM, Kleef JW. Failure of anaesthesia after accidental subdural catheter placement.Acta Anaesthesiol Scand 1992;36:707-709.

4. De Jong RH,Ronfeld RA, De Rosa RA. Cardiovascular effects of convulsant and superaconvulsant doses of amide local anaesthestics. Anesh Analg 1982;61:3
5. Crawford JS. The prevention of headache consequent upon dural puncture.Br J Anaesth 1972; 44:598-600.
6. Sumihisa A, Kiihciro T, Tomohiro Y etc. Headache after attempted epidural block (the role of intrathecal air).Anaesthesiology 1998;88:76-81.
7. Ellis H, Felfman. Anatomy for anaesthetsts,4 th ed. Blackwell scientific publications(1983)133.
8. Boys JE, Norman PF. Accidental subdural analgesia. A case report,possible clinical implications and relevance to 'massive extradurals".Br J Anaesth 1975;47:1111-3.
9. Manchanda VN, Murad SHN, Shilyansky G, etc. unusual clinicl course of accidental subdural local anaesthetic injection. Anaesthesia analgesia 1983;62:1124-6.
10. Smith GB, Barton FL,Watt JH. Extesive spread of local anaesthetic solution following subdural insertion of an epidural catheter during labour. Anaesthesia 1984;39:335-8.
11. Hartrick CT, Pai U, etc Subdural migration of an epidural catheter. Anaesth Analg 1985;64:175-8.
12. Lee A, Dodd KW.Accidental subdural catheterization. Anaesthesia 1986;41:847-9.

NEW DRUGS IN THE TREATMENT OF ACUTE PAIN

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Consistent delivery of first-class postoperative pain control is still a major challenge. Many studies suggest that postoperative and post-traumatic pain is often under treated as a result of inadequate education of health care providers and fear of causing opioid addiction. A recent survey shows that nearly 80% of patients still experience pain after surgery and of these patients; 86% had moderate, severe or extreme pain.

There is increasing evidence to suggest that long term neurobiological changes occur much more quickly than previously anticipated with the potential of progression from an acute persistent phase to a chronic phase. In some patients the mechanical allodynia and hyperalgesia that are normal in the first days or weeks after surgery, do not regress but persist beyond the usual course of an acute injury. Post traumatic neuropathic pain is a major contributor to persistent pain affecting roots, nerves, the plexus and central structures.

Therefore one needs to differentiate "acute neuropathic pain" as a separate entity of pain syndromes and the new concepts of managing pain are to treat adequately and early to prevent acute becoming chronic. The six major barriers to adequate pain care include regulatory and legal concerns, system barriers, deficits in knowledge and education, fear of side effects, assessment challenges and most importantly myths that addiction is a common result of treating with opioids. Twenty five percent of patients referred to chronic pain centres have persistent post surgical pain. The risk factors for developing chronic post-surgical pain can be tagged by the use of blue flags, yellow flags and red flags. Blue flags indicate the perioperative risk factors to note, yellow flags, the psychological and environmental factors and red flags, the postoperative physical disorders that need treatment.

Amongst the remedies which are available to relieve pain none is so universal and as efficacious as opium. Amongst the new drugs available are oxycodone, and fentanyl. Various forms of these preparations are available including slow release transdermal preparations, as well as fast acting lozenges.

The COXII inhibitors are also effective in managing acute pain although there has been much publicity about the serious cardiovascular side effect profile of these drugs. Additional warnings and contraindications have been added for all COXII's in a press release by the European Medicines Agency in June 2005.The contraindications for the use of COXII's include established IHD, cerebrovascular disease/stroke and peripheral vascular disease. Caution should be taken when using these in patients

with risk factors for heart disease and the lowest possible dose should be used for the shortest possible time.
Gabapentin may be a new drug for postoperative pain. During the elucidation of the mechanism of action of gabapentin in animals, it became apparent that it was effective in some acute pain models. There is now considerable interest in the potential use of gabapentin for postoperative pain relief. In a recent review, seven studies of reasonable quality were identified. Significant reductions in postoperative analgesic requirements 24h after surgery were found in six studies. Oral gabapentin as an adjunct to epidural analgesia has shown to decrease pain and analgesic consumption. A possible mechanism for gabapentin-mediated analgesia is the modulation of glutamate receptors and its calcium channel blocking activity. Pregabalin has recently obtained a license in many countries for treating neuropathic pain and there is already some evidence that it may have efficacy in acute pain similar to that of gabapentin.
There is no published evidence for treating acute neuropathic pain and all the evidence has been extrapolated from neuropathic pain studies. In the acute phase it is suggested that an NMDA antagonist or a local anaesthetic be used together with an adjuvant orally. There is some evidence for using lignocaine infusions as well as the use of prophylactic antidepressants for post herpetic neuralgia.
Intrathecal ziconotide binds to the calcium channel and is used in some centres and we may In the future see cannabinoids being used for pain control. A more specific task is the need to optimise perioperative pain management with improvement of multimodal pharmacological analgesic regimes and integration of acute pain services into perioperative rehabilitation.

INTRATHECAL ANALGESIA FOR MANAGEMENT OF CANCER PAIN

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Systemic pharmacologic treatment has become the mainstay of cancer pain treatment. If optically administered, systemic analgesics could control pain in most patients with cancer pain. However, even when the basic principles for the analgesics are fully used, some patients experience intractable side effects or insufficient pain relief with systemic opioids. Switching from systemic administration to neuraxial route could provide excellent analgesic effects and lessen side effect. When opioids are administer directly into the subarachnoid space, only a fraction of the systemic dose is required because there are no anatomic barriers to be crossed and reuptake is slow. Although not all side effects of neuraxial opioids are dose-related, in many instances this drastic reduction in dosage translates into reduced side effects. One of the primary indications for a trial with neuraxial opioids is a good response to opioids coupled with intractable side effects. Among the adverse opioid effects reduced by neuraxial administration are sedation and constipation. Those that are increased include pruritus, urinary retention and edema.
The following issues would be discussed in this session.
1. Indication of neuraxial analgesia with long-term use of catheter
The neuraxial infusions are used in patients who either cannot tolerate systemic opioids because of intolerable side effects, such as sedation, constipation, nausea and vomiting, or those who require large systemic opioid doses (> 4g/day morphine systemic equivalents)[1]. The good application for neuraxial technique also appears to be for patients with neuropathic pain, visceral pain from intestinal distention, incident pain on movement, and cutaneous ulceration when using opioid with local anesthetics[2].
2. Intrathecal versus epidural routes
Epidural route is often chosen as the initial procedure because of easier trial of a neuraxial analgesia and lack of post dural puncture headache. However, epidural route is characterized by a high frequency of fibrosis around the catheter, resulting in loss of efficacy and catheter obstruction or dislocation[3]. Long-term complications are lower using intrathecal route, which save opioid dose, expense and efforts for changing syringe drivers or pomp cassettes. Intrathecal route permits a rational utilization of home care resources.
3. Intrathecal opioids; equipotent analgesic doses and an update of intrathecal morphine

Intrathecal opioids including morphine, hydromorphone, sufentanil, fentanyl, meperidine and methadone are used for pain management4). The recommended conversion ratios between different opioids and routes would be shown on the slide. Recent intrathecal delivery devices with program also carry out the new problem of long term intrathecal infusion of high dose morphine. High dose morphine 15mg/day would cause inflammatory mass in the catheter tip[4]. Local histamine release with high concentration of morphine might be suspicious origin.

4. Intrathecal adjuvants; local anesthetics and other agents

Combining intrathecal bupivacaine with morphine shows potent synergic effect, particularly for neuropathic and breakthrough motion pain. Bupivacaine less than 30mg/day significantly improves sleep, gait, daily activity without adverse effects (urinary retention, motor weakness, and paresthesia.)

5. Introduction of Polyanalgesic Consensus Conference

Update of clinical guidelines for the use of intraspinal drug infusion in pain management; Polyanalgesic Consensus Conference 2003 would be reviewed. Morphine and hydromorphone are on the first line on the algorism because of the safety on neurotoxicity, efficacy, and cost for long-term use.

6. Neuraxial techniques for cancer pain

(1) Starting doses and titration and (2) Spinal delivery systems in our hospital would be showed in the slide.

References

1. Fittzgibbon DR: Cancer pain:Management. (Ed.by Loeser JD, Butler SH, Chapman R,et al.: Bonica's Manegement of Pain. 3rd ed.) Lippincott Williams & Wilkins Philadelphia, 2001 pp659-703
2. Mercadante S: Neuraxial techniques for cancer pain: an opinion about unresolved therapeutic dilemmas. Reg Anesth Pain Med 24:74-83, 1999
3. Crul BJ, Delhaas EM: Technical complications during long-term subarachnoid or epidural administration of morphine in terminally ill cancer patients: a review of 140 cases. Reg Anesth. 16:209-213,1991
4. Yaksh TL, Horais KA, Tozier NA, et al.: Chronically infused intrathecal morphine in dogs. Anesthesiology. 99:174-187,2003
5. Hassenbusch SJ, Portenoy RK, Cousins M, et al. Polyanalgesic Consensus Conference 2003: an update on the management of pain by intraspinal drug delivery-- report of an expert panel. J Pain Symptom Manage. 2004 Jun;27(6) :540-63. Review.

CANCER PAIN - BARRIERS TO CARE

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In considering the barriers I would also wish to consider ways of overcoming these barriers.

Palliative care is the positive care when there is no cure. It is not the absence of care. There has always been some form of care for the dying, but it has not always emphasised the positive with the patient at the centre.

The change in emphasis is needed due to the advances in medical care and the expectation that medicine can cure everything, but we all die. More is now known about the effects of death due to war and other tragedies on the survivors. In some societies death is not part of life or life itself is not respected.

Pain is a bio-psycho-social experience. In most cases physical pain can be relieved by simple means but many doctors only use the tools of their specialty so anaesthptists inject, oncologist give drugs or radiotherapy, surgeons operate. But is any one looking at the whole patient?

The barriers to good care include:

Ignorance

Death is a taboo subject surrounded by myths and fear. So the consequences of death are not talked about openly.

Beliefs

Every person is an individual and there is a lot to be taught to that individual about the dying process. That individuals' experience is unique to them. Lack of honesty

Doctors say, "There is nothing that can be done." When they mean there is nothing that they know to do.

Good Communication is everything. We learn more from how people are acting than from what they say.

Lack of resources and lack of co-ordination

These leads t o fragmented care, no follow up, absence of treatment. Not enough money, but it does not matter how much money you have you always want more. Lack of resourcefulness

Look for solutions. Only copy what is good and relevant from other schemes. Each pattern of care is peculiar to the individual and the situation. We are always learning. The patient knows that we are not perfect, they appreciate that we are trying to do out best.

Rules and regulations

Differences between countries and cultures may affect drug prescribing and the procedures around death.

Where to start.

Most people want to die at home but in Bangladesh there is no home care. Nurses are not yet seen as independent professionals.

Day centers provide respite; relief of loneliness; advice; meet with other people in a similar situation and so do not feel isolated; learn to live each day again.

Hospice for those who cannot be looked after at home. BUT there are bad hospices where there is not enough trained staff.

At first there is a need for a base to work from, and to develop a team of educated, skilled carers. There is a positive role for volunteers but they must be vetted, trained and managed. A volunteer is a member of the team doing a job, but not being paid for that job.

Problems with starting with a hospice. There is no skilled team in place who know what to do. A hospice costs a lot of money as the amount of nursing care is high dependency. Only a small number of people will be cared for in a hospice. Ignorance and myths: Drugs kill - only if given wrongly. Most people know they are ill and dying. People do not go mad or commit suicide with this knowledge. The right sort of talking helps.

Fear is almost more of a problem than pain, aggravated by ignorance. Drugs are not very effect in relieving fear.

Looking for solutions, not more problems

Lack of honesty. We have our own fear of what to say.

Cost: What is the biggest local need? Then devise local solutions to feeding, im drugs, and volunteers. Look at traditional and local remedies.

Predictable problems

Try to do too much too quickly, make mistakes BUT learn from them. Work as a team. Everyone needs respect and support. Look after the whole family Unpredictable. Some one will oppose the service, misinterpret what is being offered. Success takes time.

Deal with the concerns of the patient as they arise: physical, mental, socials, spiritual. At the end the patient, family and the caring team accept the powerlessness of themselves.

Other professionals can be supportive and antagonistic.

There is a continuous process of education

Drugs. Need to develop agreed protocols to avoid waste and side effects. Fear is better treated by avoiding loneliness and talking through. Need to educations on effect and side effects.

Similarly need education about symptoms like Pain - what it is: nociceptive, neurogenic, sympathetic, emotional and dysfunctional, and how to manage it. Surgery, radiotherapy, chemotherapy and other treatments should be relevant to relieve symptoms.

Try to develop care pathways but they should be flexible to accommodate the differences between patients and their families.

Reading

Organisation and development of pain clinics and palliative care in developing countries. European Journal of Anaesthesia Kumar A. 2004;21(3):169-172

Developing palliative care services for terminally ill patients in Saudi Arabia. Alan Gray et al, www.kfshrc.edu.sa.annals/154/94207/94207.htm1

Introducing Palliative Care by R. Twycross
Oxford Handbook of Palliative care
Recognising spiritual need by R. Stanwick

HEADACHE BANGLADESH PERSPECTIVE

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Headache is such a common complaint and can occur for so many different reasons that its proper evaluation may be difficult. Although underlying structural lesions are not present in vast majority of eases. However it is nevertheless important to bear this possibility in mind. About one third of patients with brain tumor, present with primary complaint of headache. It has been estimated that one person in three suffers from severe headache at some stage of life. More than 13000 tons of Aspirin are consumed annually worldwide. A major part of it is taken for the relief of headache. Most people with a mild recurrent or isolated headache do not consult physician and therefore the true incidence is unknown.
This is true for any other country as in Bangladesh. The Department of Neurology of Dhaka Medical College is running weekly "Headache Clinic" where till now 7500 patients have been registered since 1996 with preset diagnostic and classifying criteria as well as treatment and follow-up schedule maintained by a group of Neurologists, Psychologists, Psychiatrists and Physical Medicine specialists. Among the patients 91% have primary headache of which three fourth is tension type headache (TTH), and rest is migraine, 9% having secondary headache of local cause of which less than 1% presented with intra cranial space occupying lesions (ICSOL). Majority of the patients (40%) belongs to 18 to 29 years of age, of which 66% are female. Housewives constitute 50% of them. Mostly prescribed drugs for TTH and migraine are tricyclic anti depressant and non selective beta blocker. Amitriptyline had good response in 60% of cases of TTH. Nortriptyline was found to less responsive than Amitryptiline with 494/u having good responses in tension type headache. Among migraine patients good responses was achieved with Amitryptiline in 53% cases. Beta blocker alone had lower response than amitryptiline in patients with migraine and 43% having good responses but 40% did not respond at all. Combination of Amitryptiline and beta blocker had excellent response in 69% of patients having migraine. Neura-imaging, mostly in the form of CT scanning was done in 13% of cases with a clinical suspicion of intra cranial space occupying lesion (ICSOL).

CANCER PAIN - BARRIERS TO CARE

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In considering the barriers I would also wish to consider ways of overcoming these barriers.
Palliative care is the positive care when there is no cure. It is not the absence of care. There has always been some form of care for the dying, but it has not always emphasised the positive with the patient at the centre.
The change in emphasis is needed due to the advances in medical care and the expectation that medicine can cure everything, but we all die. More is now known about the effects of death due to war and other tragedies on the survivors. In some societies death is not part of life or life itself is not respected.
Pain is a bio-psycho-social experience. In most cases physical pain can be relieved by simple means but many doctors only use the tools of their specialty so anaesthptists inject, oncologist give drugs or radiotherapy, surgeons operate. But is any one looking at the whole patient?
The barriers to good care include:
Ignorance

Death is a taboo subject surrounded by myths and fear. So the consequences of death are not talked about openly.

Beliefs

Every person is an individual and there is a lot to be taught to that individual about the dying process. That individuals' experience is unique to them. Lack of honesty

Doctors say, "There is nothing that can be done." When they mean there is nothing that they know to do.

Good Communication is everything. We learn more from how people are acting than from what they say.

Lack of resources and lack of co-ordination

These leads t o fragmented care, no follow up, absence of treatment. Not enough money, but it does not matter how much money you have you always want more. Lack of resourcefulness

Look for solutions. Only copy what is good and relevant from other schemes. Each pattern of care is peculiar to the individual and the situation. We are always learning. The patient knows that we are not perfect, they appreciate that we are trying to do out best.

Rules and regulations

Differences between countries and cultures may affect drug prescribing and the procedures around death.

Where to start.

Most people want to die at home but in Bangladesh there is no home care. Nurses are not yet seen as independent professionals.

Day centers provide respite; relief of loneliness; advice; meet with other people in a similar situation and so do not feel isolated; learn to live each day again.

Hospice for those who cannot be looked after at home. BUT there are bad hospices where there is not enough trained staff.

At first there is a need for a base to work from, and to develop a team of educated, skilled carers. There is a positive role for volunteers but they must be vetted, trained and managed. A volunteer is a member of the team doing a job, but not being paid for that job.

Problems with starting with a hospice. There is no skilled team in place who know what to do. A hospice costs a lot of money as the amount of nursing care is high dependency. Only a small number of people will be cared for in a hospice. Ignorance and myths: Drugs kill - only if given wrongly. Most people know they are ill and dying. People do not go mad or commit suicide with this knowledge. The right sort of talking helps.

Fear is almost more of a problem than pain, aggravated by ignorance. Drugs are not very effect in relieving fear.

Looking for solutions, not more problems

Lack of honesty. We have our own fear of what to say.

Cost: What is the biggest local need? Then devise local solutions to feeding, im drugs, and volunteers. Look at traditional and local remedies.

Predictable problems

Try to do too much too quickly, make mistakes BUT learn from them. Work as a team. Everyone needs respect and support. Look after the whole family Unpredictable. Some one will oppose the service, misinterpret what is being offered. Success takes time.

Deal with the concerns of the patient as they arise: physical, mental, socials, spiritual. At the end the patient, family and the caring team accept the powerlessness of themselves.

Other professionals can be supportive and antagonistic.

There is a continuous process of education

Drugs. Need to develop agreed protocols to avoid waste and side effects. Fear is better treated by avoiding loneliness and talking through. Need to educations on effect and side effects.

Similarly need education about symptoms like Pain - what it is: nociceptive, neurogenic, sympathetic, emotional and dysfunctional, and how to manage it. Surgery, radiotherapy, chemotherapy and other treatments should be relevant to relieve symptoms.

Try to develop care pathways but they should be flexible to accommodate the differences between patients and their families.

Reading

Organisation and development of pain clinics and palliative care in developing countries. European Journal of Anaesthesia Kumar A. 2004;21(3):169-172
Developing palliative care services for terminally ill patients in Saudi Arabia. Alan Gray et al, www.kfshrc.edu.sa.annals/154/94207/94207.htm1
Introducing Palliative Care by R. Twycross
Oxford Handbook of Palliative care
Recognising spiritual need by R. Stanwick

HEADACHE BANGLADESH PERSPECTIVE

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Headache is such a common complaint and can occur for so many different reasons that its proper evaluation may be difficult. Although underlying structural lesions are not present in vast majority of eases. However it is nevertheless important to bear this possibility in mind. About one third of patients with brain tumor, present with primary complaint of headache. It has been estimated that one person in three suffers from severe headache at some stage of life. More than 13000 tons of Aspirin are consumed annually worldwide. A major part of it is taken for the relief of headache. Most people with a mild recurrent or isolated headache do not consult physician and therefore the true incidence is unknown.

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THE IMPORTANCE OF CENTRAL SENSITISATION IN CHRONIC PAIN: IMPLICATIONS FOR TREATMENT

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Introduction

In the last years, pain research has enormously increased the current knowledge on the central mechanisms involved in pain modulation. Overwhelming evidence from animal studies have demonstrated the occurrence of profound changes in the central nervous system after peripheral injury that are responsible for enhanced neuronal excitability and enhanced pain perception[1]. Studies in healthy volunteers have shown that experimentally induced peripheral injury or inflammation determines exaggerated pain response, which results from increased excitability of the central nervous system[2]. The logical development of this basic research is its transposition into clinical setting, whereby the presence and clinical relevance of hypersensitivity states in patients with pain is investigated. The involvement of central hyperexcitability in pain after whiplash injury is appealing, given the limited knowledge on the causes of the pain complaints in this syndrome.

Mechanisms of Post-injury Central Hypersensitivity

This section presents the pre-clinical evidence on the presence and mechanisms of central hyperexcitability after peripheral injury. It is divided into three parts, according to the sites at which changes in the excitability of the nociceptive system after peripheral injury are observed: the periphery, the spinal cord and the brain.

Peripheral Sensitization

Tissue injury due to trauma or surgery leads to an inflammatory response with release of potassium ions, substance P, bradykinin, prostaglandins and other substances (often termed the "inflammatory or sensitizing soup")[4]. These substances may induce a sensitization of peripheral receptors with changes in the response characteristics of primary afferent fibers[4]. They may also activate normally inactive or "silent" nociceptors[6]. Furthermore, the inflammatory response induces a gene expression in the dorsal root ganglion resulting in an increased synthesis of peripheral receptors, which contributes to the increased sensitivity of the nociceptor[7]. Long lasting nociceptive stimulation may lead to a modification in the peripheral fibers: A■-fibers may start synthesizing receptors that are normally found only in C-fibers, thereby simulating a phenotype shift, with the A■-fiber adopting C-fiber characteristics[8]. These sensitizing events mediate primary hyperalgesia[9], in which a reduced threshold for eliciting pain and enhanced pain to supra-threshold stimuli within the injured area can be recorded. Peripheral sensitization ultimately results in an increased nociceptive input to the spinal cord.

Spinal Cord Plasticity

Prolonged afferent nociceptive input may induce a reversible increase in the excitability of central sensory neurons[1]. The important role of the N-methyl D-aspartate (NMDA) receptor in the development of spinal cord hyperexcitability has been shown in early animal experiments[10,11]. Activation of NMDA receptors seems to be linked to expression of cyclooxygenase-2 (COX-2) in the spinal cord, and there is evidence for COX-2 inhibition of central sensitization in the animal[12]. Importantly, COX-2 expression is not confined to the neural structures connected to the site of inflammation, but involves the whole spinal cord and the supraspinal centers[13]. This phenomenon seems to be mediated by humoral factors, rather than by a neural transmission of the peripheral input into the spinal cord[13]. It may be responsible, at least in part, for a generalized hypersensitivity to peripheral stimulation, such the one evoked after stimulation of tissues that are at distance from the site of injury.

An expansion of the receptive fields (the cutaneous area which is innervated by a single spinal neuron) of individual dorsal horn neurons is also documented[14]: afferent input from areas adjacent to the normal receptive field may be able to depolarize the hyperexcitable dorsal horn neuron. As a result, a peripheral stimulus activates a higher number of dorsal horn neurons and hyperalgesia may also be evoked in areas outside the injured region. The glial cells, which were earlier regarded as purely supportive, have become

implicated in exaggerated pain states[15]. They may be activated by peripheral injury and can contribute to central hyperexcitability.

Additional profound structural changes include destruction of inhibitory interneurons and aberrant excitatory connections[1]. Destruction of inhibitory interneurons that has been observed after nerve injury contributes to hyperexcitability[16]. Interestingly, this phenomenon is prevented by NMDA-antagonists[16]. After nerve injury, A■-fibers that normally terminate in the deep dorsal horn may sprout to establish functional synaptic contacts in superficial dorsal horn layers where nociceptive C-fibers terminate[17]. This is one of the possible explanations for the induction of pain sensations after stimulation of A■-fibers by e.g. touch.

Both the peripheral sensitization and the hyperexcitability of dorsal horn neurons will reduce the threshold for eliciting Ad- and C-fiber pain. While peripheral sensitization is responsible for primary hyperalgesia (i.e. hyperalgesia recorded within the injured area), secondary hyperalgesia (i.e. hyperalgesia recorded in the surrounding uninjured tissue) is the result of central hyperexcitability[17]. A■-fiber-transmitted mechanical stimuli, which do not produce pain under normal conditions, may activate the hyperexcitable dorsal horn neurons, ultimately resulting in pain sensation (allodynia)[1,18].

Assessing Central Hyperexcitability in Chronic pain

In patients, direct measurements at spinal cord neurons can not be made. Therefore, it is impossible to provide direct evidence for neuronal hyperexcitability. However, hypersensitivity can be investigated indirectly by quantitative sensory tests. Typically, a sensory stimulus is applied at a peripheral tissue. Then the stimulus intensity is increased gradually until the subject perceives the stimulus as painful. The intensity at which the stimulus perception turns to pain is defined as pain detection threshold. The intensity at which the pain is perceived as intolerable is defined as pain tolerance threshold. Alternatively, a standardized painful stimulus is applied and the intensity of the evoked pain is recorded. Using these methods, hypersensitivity is detected when sensory stimulation evokes pain at stimulus intensities that do not induce pain in normal subjects (lower pain threshold) or when a standardized painful stimulus evokes stronger pain in patients than in normal subjects. Other methods to explore the sensory system are available, but a detailed description is beyond the scope of this paper. The interested reader can find more information in a review[19].

In the above examples, however, the reader would probably wonder whether hypersensitivity to sensory stimulation is the result of peripheral or central mechanisms. In fact, enhanced pain response is also observed after peripheral sensitization, in which the sensitizing factor (typically, trauma or inflammation) decreases the threshold to activate the nociceptors. However, peripheral sensitization is limited to the site of injury or inflammation. At this level, quantitative sensory tests can not distinguish peripheral from central hypersensitivity. Conversely, whenever pain hypersensitivity is observed after sensory stimulation of healthy areas, its cause must be a hyperexcitability of the central nervous system (central hypersensitivity). Indeed, there is no evidence that peripheral mechanisms could account for a higher pain sensitivity at healthy tissues. Therefore, it is generally accepted that sensory stimulation of healthy tissues explores the excitability state of the central nervous system.

Sheather-Reid & Cohen examined pain detection and tolerance thresholds after cutaneous electrical stimulation of the neck in whiplash patients and healthy volunteers[20]. They found lower pain thresholds in patients than in healthy subjects. The absence of tissue damage at the site of testing suggested a central sensitization of nociceptive pathways as the cause of the pain hypersensitivity. In particular, the findings were interpreted as secondary hyperalgesia. As mentioned in the section "Mechanisms of post-injury sensitization", secondary hyperalgesia is defined as enhanced perception of painful stimuli at healthy areas that surround damaged tissues, and has been shown to be determined by central sensitization[21].

Koelback Johansen et al investigated muscle pain sensibility after intramuscular injection of hypertonic saline[22]. This method typically induces pain lasting few minutes both at the area of injection (local pain) and at areas at distance from the site of injection (referred pain). Additionally, pressure pain thresholds were assessed. Whiplash patients displayed higher pain scores, longer duration of pain and larger areas of local and referred pain after intramuscular injection of hypertonic saline, compared with healthy controls. They also had lower pressure pain thresholds. These differences were found at both neck and leg. Interestingly, several patients reported pain spreading to the whole leg and on the contralateral side, which was not the case in healthy subjects. These data suggest that pain hypersensitivity is not limited to the injured and surrounding areas (primary and secondary hyperalgesia), but may be generalized to the whole central nervous system.

More recently we measured pain thresholds to electrical and heat stimulation, applied at both skin and muscles of both neck and lower limb, in whiplash patients and healthy controls[23]. Measurements were made before and after injection of a local anesthetic into the painful muscles. Patients displayed lower pain thresholds with both cutaneous and muscular electrical stimulation, applied at both the neck and the lower limb. This confirms the previous findings of generalized central hypersensitivity. Interestingly, the pain thresholds after heat stimulation were similar in the two groups.

Summary

There is clear evidence that tissue trauma leads to a reversible increase in the excitability of the central nervous system. Potentially irreversible changes have been documented. These alterations may be responsible, at least in part, for persisting pain after injury. Central hyperexcitability can be assessed in chronic pain patients.

References

1. Woolf CJ, Salter MW. Neuronal plasticity: increasing the gain in pain. Science 2000;288:1765-9.

2. LaMotte RH, Shain CN, Simone DA, Tsai EF. Neurogenic hyperalgesia: psychophysical studies of underlying mechanisms. J Neurophysiol 1991;66:190-211.

3. Spitzer WO, Skovron ML, Salmi LR. Scientific monograph of the Quebec Task Force on whiplash-associated disorders: Redefining "whiplash" and its management. Spine 1995;20 (Suppl.):1S-73S.

4. Rang HP, Bevan S, Dray A. Chemical activation of nociceptive peripheral neurones. Br Med Bull 1991;47:534-8.

5. Treede R-D, Meyer RA, Raja SN, Campbell JN. Peripheral and central mechanisms of cutaneous hyperalgesia. Prog Neurobiol 1992;38:397-421.

6. Schmidt R, Schmelz M, Forster C, Ringkamp M, et al. Novel classes of responsive and unresponsive C nociceptors in human skin. J Neurosci 1995;15:333-41.

7. Michael GJ, Priestley JV. Differential expression of the mRNA for the vanilloid receptor subtype 1 in cells of the adult rat dorsal root and nodose ganglia and its downregulation by axotomy. J Neurosci 1999;19:1844-54.

8. Neumann S, Doubell TP, Leslie T, Woolf CJ. Inflammatory pain hypersensitivity mediated by phenotypic switch in myelinated primary sensory neurons. Nature 1996;384:360-4.

9. LaMotte RH, Thalhammer JG, Torebj÷rk HE, Robinson CJ. Peripheral neural mechanisms of cutaneous hyperalgesia following mild injury by heat. J Neurosci 1982;2:765-81.

10. Woolf CJ, Thompson SWN. The induction and maintenance of central sensitation is dependent on N-methyl-D-aspartic acid receptor activation: Implications for the treatment of post-injury pain hypersensitivity states. Pain 1991;44:293-9.

11. Dickenson AH, Sullivan AF. Evidence for a role of the NMDA receptor in the frequency dependent potentiation of deep rat dorsal horn nociceptive neurones following C fibre stimulation. Neuropharmacology 1987;26:1235-8.

12. McCrory CR, Lindahl SG. Cyclooxygenase inhibition for postoperative analgesia. Anesth Analg 2002;95:169-76.

13. Samad TA, Moore KA, Sapirstein A, Billet S, et al. Interleukin-1beta-mediated induction of Cox-2 in the CNS contributes to inflammatory pain hypersensitivity. Nature 2001;410:471-5.

14. McMahon SB, Wall PD. Receptive fields of rat lamina 1 projection cells move to incorporate a nearby region of injury. Pain 1984;19:235-47.

15. Watkins LR, Milligan ED, Maier SF. Spinal cord glia: new players in pain. Pain 2001;93:201-5.

16. Azkue JJ, Zimmermann M, Hsieh TF, Herdegen T. Peripheral nerve insult induces NMDA receptor-mediated, delayed degeneration in spinal neurons. Eur J Neurosci 1998;10:2204-6.

17. Mannion RJ, Woolf CJ. Pain mechanisms and management: a central perspective. Clin J Pain 2000;16:S144-56.

18. Coderre TJ, Katz J, Vaccarino AL, Melzack R. Contribution of central neuroplasticity to pathological pain: review of clinical and experimental evidence. Pain 1993;52:259-85.

19. Curatolo M, Petersen-Felix S, Arendt-Nielsen L. Sensory assessment of regional analgesia in humans. A review of methods and applications. Anesthesiology 2000;93:1517-30.

20. Sheather Reid RB, Cohen ML. Psychophysical evidence for a neuropathic component of chronic neck pain. Pain 1998;75:341-7.

21. Torebj÷rk HE, Lundberg LE, LaMotte RH. Central changes in processing of mechanoreceptive input in capsaicin-induced secondary hyperalgesia in humans. J Physiol (London) 1992;448:765-80.
22. Koelbaek Johansen M, Graven-Nielsen T, Schou Olesen A, Arendt-Nielsen L. Generalised muscular hyperalgesia in chronic whiplash syndrome. Pain 1999;83:229-34.
23. Curatolo M, Petersen-Felix S, Arendt-Nielsen L, Giani C, et al. Central hypersensitivity in chronic pain after whiplash injury. Clin J Pain 2001;17:306-15.

NON CONVENTIONAL ANALGESICS

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Clinicians treating pain sometimes come across strange and difficult types of chronic pain. Pain initiated or caused by a primary lesion or dysfunction in the nervous system, (otherwise known as neuropathic pain) very often belongs to such category of difficult pains. Neuropathic pain is common in cancer and can happen as a result of the disease or treatment to the disease. The common types of neuropathic pain encountered in cancer are Cranial Nerves Neuralgia, Post Herpetic Neuralgia, Cervical,Brachial and Lumbosacral Plexopathies, Paraneoplastic peripheral neuropathy, Phantom pain etc. All these are characterized by pain in areas of altered sensation. Typical clinical features of neuropathic pain include shooting / lancinating pain, burning pain, paraesthesia / dysaesthesia, numbness and allodynia (pain produced by a normally non-painful stimulus).

Neuropathic pains some times respond partially or fully to opioid drugs. But most of these pains will need drugs other than traditional analgesics. These medicines which are used as analgesics in certain situations are often called 'adjuvant analgesics'. This adjective arise from the fact that in the context of cancer pain, they are used as adjuvants to the analgesics in WHO analgesic ladder. But this is actually a misnomer as most of these pharmacological agents are being used on their own to treat neuropathic pain.

Some of these non conventional analgesics used to treat neuropathic pain are discussed below.

Anti Depressants:

The most widely used drugs in this category belongs to oral tricyclic antidepressants. The commonly used drugs in the group are amitriptyline(25-75mg HS) imipramine (25-75mg HS) desimipramine and nortriptyline . This analgesic effect is independent of their antidepressant effect. TCAs have an effect of 5 hydroxytriptamine release, the noradrenergic pathways and a sodium channel blocking effect , the later effect being shared by the local anaesthetic and anticonvulsant groups. Unfortunately, the the analgesic effect of the TCAs is tempered by their very poor side effect profiles. Drowsiness and dry mouth are the predominant side-effects. The other antidepressants like Selective Serotonin Reuptake Inhibitors (SSRIs) like Citalopram with less side effects are also effective, but not to the same extent as TCAs. The Noradrenalin Serotonin Reuptake Inhibitor Venlafaxine probably combine good analgesic potency with a better side effect profile. But it is more expensive than TCAs. Recent studies have shown that doxepin, a TCA good analgesic effects in certain in neuropathic pains on topical application. The topically applied of doxepin does not have many side effects of the systemic drug.

Anticonvulsants:

Antiepileptic drugs have been found to have therapeutic efficacy in neuropathic pain states. They are believed to depress abnormal neuronal discharges and raise the threshold for the propagation of the abnormal pain impulse. Carbamazepine and Phenytoin were the drugs of choice for treating trigeminal

neuralgia for more than four decades. A large number of newer, better-tolerated and safer antiepileptic drugs useful in the management of neuropathic pain are now available. Carbamazepine, Phenytoin, Gabapentin and Lamotrigine have been evaluated in double blind trials.

Due to propensity for sedation, the initial dosing is done at a low dose administered at bedtime and increased slowly up to a therapeutic level over a few weeks. Anti epileptics are usually more effective at higher doses. A practical option will be to titrate the dose upwards until side effects are encountered and then back down a small amount.
Carbamazepine and Sodium Valproate are used in a dose of 200 - 1200mg per day. Gabapentin is usually started in a 100 - 300 mg at night and slowly increased to 1200 mg per day. The Gabapentin analogue Pregabalin is believed to be a better drug than Gabapentin. A large number of newer agents like Felbamate, Zonisamide, Tiagabine, Vigabatrin are now in the market. But an algorithm of use has not yet been developed for these newer agents. The anti epileptic of choice for different painful states has also not yet been determined

Membrane Stabilisers:

Anti arrythmics have been found to be effective in certain neuropathic pains. For many years intravenous infusions of local anaesthetics have been used in the management of both acute and chronic pain. Despite much anecdotal evidence of analgesia with IV lignocaine there are few double blind studies to verify this effect. It does seem that a short term infusion (eg; 6mg/kg in 24 hours) may give relief in some patients for a sustained period (weeks to months). Lignocaine is also used topically, particularly in the newly approved patch form, with modest effects in the treatment of postherpetic neuralgia. There is also some evidence to suggest that the oral flecainide and mexiletene may also have an analgesic effect in neuropathic pain.

NMDA receptor antagonists:

Though Amantadine and Dextromethorphan have been occasionally used to treat neuropathic pain, the most popular drug in the NMDA receptor antagonist group has been the intravenous anaesthetic agent Ketamine. This drug has an advantage in being effective by subcutaneous, oral and sublingual routes. Ketamine 0.5mg / kg three to four times a day shows good analgesic effect with tolerable side effect profile. Still lower doses are effective by sublingual route. Bitter taste is a problem while using Ketamine by these routes.

Capsaicin

Capsaicin, an active constituent of the chili pepper, when repeatedly applied topically in appropriate concentration causes reversible depletion of the neurotransmitter substance P (SP) from the sensory nerve endings and hence pain relief, which may take several weeks to occur. Anecdotally, topical application of capsaicin has been shown to reduce the pain of a variety of conditions, including post herpetic neuralgia, painful diabetic neuropathy, and post mastectomy syndrome. The major side effect is that of burning discomfort which can be reduced by addition of glyceryl trinitrate (GTN). Pain relief with capsaicin cream in clinical trials has been inconsistent. Two trials of its effects on diabetic neuropathy were either negative or only slightly effective. Two trials in postherpetic neuralgia had conflicting results. A trial in polyneuropathy had negative results.

Baclofen

Baclofen, has shown an analgesic effect in trigeminal neuralgia, probably due to reduction in the release of the excitatory neurotransmitters glutamate and aspartate. Like gabapentin, baclofen is also structurally similar to the inhibitory amino acid gamma amino butyric acid (GABA) and yet seems to have a mechanism of action that differs to that of GABA.

Clonidine

The alpha adrenoreceptor agonist clonidine has an effect on the descending noradrenergic pathways when administered by the epidural or intrathecal route. It produces analgesia if used by these routes, but not on oral administration.

CANCER PAIN

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Cancer is significantly prevalent in Bangladesh. No reliable statistical data is available regarding the cancer pain. However pain is reported at diagnosis by about 20-25% of cancer patients and is experienced by at least 75% as those with advanced disease.

The need for cancer pain treatment was identified as a major health problem in the 1980s.
Choice of therapy is directed by the severity, type and cause of pain. The severity of pain determines the strength of analgesic required, and type & cause of pain will influence the choice of adjuvant. Type, cause and severity can only be determined from a thorough patient assessment. Effective use of WHO 3 step ladder, therefore, depends on accurate regular pain assessment.
General therapeutic strategy of cancer and the basic success of the cancer pain management are discussed.

CURRENT CONCEPTS IN DIAGNOSIS AND MANAGEMENT OF CRPS

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Complex Regional Pain Syndrome (CRPS) is a multi-symptom, multi-system syndrome that affects one or more extremities but virtually may affect any part of the body. CRPS often follows injury to affected limb. CRPS type I also known as Reflex Sympathetic Dystrophy (RSD) follows minor injuries or fracture of a limb. CRPS type II also known as Causalgia develops after injury to a major peripheral nerve. The best way to describe CRPS is in terms of an injury to a nerve or soft tissue that does not follow normal healing path. The development of CRPS does not depend on the magnitude of the injury. In fact, the injury is so slight that the patient may not recall it. For reasons not understood, the sympathetic nervous system seems to assume an abnormal function after injury.

Some other names given to this syndrome are -
Sudeck's Atrophy, Post Traumatic Dystrophy, Shoulder Hand Syndrome, Reflex Neurovascular Dystrophy
Incidence - Exact prevalence of CRPS is not known but data from several studies suggest it is more frequent than commonly believed. Both sexes are affected but women are affected more, specially in paediatric age group.
Etiology - A number of precipitating factors have been associated with CRPS: -

- Trauma (often minor) ranks as the leading provocative event.
- Ischemic heart disease and myocardial infarction.
- Cervical spine or spinal cord disorders.
- Cerebral lesions.
- Infections.
- Surgery.
- Repetitive motion disorder or cumulative trauma, causing conditions such as carpal tunnel.

However, in some patients a definite precipitating event can not be identified.
Pathogenesis - Current research suggests that the mechanism by which an injury triggers CRPS is unclear. Original injury initiates a pain impulse carried by sensory nerves to the CNS. The pain impulse in

turn triggers an impulse in the sympathetic nervous system which returns to the original site of injury. The sympathetic impulse triggers the inflammatory response causing the vessels to spasm leading to swelling and increased pain. The pain triggers another response, establishing in a vicious cycle of pain and swelling. Resulting condition with burning extremity pain, red mottling of the skin.

Duration - The duration of CRPS varies, in mild cases it may last for weeks followed by remission; in many cases the pain continues for years and in some cases, indefinitely. Some patients experience periods of remission and exacerbation. Periods of remission may last for weeks, months, or years.

Diagnosis - There is no single laboratory test to diagnose CRPS. The diagnosis of CRPS is a clinical one, predominantly based on signs and symptoms. Medical history is very important. Objective findings if present, supports the diagnosis. Early diagnosis is important because if not treated early, the patient may have significant psycho social, psychiatric problems so also dependency on narcotics and may be completely incapacitated by the disease.

The diagnosis can be made in the following context : -

A history of trauma to the affected area associated with pain that is disproportionate to the inciting event plus evidence at some time for one or more of the following :

- Abnormal function of the sympathetic nervous system, e.g. abnormal changes in skin blood flow, sweating or goose flesh.
- Swelling
- Movement disorder
- Changes in tissue growth (dystrophy and atrophy).

Clinical features -

1. **Pain**: - Severe, constant, burning and / or deep aching pain and mobility problem out of proportion to those expected from the initial injury is the hall mark of CRPS. Allodynia, hyperpathia, paroxysmal dysesthesia, lancinating pains and presence of myofascial trigger points are other features of CRPS.

2. **Skin Changes**: - Skin may appear shiny (dystrophy-atrophy), dry or scaly. Hair may initially grow coarse and then thin. Nails in the affected extremity may be more brittle, grow faster and then slower. Vasomotor, sudomotor and pilomoter changes occur. The changes can be trigger by room temperature specially cold environment.

3. **Swelling:** - pitting or hard (brawny) edema is usually diffuse and localized to the painful and tender region.

4. **Movement disorder:** - Difficulty in moving the affected part due to pain and swelling results in stiffness and disuse atrophy. Tremor and involuntary severe jerking of extremities may be present. Psychological stress may exacerbate these symptoms. Sudden muscle cramps and dystonia can be extremely disabling.

5. **Spreading symptoms:** - The initial localized symptoms may spread to trunk, face and other extremities. The spread may be of "continuity type", "mirror-image type" or "independent type".

Laboratory Diagnostic Aids -

(A) Evaluation of sensory dysfunction: -

- Quantitative sensory testing (QST) has been found to be useful to evaluate the sensory dysfunction in CRPS.

- Magnetoencephalography (MEG) can demonstrate shrinkage of primary sensory cortex ie. Post central gyrus for the CRPS affected hand.

(B) Assessment of vasomotor dysfunction : -

- Measurement of skin perfusion can be done by noninvasive laser doppler device.

- Bilateral differences in skin temperature measured by infra red thermometry and infra red thermal imaging (tele thermography).
- Positron emission tomography (PET) and single photon emission tomography (SPECT) provide a fixed image of neural and circulatory process.
- Edema in patient with dystrophy can be measured by hand volumeter device (99% accuracy).

- Goniometer is a necessary tool to access active or passive range of motion (ROM) of joints.
- Grip strength of upper limb can be measured by dynamometer. Overall hand function can be assessed by the DASH questionnaire.
- Measurement of pain intensity by VAS.

(C) Sympathetic function test. : -

- Peripheral vasoconstrictor reflex assessed by laser Doppler flowmetry or indirectly by thermography.
- Sudomotor function test : sweat output can be quantify by evoporative measurement with a hygrometer. The resting sweat output (RSO) as well as sweating induced by raised body temperature (thermoregulatory sweat test - TST) can be recorded. Quantitative sudomotor axon reflex testing (QSART) by peripheral stimulation is measured.
- Sympathetic skin response recordings reflect autonomic and inflammatory disturbances in CRPS. SSR is more important than NCV and SEPs.
- NCV testing is recommended to exclude peripheral nerve lesion in CRPS. Distinct abnormality exceeding 20 % indicate peripheral nerve lesion i.e. Carpaltunnel syndrome or CRPS - II.
- Somatosensory evoked potential (SSEP) is helpful in patients with CNS dysfunction i.e hemisensory deficit or dystonia or patients with CRPS - II having proximal nerve lesions.

(D) Assessment of inflammatory parameters : -

- Usual systematic inflammatory parameters e.g. CRP, ESR, Leucocytes in serum are not elevated in CRPS rather neuro inflammatory mediators (substance P, bradykinin, and calcitonin gene related peptide - CGRP) are elevated.

(E) Imaging methods : -

- X-Ray : - X-rays may show wasting of bones with patchy osteoporosis.
- Three phase radio bones scanning - TPBS seems to be useful diagnostic tool in non trauma patients.
- CT scan and MRI studies - All of the these tests may be normal in CRPS. They may be helpful in exclusion of differential diagnosis.

Diagnostic Sympathetic Blocks - may help in diagnosing CRPS.

Treatment - The single most important modality for treating the patient with CRPS is education. The informed consent process should be the focus of education. The physician defines the potential benefits, risks, alternatives (and costs). From the start, the therapeutic goals must be defined and accepted by the patient.

- Education About Therapeutic Goals
- Encourage Normal Use of the Limb (Physical therapy in some cases).
- Minimize Pain.
- Determine the Contribution of the Sympathetic Nervous System to the Patient's Pain.

The cornerstone in the treatment of CRPS is normal use of the affected part as much as possible. Therefore, all modalities of therapy e.g. drugs, nerve blocks, TENS, acupuncture, physical therapy, etc. are employed to facilitate movement of the affected region of the body.

The physicians should establish a written treatment protocol designed to rehabilitate the patients in the shortest possible time. Initiate the safest, simplest and most cost effective therapies fast. The treatment modalities cover five areas of care - (1) Procedures e.g. nerve blocks, (2) Medications, (3) Physical / occupational therapy, (4) Psychosocial issues, (5) New laboratory tests.

Medications: - are generally prescribed according to the characteristics of pain -

- Constant pain without inflammation - agents acting on CNS by atypical mechanism (e.g. Tramadol).

- Constant Pain with inflammation - NSAIDs (eg. Aspirin, ibuprofen, naproxen, indomethacin etc.).
- For spontaneous (paroxysmal) jabs and lancinating pain - Anticonvulsants (e.g. carbamazepine, gabapentin etc.).
- Constant pain or spontaneous (paroxysmal) jabs and sleep disturbances - Antidepressants (e.g. amitriptyline, doxepin, nortriptyline, trazodone etc.), Oral lidocaine (mexilitine).
- Wide spread severe and refractory pain - Oral Opioids (morphine, codeine, methadone etc.) and Opioids patch (Fentanyl).
- For sympathetically maintained pain (SMP) - Clonidine patch, oral clonidine.
- For muscles cramps (spasm and dystonia) - Clonazepam, Baclofen and Tizanidine.
- For localized pain related to nerve injury - Capsaicin cream applied locally.
- Other drugs useful in early CRPS - Calcitonin, bisphosphonates, calcium channel blockers and anti oxidants.

Physical and occupational therapy: - Patients educated to use the affected the body part through activities of daily living. Lower extremity CRPS patients are taught weight bearing vs. non-weight bearing exercises. Hydrotherapy in a heated pool is necessary for myofascial pain and spasm. Massage and moist heat application relieves severe muscle cramps. TENS therapy is very useful in CRPS pain. Pool therapy can be very effective for improving mobility. Acupuncture has been found helpful by many workers.

Sympathetic Blocks: - There are three reasons to consider sympathetic blockade in management of CRPS - (1) To provide permanent cure or partial relieve. (2) To gain diagnostic information about what is causing pain. (3) Prognostic information about potential merits of other treatments (rehabilitation).

Sympathetic blocks should be performed by experienced pain specialist. The procedure can be performed with or without IV sedation should be done with vital signs monitoring and under fluoroscopic guidance. A good sympathetic block should increase the temperature of the extremity without producing increased numbness or weakness.

- For upper extremity the sympathetic block is stellate ganglia block (SGB).
- For lower extremity the sympathetic block is lumbar sympathetic block (LSB).

Other Sympathetic blocks: **-**

(a) Alpha adrenergic antagonist, phentolamine, given I.V. has been used as diagnostic test for SMP. This can be used as therapeutic option in situations where sympathetic block is not possible or when multiple extremities are involved.

(b) Epidural blocks are less specific in blocking sympathetic nervous system but may be useful in refractory cases.

(c) I.V. injections of sympathetic blocking agents (guanethidine, bretylium, clonidine, lidocaine, droperidol, reserpine and ketorolac used alone or in combination) into an extremity and limiting spread by applying tourniquet. This is a good option where SGB or LSB are contra indicated (anticoagulation therapy).

Sympathectomy : - If there is significant decrease in pain following sympathetic block, the patient is said to have sympathetically maintained pain (SMP) and they are considered for a sympathectomy - a relatively invasive procedure.

Percutaneious sympathectomy can be done with chemicals (Phenol, Alcohol) in awake patient. Surgical sympathectomy under general anaesthesia can also be done in these patients. Patients must be informed about the pros and cons of each approach.

Recently laporoscopic sympathectomy has been introduced in to clinical practice. Nondestructive Radio frequency lesions of the sympathetic chain can be done by precutaneious approach or through laporoscope. The role of pulsed RF to treat SMP lacks sufficient clinical trial.

Implanted therapies : - Advanced interventional pain medicine techniques applicable to the treatment of CRPS are -

(1) Spinal cord stimulation (SCS),
(2) Peripheral nerve stimulation (PNS),
(3) Intrathecal drug delivery (ITDD).

Indications for interventional pain therapies (SCS, PNS, ITDD) -

(a) Failure to progress in overall rehabilitation when functional restoration is limited by pain, motor dysfunction and vasomotor instability.

(b) Inadequate or partial pain relief from conservative care e.g. physical therapy, oral medication, psychotherapy, regional nerve blocks or larger doses of narcotics.

SCS relieving the pain and improving blood flow to the extremities in CRPS patients have been studied by many workers. Neuro-stimulation modulates efferent impulses from autonomic nervous system to produced vasodilation in the dermatomes that are stimulated. Patients with persistent pain or motor dysfunction or limited response to epidural infusion are considered candidates for SCS. The electrode is implanted percutaneously under local anaesthesia with sedation. The procedure is done under fluoroscopy guidance.

An ITDD system should be considered only after SCS has been attempted and failed to produce relief. Surgical implantation is performed in lateral position with sedation and local anaesthesia. The catheter is advanced to dermatomal distribution of pain site. Morphine and baclofen are the only FDA-approved drugs for i.t. administration. In carefully selected patients, the implantable programmable pump is cost-effective over the long term (after 28 months) compared to conservative management, despite high initial costs.

Psychotherapy - like all chronic pain conditions, CRPS is a complex biopsychosocial disorder, for which successful treatment must target concurrently the medical, psychological, and social components. A vicious cycle in which pain provokes disuse and emotional arousal, both of which in turn further exacerbate the pain, could contribute to maintenance of CRPS. Psychological / behavioral treatments may therefore play an important role in CRPS management by targeting learned disuse and stress / distress to help break vicious cycles. The various psychological interventions include (1) relaxation training, (2) biofeedback, (3) cognitive and behavioral therapy. The family has to be educated in this aspects.

EPIDURAL STEROIDS : CONTROVERSIES

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Lievre and associates used hydrocortisone injected epidurally via the first lumbar route for relief of low back pain in 1957. Many papers have been published regarding the treatment of epidural steroid injection in patients with low back pain. There had been a controversy as to the efficacy and safety of epidural steroid injection for low back pain because there were few prospective, randomized, controlled trial in these studies. However, epidural steroid injection is currently considered to have beneficial effects for low back pain.

[Indication]

If the conservative therapies such as bet rest or controlled physical activity, medication (anti-inflammatory drugs, muscle relaxants), traction, physical modalities (massage, hot packs, ultrasound, TENS, acupuncture), local injection or nerve blocks with local anesthetics is have failed, epidural steroid injection is considered.

Epidural steroid injection has been performed in patients with low back pain, radiculopathy, lumbar disc herniation, and lumbar spinal stenosis. McLain et al. have recently assessed whether epidural steroid injection is effective in the treatment of symptoms due to compression of the nerve root in the lumbar spine in patients received either epidural steroid injection or intramuscular injection of a local anesthetic and steroid. They demonstrated that there was a significant reduction in pain early on in those having an epidural steroid injection but no difference in the long term between the two groups. Delport et al. examined patient satisfaction, relief of pain, frequency of injections, change of function, and subsequent surgical rate in patients who received epidural steroid injection for the diagnosis of lumbar spinal stenosis. They reported that 32% of them had more than 2 months of pain relief, 39% had less than 2 months of pain relief, and 29% had no pain relief from the injection. Therefore, 20% of them subsequently had surgery.

[Mechanism of action]

Epidural steroid injection reduces inflammation on the nerve root and its surrounding connective tissue. Corticosteroids also exert a membrane-stabilizing effect on injured nerve segments, reducing ectopic discharge from the affected nerve root. It reduces conduction in nociceptive C fibers.

[Technique]

A single-shot or continuous epidural technique is almost same. Proper disposable equipment and attention to asepsis is important in epidural steroid injection to avoid serious complications. The patient is placed in the lateral decubitus position with the knees drawn up to the stomach, and the skin is prepared with an antiseptic solution. The skin and deeper tissues are anesthetized. The disposable single-shot needle or Tuohy needle used for continuous epidural block is inserted at a spinal interspace corresponding as closely as possible to the dermatome at the center of the area of analgesia required. When the needle is introduced through the skin puncture, subcutaneous tissue, supraspinous or interspinous ligament, a 5-ml syringe filled with saline is attached to the needle. It is advanced with firm, but gentle pressure on the plunger using a loss-of-resistance technique. When the needle pierces the ligamentum flavum there is sudden loss of resistance, the saline entering the epidural space. The needle must not be allowed to advance further. After aspiration is tried to detect CSF or blood, a test injection of 3 ml of local anesthetics is then made. After confirming no sensory or motor loss, methylprednisolone 40 or 80 mg with saline or local anesthetics is injected into the epidural space through the needle or catheter in Asians.

[Complications]

 infection, meningitis, epidural abscess, arachnoiditis, paraplegia, lipomatosis, Cushing's syndrome, steroid myopathy.

RADIO FREQUENCY COAGULATION- AN UPDATE

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Introduction

The use of direct current experimentally was done as early as 1870 and was introduced in the clinical practice in 1944. The radiofrequency technique was first used for generating neural lesions by Aranow and Cosman. In contrast to DC generators radiofrequency generators use continuous high frequency waves of about 1 MHz.

The radiofrequency scores over DC generators in following way:-

1- Radiofrequency lesions are generated by ionic means hence they are more predictable.
2- The tissue temperature and the extent of lesions are controllable. The modern generators have automatic temperature control that prevents overheating and boiling of the tissues. The design of active electrode have improved as they have low thermal coefficients, which lead to faster warming of electrode and thus more accurate depiction of the tissue temperature.
3- Electrical stimulation can be used to locate the nerve and also prevent unwanted nerve damage.
4- Tissue resistance (impedance) can be measured. Low tissue impedance may affect the size and characteristics of the lesion generated can be measured.

Physical Principle and Equipment

The basic principle of RF coagulation, which may be performed in any part of body is the passage of high frequency current from an electrode placed in the body through surrounding tissues, resulting in heating and destroying tissue around the electrode. The human body acting as a conductive electrolytic medium closes the electrical circuit and an electrical current is flowing through tissue with an electrical force and electromagnetic field has been created between the electrodes.

The circuit consists of an active electrode, which delivers the current; a method of measuring tissue temperature (Thermistor or thermocouple); a radiofrequency generator; and a passive electrode with a large surface area. The heat generated is a fraction of the amount of per unit area that flow in the region of the electrode. The active electrode does not generate heat but is heated as a result of local tissue warming. The current flows from the active to the passive electrode. However because of the much greater surface area of the passive electrode, the current density is less. Therefore, heating and tissue damage do not occur at passive electrode.

Heating of an active electrode is an important safety feature of this system, because of tissue damage is related to the temperature generated. The newer electrode have low thermal coefficient that means the electrode absorbs heat well and heats rapidly leading to a faster response and improved safety of the system. As excessive heating may causes diffuse and permanent tissue damage.

Most of the electrodes available in various sizes and lengths and are reusable and are with disposable needles. The exposed tip of the needle has various lengths for ex. An 18 gauge reusable electrode has 2mm exposed tip suitable for trigeminal nerve whereas 22 gauge electrode with 4mm tip is appropriate for lumbar facet blocks.

Lesion Characteristic

It is critical to control lesion size. The size and consistency of the lesion are governed by following factors.

A- Temperature generated- The local tissue reaction is directly proportional to the temperature i.e. at higher temperatures, the local tissue reaction is greater

B- Rate of thermal equilibrium- If there is more rapid equilibrium between tissues, the lesion is more uniform. If there is slow and incomplete equilibrium then the lesion is erratic.

C- Electrode size and configuration- Larger electrode generate larger lesions. For example an 18 gauge electrode generates a radius of 2.2mm where as a 22 gauge electrode generates a radius of only 1.9.

D- Local tissue characteristics- Lesion in tissue in contact with tissues of low electrical resistance such as blood and CSF may be reduced or irregular in size and shape. Blood may also act as a heat sink, removing heat from the area and there by limiting the tissue size and lesion size.

Advantages Of Radio Frequency Lesioning

1- It has edge over other neurodestructive procedures.
2- Relative safe, accurate, and provides long term pain relief
3- Neuroma formation is minimal.
4- No radiculopathy, minimum morbidity.
5- If done carefully tissue charring, boiling can be avoided.

Technique

These procedures are done in either in operating room or procedure room under strict aseptic precautions along with monitoring of vital signs and stand by resuscitation facility. The investigations

done are bleeding and clotting time. It is mandatory to have i.v. line and a shot of antibiotic is given. The patient requires minimal sedation as one requires maximum cooperation while performing of the procedure. All the equipment and in particular cables, thermister, size and length of needles, appropriate size probes should be checked.

The cannulla is placed in close proximity to the desired nerve under fluoroscopic control. The stylet is removed from the cannula and replaced with thermocouple probe. Then in order to locate the nerve low voltage stimulation at frequency 50-100 Hz, strongest possible sensory stimulation at lowest possible voltage. If the placement of cannula is correct then nerve is stimulated with 0.5mA or 0.25 voltage for standard 500ohms tissue impedance. Cannula should be within 3mm vicinity of the nerve for adequate lessoning. To ensure correct placement of cannula low frequency stimulation of 2Hz is given and twitching is noted. After the confirmation the lessoning is done with desired temperature of 90degree CG for 60-120 seconds.

Indications for Radiofrequency Lesioning

1- Gasserian ganglion or one of its divisions such as Ophthalmic, Maxillary, Mandibular.
2- Stellate ganglion lessoning for CRPS of upper extremity.
3- Cervical, Thoracic, lumbar facet denervation.
4- Median branch lessoning.
5- Dorsal root ganglion lessoning.
6- Lumbar sympathectomy for peripheral vascular diseases, CRPS, phantom limb.
7- Sacroiliac joint denervation.
8- Intervertebral disc neuroplasty.

Pulsed Radiofrequency

Sluijter (1997) presented the technique of pulsed RF, where only short bursts of RF are applied to the nerve. The temperature at the tip of the probe was only 42 degree CG. The RF machine which could deliver a pulse of RF (of 300 kHz) for 30 milliseconds out of a cycle. The voltage reached by this machine was in the region of 25-35 V and the output was adjusted so the temperature reached was no more than 42 degree CG.

The mechanism how pulsed RF works is not yet clear. One theory could be that it works like transcutaneous electrical nerve stimulation, activating both spinal and supraspinal mechanisms which may reduce pain perception.

Advantages OF Pulsed RF

A painless procedure.
There are no signs of neurodestruction.
Can be used for neuropathic pain.

Requirements of Ideal Lesioning Machine

Measurement of tissue impedance.
Wider range of frequency to stimulate the nerve
Accurate timing of the duration of the lesion.
Accurate measurement of the lesion temperature.
Accurate measurement of amperage and voltage.
The ability to slowly increase the temperature with time.

There are following Radio Frequency lessoning machines are available.

1- NeuroTherm
2- Radionics

Conclusions

The application of RF lesioning techniques has added new perspective in the management of acute and chronic pain. These techniques have greater advantage as it produces prolonged blockade over other neurodestructive procedures. However these techniques should be done by a competent specialist. At times wrong patient selection may produce poor results. For consistent lesion making, the physical lesioning parameters RF current, RF power and time to control the lesioning process are important. However the main parameter during lesioning is temperature control, being the end result of RF.

Recommended Reading

P.Prithvi Raj- Practical management of Pain- Third edition Mosby-2000.

NeuroTherm™ JK25T 5000 series

RADIOFREQUENCY GENERATOR MACHINE

Stage of Labour	Degree of discomfort
First stage (Latent Phase) (Cervix < 3cms dilated)	Usually mild pain; Fear and anxiety may led to moderate or severe pain
First Stage (Active Phase) 3 - 10 cms dilatation	Moderate uterine pain. May progress to severe pain.
Second Stage:	
a) Early	Severe uterine pain:
b) Late.	Moderate perineal pain.Severe Perineal pain.
Third Stage: Delivery of the placenta.	Minimal uterine pain
Fourth stage:	Little or no pain.

T11
L1
MAJOR PAIN PATHWAYS OF LABOR PAIN

An evaluation of the severity of labour pain in comparison to other forms of pain using the McGill Pain Questionnaire.
Note the effect of 'Prepared Childbirth Training' in reducing the intensity of pain experienced by primiparae.
The Pain Rating Index is derived by ranking the values of 20 sets of pain-descriptive words.

(Data have been replotted by the editors from: Melzack R 1984. 'The Myth of Painless Childbirth.')

LABOUR ANALGESIA

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The pain of childbirth is often rated by women as being the most painful experience of their lives.
It is estimated that about two thirds of normal healthy pregnant women suffer severe intolerable pain during labour and only 2% describe little or no discomfort. It is influenced by parity. Primiparous woman experiences more pain during early labour while multiparous woman feel greater pain in the second stage. There are several factors which influence parturition pain and its severity varies widely, the occurrence of truly painless labour is rare. It is frequently severe but due to the large emotional experience of pain, each woman's experience of labour pain is unique. Analgesic options must therefore be varied to allow for such a wide variation in the pain experienced. The most appropriate time to discuss the options for pain relief is before the woman goes into labour. There needs to be a degree of flexibility so that as the painful experience of labour progresses, the woman is allowed to exercise further options.

Physiological effect of labour pain.

A stress response is mounted to severe pain in labour. Mother experiences anxiety and fear; she may become pale and sweaty. Hyperventilation may be there which may lead to giddiness, fatigue and circumoral tingling as well as to uterine vasoconstriction in response to a low carbon dioxide concentration. The autonomic response to pain leads to an increase in the cardiac workload with tachycardia and vasoconstriction. Adrenaline release leads to hypertension and acidosis. There is delayed gastric emptying which may lead to nausea and vomiting. The progress of labour may be impaired due to severe pain as a result of inefficient contractions.

The First Stage Of Labour:- Pain in the first stage of labour results from dilatation of the cervix and lower uterine segment and from distension of the body of the uterus by uterine contractions and is transmitted by afferent nerve fibers (accompanied by sympathetic fibers) of the 10th, 11th and 12th thoracic and 1st lumbar nerves. The descending head causes pressure on the lumbosacral plexus responsible for pain felt in the back, thighs and legs

The Second Stage of Labour: - It is defined as the period after complete cervical dilatation until delivery of the foetus. The pain of second stage is due to stretching of the vagina and perineum and is somatic. It is better localized and is transmitted via the pudendal nerves to the spinal cord S2 to S4.

There are different modalities available to manage labour pain. The relief of labour pain ranges from the non-pharmacological to systemic opioids to regional anaesthesia.

Relieving labor pain.

There are different modalities available to manage labour pain. The relief of labour pain ranges from the non-pharmacological to systemic opioids to regional anaesthesia.

Ideal Method of Labour Analgesia:

1. It should produce efficient pain relief
2. It should not depress the foetal respiration
3. It should not depress the uterus there by preventing prolonged labour
4. Fall of blood pressure should be avoided to safeguard foetal hypoxia
5. It should be safe for both mother and child

A. Non-Pharmacological Methods

Generally learned beforehand during antenatal classes' .It is effective in the early part of labour and in conjunction with pharmacological methods. They include:

Psychological Preparation of the Parturient and Her Partner, having a support person present throughout labour, In 'Natural' or 'Prepared' childbirth the emphasis is on securing delivery without the need for drugs using a combination of techniques which may include:

1. Reduction of fear and anxiety by thorough education in the processes of childbearing;
2. Promotion of the concept that childbirth can be a drug-free, pleasurable experience ('psycho prophylaxis')
3. Variety of postures for labour, e.g. leaning over a bean bag, intermittent ambulation, rocking;
4. Conditioning exercises to strengthen back and abdominal muscles and relax pelvic joints;
5. Pleasurable, quiet and (sometimes) dimly-lit environment with soothing music (4);
6. Psychological exercises and conditioned reflexes;
7. Breathing control and mental exercises that focus on the breathing pattern;
8. Hot packs, gentle massage and warm showers; and
9. Supportive partner who acts as a 'coach'.

Relaxation and breathing technique.

Ç£psychoprophylaxis Ço means to prevent pain through psychological methods. It requires combination of antenatal instruction and the use of coping methods during labour. The basis of psycho prophylaxis is the belief that pain of labour can be suppressed by reorganization of cerebral cortical activity. The expectant mother is taught to respond to the beginning of a contraction by immediately taking a deep Ç£cleansing breath Ço, gently exhaling, and then breathing in a shallow pattern until the contraction ends as well as focusing on a specific object. It is observed that by using this technique mothers experienced 30% less pain in labour and reduced the incidence of forceps delivery.

Positioning and movement:

Pain relief requirements may be decreased again by up to 30% if the mother is mobile during labour. Changing to a more comfortable position may be of great benefit as long as lying flat on the back (aorto-caval compression) is avoided.

Heat, cold showering and massage.

Are all harmless techniques that may provide additional comfort.

Hypnosis.

It is claimed that the hypnotic trance achieves analgesia, shortens labour and that the acid-base status of the neonate is better at birth. In reality only about 25% of patients in labour, with the hypnotherapist present, can be hypnotized so that pain appreciation is adequately reduced. Usually hypnotic conditioning begins with sessions obtaining a greater degree of trance until a level of analgesia is acquired. The failure rate for self-hypnosis by pre-hypnotic suggestion is very high. Hypnosis is not without complications. Side effects include anxiety, and even frank psychosis.

Acupuncture.

The success rate of acupuncture is relatively low i.e. less than 25%.

Transcutaneous electrical nerve stimulation (TENS)

The gate theory of pain proposes that stimulation of large myelinated A-├ƒ nerve fibers will close the gate i.e. increase the pain modulating function of the substantial gelatinosa. Pain sensation from A-delta and C nerve fibers may thus be altered or blocked. TENS is thought to affect A-├ƒ fibers, although others suggest that the endogenous opioid system is responsible for TENS. Regardless of the etiology, it produces pain relief in 20 to 25% of mothers and to be of slight benefit in up to 60%.

B. Pharmacological methods

I. Inhalational Agents-

a. Nitrous Oxide. (ENTONOX)

Nitrous oxide is widely used for obstetric analgesia in most developed countries. The most commonly used mixture, a 50-50 blend of nitrous oxide and oxygen called Entonox, can be used in any stage of labor. Nitrous oxide is an analgesic. The exact mechanism of action is unknown. About 50% of women find it effective for labour. For it to achieve its peak analgesic effects, it is necessary to start breathing it 45 seconds before a contraction, which is very difficult to time. Its onset of action is 15 seconds and the elimination is rapid as it is not very soluble in blood. A concentration of 50% is required to produce worthwhile analgesia. The side effects include a feeling of disorientation or confusion and possibly nausea. Because it is completely eliminated via the lungs without being metabolized, there are no effects on the foetus. Unfortunately it is difficult to time effectively when in labour and so about 30% of women have no relief from N2O. The full analgesic effect usually is felt 50 seconds after inhalation. Entonox generally is self-administered as needed, but it can be administered continuously with medical supervision. Adverse effects included nausea, vomiting, and poor recall of labor.

b. Trichloethylene (Trilene) - 0.5% in air inhalation, sweet smell tolerated well

c. Methoxyflurane -0.35% used in inhalation

II. Narcotics.

Women in labour are commonly prescribed pethidine 1 to 1.5mg/kg intramuscularly 4 hourly prn. This alone is effective in about 60% of patients. The dose is usually timed to be at least three hours before delivery to avoid fetal respiratory depression. Patient controlled analgesia narcotics have also been used with patients receiving 15 to 25 μg bolus of fentanyl with a 5-minute lockout.

III. Parenteral Opioids

Parenteral narcotics are used to alleviate pain in 39 to 56 percent of labors in developed countries but still the safety and efficacy of opioids for labour analgesia is questioned. Primary maternal outcomes included maternal satisfaction with pain relief one to two hours after drug administration and characteristics of the labor process; secondary outcomes includes subsequent use of epidural analgesia, adverse symptoms e.g., nausea, drowsiness, inability to urinate or participate in labor, cesarean delivery or instrument-assisted vaginal delivery, and maternal qualitative outcomes such as satisfaction with the overall birth experience. Neonatal outcomes focused on respiratory depression, use of naloxone (Narcan), and feeding and bonding problems.

Meperidine has been extensively studied, but few trials have examined the effectiveness and safety of shorter acting agents such as fentanyl (Sublimaze). The safety and effectiveness of alternative modes of opioid administration, such as patient-controlled analgesia pumps still in trial.

Although opioids did provide superior pain relief and maternal satisfaction with pain management, the effect was small. Use of parenteral opioids is associated with lower rates of oxytocin augmentation, shorter stages of labor, and fewer cases of malposition and instrument-assisted delivery. Compared with epidural analgesia, parenteral opioids provide less pain relief and satisfaction with pain relief at all stages of labor. Bricker and Lavender found a lack of data to measure infant safety. Opioids are associated with neonatal respiratory depression, decreased alertness, inhibition of sucking, lower neurobehavioral scores, and a delay in effective feeding. Long-term effects cannot be excluded.

IV. Paracervical Block

The injection of local anesthetics into paracervical tissue for first-stage labor analgesia (1960s -1970s). Its use decreased after it was associated with fetal bradycardia and epidural analgesia became increasingly available. It is 75 percent effective in achieving good or excellent pain relief during the first stage of labor.

The incidence of postparacervical block bradycardia (PPCBB) ranged from zero to 40 percent. The exact etiology of PPCBB is unknown; it is thought to be due to inadvertent neonatal injection, vasoconstriction of the uterine or umbilical arteries, and elevated uterine tone or toxicity as a result of fetal absorption.

Paracervical blocks have a short duration of action, which requires repeated blocks during the first stage of labor, and they cannot be administered in the second stage of labor because of the position of the fetal head. Paracervical block is effective in the first stage of labor and does not require anesthesia personnel

for administration. No adverse effects of paracervical block on the progression of labor, likelihood of vaginal delivery, or neonatal outcomes unrelated to PPCBB. Uncommon complications include maternal haematoma or abscess and sacral neuropathy. It should not be used in women with non reassuring fetal heart tracings or uteroplacental insufficiency because the significance of PPCBB in this setting is difficult to determine.

V. Combined Spinal Epidural Analgesia (CSE).

Breen et al (1993) coined the term when ultra low dose of bupivacaine, epinephrine and fentanyl for labor which allowed patient to ambulate, often known as "Minimal motor block epidural". It can be single needle through needle or double space (spinal followed by epidural) technique. It allows the benefit of spinal anesthesia i.e. rapid onset and low failure rates and epidural analgesia i.e. use of catheter for continuous / prolonged infusion of local anesthetic of variable concentration.

The **indications** for the use a combined spinal epidural include:

- Very early labour in women who wish to ambulate
- Late in labour for multiparous women
- Operative or instrumental delivery where epidural analgesia is indicated postoperatively
- Limitation of initial analgesic is advantages (e.g. early in labor expected to be of long duration)

Advantages of CSE in labor

1. Increased intensity and decreased frequency of uterine contractions
2. Shorter first stage of labor
3. Less need for augmentation
4. Fewer assisted deliveries
5. Less frequent fetal heart rate changes
6. Improved APGAR scores
7. Greater maternal satisfaction
8. Enhancement of the rate of cervical dilatation by gravitational effects
9. Possible correct positioning of the fetal head due to this gravitational effects and movements of the pelvis during ambulation
10. Lesser incidences of aorto caval compression due to upright position

Criteria to allow ambulation

1. Desire to ambulate and not forced to ambulate
2. Absence of any obstetrical or medical contraindications
3. Ability to perform a straight leg raising test
4. Ability to do partial deep knee bends at bedside
5. At all times some one is present to walk with the parturient

Side Effects of CSE In Labor:

1. Does not provide satisfactory analgesia, higher concentration used might provide motor block
2. Pruritus
3. Nausea
4. Vomiting
5. Hypotension
6. Respiratory depression
7. Post dural puncture headache
8. Urinary retention
9. Fetal heart rate abnormalities

Drugs Used-

Spinal injection:-Bupivacaine 2.5mg (0.5%) or Fentanyl 25mg and bupivacaine 2.5mg

Epidural injection: - 0.1% bolus bupivacaine and fentanyl 2mg/ml or 0.1% bupivacaine and sufentanyl 5-10mg; other opioids can be used as an adjuvant to local

VI. Epidural Anaesthesia For Labour

Epidural anaesthesia can provide complete analgesia for labour and delivery as well as for caesarean section; however, epidural anaesthesia requires a greater level of skill for the anaesthetist and nursing staff. Epidurals are the most effective and consistently reliable way of relieving childbirth pain. An epidural will provide conduction anaesthesia of the spinal nerves and the spinal cord. (Neuraxial block) The aim is to provide analgesia by blocking the A-delta and C fibers of the spinal segments involved in the transmission of labour pain. However, because spinal nerves transmit motor, autonomic and other sensory impulses, they will also be blocked if a large enough dose of local anaesthetic is applied to them.

Indications Include:

1. Maternal distress caused by painful uterine contractions not adequately relieved by simpler forms of analgesia
2. Caesarean section. The majority of Caesarean sections are performed under epidural or combined spinal-epidural (CSE) anaesthesia
3. Provision of anaesthesia for instrumental delivery;
4. Treatment of pregnancy-induced and -associated hypertension (PIH). Epidural anaesthesia not only reduces the amount of sedation required but also lowers the arterial blood pressure. Once the blood pressure is controlled, the likelihood of eclampsia becomes more remote
5. Provision of analgesia for repairs to birth canal tears, e.g. episiotomy;
6. Breech delivery where the presence of an epidural in situ gives the obstetrician increased flexibility to intervene expeditiously should the need suddenly arises
7. Multiple delivery where anaesthesia (or, at least, superior analgesia) may be required urgently to deliver the second (and subsequent) infants, e.g. for version of the second twin
8. Cardiac disease or pulmonary hypertension where the epidural will attenuate the increase in cardiac output, mean arterial pressure and cardiac work which occurs during labour.
9. Restoration of coordinate uterine activity when the endocrine response to stress and pain has induced abnormal uterine activity.

Technical Problems:

1. Accidental dural puncture
2. Detecting misplaced catheters- intradural, intravascular, use of air to detect the epidural space
3. Patient controlled epidural analgesia(PCEA) Vs controlled epidural analgesia
4. Less local anesthetic concentration is required
5. Less motor block
6. Better pain relief
7. Less likely anesthetic or clinician interventions
8. Bolus doses produces more extensive spread with mechanical pump

Potential Advantages of Lumbar Route

1. Less local anesthesia is required
2. More rapid onset of analgesia
3. Less risk of infection
4. Less risk of damage to the rectum
5. Less risk of trauma to presenting fetal part
6. Higher overall success rate

Potential Advantage of Caudal Route

1. Lesser risk of spinal tap

2. Better perineal analgesia and relaxation

Effects of Epidural Analgesia-

Uterine Activity- Slow labor by decreasing uterine activity due to transient decrease in the frequency and intensity of the uterine contractions which was greater when epinephrine was used with local anetehtic agent. Supine position of the patient and rapid fluid administration prior to epidural block might decrease the uterine contractibility.

Uterine Contractions Mechanism

Increased Frequency and intensity Analgesia
Decreases catecholamine levels
Decreased Frequency and intensity- contractions
Decreased blood supply to uterus.
? Inhibition of motor nerve supply to uterus
? Direct effect of Local Anaesthetic on smooth muscle

Prolonged Labor- woman who receives epidural analgesia has prolonged labor, especially second stage of labor which is not harmful for the mother or baby. Woman with severe pain are more likely to request epidural and painful labor is itself known to make labour abnormally long and complicated. Woman with less cervical dilatation and with fetal part presenting higher up in the pelvis.

Malpresentations: - "risk of increased malpresentations" it is more due to maternal factors and technical issues than block as such. Fetal head malposition and motor block can increase the instrumental delivery it can be due to-

1. Inability to push down due to motor block
2. Reduction of serum oxytocin level results in weakening of uterine activity which may be due to part of fluid infusions
3. Increase incidences of occipito-posterior position of the fetus at delivery
4. Decreases the doctors threshold for performing instrumental assisted vaginal deliveries
5. Resident teaching becomes easier with epidurals

Prolonged Fetal Decelerations and Periodic FHR Changes- Exact cause is unkown.One of the cause is aortocaval compression. Others can be due to hypotension, absorption of LA from the epidural space resulting in uterine artery constriction or systemic toxicity or uterine hypertonus. It is more common with intrathecal opioids (sufentanil).

Fetal heart rate: beat to beat variability

Increase acute fetal hypoxia due to placental hypo perfusion Decrease Direct effect of local anaesthetic agents.

Chronic hypoxia and acidosis from placental hypo perfusion
Fetal heart rate: Long term deceleations
Early Head compression from increased uterine tone. Late Placental insufficiency worsened by placental hypo perfusion Variable/td> Cord compression aggravated by maternal hypotension and increased intensity of uterine contractions.

Problems of Epidural Analgesia

Brest feeding	Shivering
Maternal fever and neonatal sepsis	Abscess
Respiratory depression	Convulsions
Pruritus	Death (1 in 200,000)
Hypotension	
Postduralpuncture headache	
Backache	
Accidental subarachnoid puncture	

Injury to nerves- permanent or paresis

The success of the labour analgesia depends on the

1. Skill and experience of anaesthesiologist
2. Willing ness of the patient
3. Patient's mental makeup and positive attitude

Though labor is painful but the ultimate choice of the painless labor is as per mother's choice. Pros and cons of an invasive analgesic regime must be considered and the technique should be tailored according to the individual needs.

References:

1. Bofill JA, Vincent RD, Ross EL, Martin RW, Norman PF, Werhan CF, et al. Nulliparous active labor, epidural analgesia, and cesarean delivery for dystocia. Am J Obstet Gynecol 1997;177:1465-70.
2. Bonica JJ. Obstetric analgesia and anesthesia. Springer-Verlag Berlin, Heidelberg, New York 1972. p 31-33.
3. Breen TW, Shapiro T, Glass B. Epidural anaesthesia for labor in an ambulatory patient. Anesthesia analgesia 1993; 77; 919-124
4. Bricker L, Lavender T. Parenteral opioids for labor pain relief: a systematic review. Am J Obstet Gynecol 2002;186(Suppl 5):S94-109.
5. Brownridge P, Wood M. Soothing the pain of childbirth. Flinders Media, Flinders Medical Centre. 1992
6. Chantigian R. Non-Pharmacological methods for pain relief in obstetrics. In: Clinics in Anesthesiology - Obstetric analgesia and anesthesia. Ostheimer GW Editor. WB Saunders, Philadelphia, 197-207 1986
7. Elton CD, Ali P, Mushambi MB. Walking extradurals in labor. A step forward? British Journal of Anesthesia 1997; 79 (5): 551-4.
8. Hawkins JL, Beaty BR, Gibbs CP. Update on obstetric anesthesia practices in the U.S. Anesthesiology 1999;91:A1060.
9. Kangas-Saarela T, Kangas-Karki K. Pain and pain relief in labour: Parturients' experiences. Int J Obstet Anesth 1994; 3:67-74.
10. Loughnan BA, Carli F, Romney M, Dore CJ, Gordon H. Randomized controlled comparison of epidural bupivacaine versus pethidine for analgesia in labour. Br J Anaesth 2000; 84:715-9.
11. Melzack R. The myth of painless childbirth. Pain 1984; 19:321-325.
12. Morgan BM. "Walking epidurals" in labor. Anesthesia 1995.50(10) 839-40.
13. Pain relief and anesthesia in obstetrics. Eds: van Zundert A, Ostheimer GW. 1995. Churchill Livingstone, New York. p19-52
14. Rosen MA. Nitrous oxide for relief of labor pain: a systematic review. Am J Obstet Gynecol 2002; 186 (Suppl 5):S110-26.
15. Rosen MA. Paracervical block for labor analgesia: a brief historic review. Am J Obstet Gynecol 2002; 186(Suppl 5):S127-30.
16. Sharma SK, Sidawi JE, Ramin SM, Lucas MJ, Leveno KJ, Cunningham FG. Cesarean delivery: a randomized trial of epidural versus patient-controlled meperidine analgesia during labor. Anesthesiology 1997; 87:487-94.
17. Writer WDR Physiology of Labor. In: Handbook of obstetric analgesia and anesthesia. (Eds McMorland G, Marx F). WFSA p24 - 34, 1992

DIAGNOSTIC ISSUES RELATED TO NEUROPATHIC PAIN

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Neuropathic pain syndromes refer to heterogeneous painful conditions in terms of etiologies, topography of the injury, symptoms, signs and pathophysiology. About twenty five percent of patients referred to pain clinics have neuropathic pain and the estimated prevalence is two to four percent in the general population. It is associated with co-morbidities such as difficulty in sleeping, lack of energy, drowsiness, concentration difficulties, depression, anxiety and poor appetite. It is becoming a growing problem and hence early detection and management is vital.

Neuropathic pain is defined as "initiated or caused by a primary lesion or dysfunction in the nervous system" and the term "dysfunction" makes this definition vague and unacceptably broad. A proposed solution is to define neuropathic pain "as pain caused by a lesion of the peripheral or central nervous system (or both) manifesting with sensory symptoms and signs".
Neuropathic pain has a complex aetiology and over-excitability at a neuronal level plays an important role in neuropathic pain.
The clinical features of neuropathic pain are:

a) Spontaneous, relatively persistent pain superimposed by paroxysmal pain
b) Thermatomal (arteries and arterioles) or a combination of dermatomal and thermatomal distribution.
c) Hyperpathic and at times allodynic characteristic.
d) Causes an efferent response in the involved area in the form of vasomotor/somatotomotor symptoms.
e) The efferent responses result in temperature changes.
f) The efferent changes may result in either muscle weakness or loss of tone.

A careful history trying to assess the neuropathy is the foundation of the diagnostic workup. Based on the history, a focused examination, including sensory, motor and autonomic functions is crucial to confirm or reject the suspected anatomical site of the lesion. Since neuropathic pain in most cases involves alterations in skin sensitivity, quantitative sensory tests (QST) have proved to be of value. It is important to test all somatosensory modalities since dysfunction confined to a single modality may otherwise escape detection. Because tests of deep sensation are lacking, one can only examine sensory function in a nerve or tract that has a cutaneous representation. If nerve damage is limited to deeper tissues, the second diagnos tic criterion cannot be applied and the diagnosis of neurogenic pain remains tentative and based on indirect evidence. Quantitative sensory testing to assess perception thresholds for different modalities has been refined in recent years. Methods now exist to directly quantify tactile perception and vibratory thresholds, thermal non-nociceptive and nociceptive perception thresholds, and pinch-or-pressure pain thresholds. When bedside testing is inconclusive, QST of different sensory modalities is vital to profile somatosensory function.
Several independent pathophysiological mechanisms are responsible for different pain types in peripheral neuropathies. These operate in both the peripheral and central nervous system:

a) Pathological sensitization and ectopic impulse generation in primary afferent nociceptive fibres after nerve damage
b) Influence of sympathetic activity and catecholamines on sensitized and damaged primary afferents
c) Inflammation of the peripheral nerve trunk
d) Central sensitization as a consequence of nociceptor hyperactivity
e) Central synaptic reorganization as a consequence of nociceptor degeneration

Judicious use of infrared thermography gives fundamental information about the pathophysiology of a neuropathic painful syndrome as it sensitively detects and precisely delineates areas of cutaneous thermal changes of neural origin. The main role of infrared thermography in the diagnosis and management of neuropathic pain is to identify the nature of the pathologic lesion, and to stay out of harm's way. This is not an academic exercise, but such tests may spare the patient from unnecessary operations from application of ice, and from the dangerous insertion of needles for the purpose of trigger point injections or nerve blocks in pathologic areas identified by thermography.

FACTORS CONTRIBUTING TO OSTEOARTHRITIS

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[Definition of OA]

A disorder of synovial joints involving focal degeneration of articular cartilage with irregular regeneration and remodeling of adjacent subchondral bone.

[Risk factors]

Risk factors for OA include age, gender, genetic predisposition, obesity, occupation, race, injury, and sport activity.

1. Age

The prevalence of OA increases strikingly with age at all joints. OA is uncommon in adults younger than age 40 years, but OA is more common with increasing age. It is the most common in later life, and 80% of adults older than age 75 years suffer from OA.

2. Gender

OA in women is approximately twice than that of men. OA in the hands, knees, and hip is more common in women. The gender differences in OA incidence may be linked to postmenopausal estrogen deficiency. Estrogen receptors are present in articular cartilage, and estrogen potentiates cytokines involved in cartilage metabolism.

3. Genetic predisposition

OA appears to have a generic predisposition. Many genes predisposing to OA may be joint specific. In the hands, greater than 50% of OA occurrence may be associated with inheritance, but for knees this percentage appears to be smaller.

4. Obesity

Obesity is a high risk factor for OA of the knees, and the relationship of obesity to OA of the knees is stronger in women than men. Obesity also increases the risk for symptomatic OA of the hip. A weight loss produces a decrease in the risk for developing symptomatic OA.

5. Occupation

Several occupations are associated with OA involving a variety of joints. Stereotyped repetitive use of specific joints by workers produces degeneration of joints. OA of the hip in farmers and construction workers is more common. Standing for more than two hours/day, prolonged employment, and heavy lifting may produce coxarthrosis.

6. Race

The prevalence of OA of the hip is low among Asians. It is reported that the age-adjusted prevalence of radiographic OA of the hip in China is roughly one tenth of that in America. On the other hand, OA of the knees in China is as prevalent or more prevalent than that in America.

7. Injury

Acute major joint injuries frequently lead to OA. A large proportion of OA cases in joints such as the ankle and shoulder may be induced by injury.

8. Sport activity

Sport activities may increase the risk for developing OA. Elite athletes are at high risk for later OA, especially in weight-bearing joints. It is considered that football players have high rates of OA of the knees.

FAILED BACK SURGERY SYNDROME (FBSS)

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Definition

FBSS can be defined as a persistent or recurring low back pain with or without sciatica, following one or more lumbar spine operations. It can be associated with intractable pain and various degrees of incapacitation. These symptoms may follow after discectomy, laminectomy, or lumbosacral fusion and seen in 5%-10% of patients.

History

In 1951 Barr determined that t patient might have persistent low back pain, sciatica, or both despite surgical intervention. Finnneson and Cooper made a statement that "No matter how severe or intractable pain, it can be made worse by surgery"

Etiology

The causes of FBSS can be classified according to clinical presentation.

1- No improvement
   A-Faulty technique.
   B-Poor patient selection
   C-Creating new source of impingement

2- Recurrence after a period of improvement.
   A-Recurrent herniation
   B-Post operative fibrosis.
   C-Other source of impingement

3- Persistent low back pain.
   A-Destabilization
   B-Disc degeneration
   C-Facet joint syndrome.

Poor Patient Selection

The commonest cause of FBSS is remains the poor selection of patient. In a multi-center, nation wide, prospective study of the outcome first time spine surgery, it was determined that over 90% improved when chosen by spinal surgeons.

Waddell, in an assessment of the outcome of low back surgery stated that most important to remember that failure of first surgery residual pain is not an indication for second surgery. It was noticed that some surgeons carry out decompression procedure with or without discectomy, and then to follow that with spinal fusion. The problem is frequently that the first procedure ill chosen. In addition psychological or environmental factors also have a major impact on presentation of low back pain.

Another important factor even after refusal by a surgeon to perform surgery, many patient will "doctor shop" to pursue lawsuit, attract sympathy, attention, avoid unpleasant job or employer or to acquire

financial compensation. Thus inappropriate behavior not only by surgeon but also by patient can lead to FBSS.

Work related injuries or patients may not respond to any modality of treatment. Lastly some patient have an unrealistic expectation of complete return to full premorbid function with total absence of pain postoperatively, this expectation often leads to disappointment.

Failure To Corrct Initial Pathology

This can be one of the important causes of FBSS. At times operating wrong level or no stabilizing unstable segment can contribute to FBSS.

Epidural Fibrosis- EF

EF is one of the major causes of FBSS. The reported incidence of EF is from 10%-75%. The exact pathogenesis of EF is not known. One possible cause can be persistent debris from sponges may serve fibrogenic stimulus. Metal dust arising from poor quality surgical tools (eg.periosteal elevators) can be another cause.

Arachnoepiduritis may develop causes ischemia ant atrophy of nerve roots. In addition adhesions may bind the dural sac and nerve root sheath to the wall of the spinal canal, stretching nerve roots.

Non surgical causes of EF are annular tear, hematoma, infection and intrathecal contrast media.

Regardless of the cause, the pathology involves collagenous fibers that encapsulate nerve tissues, resulting in lateral spinal stenosis which could impair arterial tissue perfusion and venous return. EF causing adhesions may restrict nerve mobility, leading to increased incidence of lateral herniated disc symptoms.

Scar tissue is found in three components of epidural space.

Dorsal epidural scar tissue is formed by resorption of surgical hematomas and may be involved in pain generation.

In ventral epidural space, dense scar tissue is formed by ventral defects in the disc.

The lateral epidural space includes nerve roots, dorsal root ganglion, called sleeves that exit the root canals, are susceptible to the lateral disc defect, facet over growth, and neuroforaminal stenosis.

Recurrent Disc Herniation

Recurrent disc herniation either at same level or adjacent level can be the important cause of FBSS. The reported incidence is about 20%. At times it is difficult to determine the pain generator between EF and recurrent disc herniation. A herniated disc can cause pain through physical or chemical mechanisms. Pressure induced changes in nerve fibers are dependent of degree of force. For instance 10mm pressure applied decreases nutrient transport to the nerve roots by 20%-30%.

Chemical irritants from denerative disc or tears of annulus fibrosus results in leakage of chemical irritants such as phopholipase A2

Perioperative Surgical Comlications

Haemotoma formation

Pseudoarthrosis

Flat back syndrome-loss of normal lordosis.

Abnormal Posturing

Non Surgical Causes OF FBSS

Arachnoiditis

Inflammatory changes in the nerve root and surrounding arachnoid mater secondary to surgical trauma, infection, bleeding, disc disease, myelography can result in arachnoiditis.

Clinically the patient present with unremitting pain in low back and leg, which increases with movements and positions that stretches lumbar nerve roots. There may be patchy neurological deficit.

MRI findings can be divided in three groups:-

1- Conglomeration of adherent roots residing centrally within the nerve sac.
2- Distinguished by nerve roots, adherent peripherally to the meninges and give rise to empty sac appearance.
3- Soft tissue mass replacing subarachnoid space.

Spondylodiscitis

Wound infection immediately after surgery may result in to recurrent or persistent atypical lumbar pain with or without pain radiating down to the legs or back muscle spasm. It is usually associated with low grade fever, leucocytosis and high ESR.

MRI shows central disc enhancement, posterior annulus enhancement, and edematous bone marrow of both adjacent vertebrae

Spinal Stenosis

Most common cause of FBSS. Both central and lateral canal stenosis.

Lateral spinal stenosis is seen commonly as a result of gradual loss in disc height. The symptoms of pain and neurological deficit may be due to random ischemia of the vascular supply to the spinal nerves. The pain is deep aching in low back area, buttock, and thigh with radicular pain down the leg. Standing and walking are painful. Most of the pain reduced by forward flexion. Walking distance progressively diminishes over time.

Facet Joint Arthropathy

A previous discectomy, laminectomy or spinal fusion may cause adjacent facet joints to undergo accelerated degenerative arthritic changes. The pain is axial, seldom radiating below the knee, aggravated by extension and prolonged sitting. Diagnostic block with local anaesthetics is useful to differentiate facet disease from other causes of back pain.

Pseudomeningocele

It is leakage of CSF under pressure. Patient may complain low back pain. MRI is diagnostic, surgical repair is the treatment.

Tumors

Scwannoma or ependymoma is rarely, a cause of FBSS.

Diagnosis

FBSS can be diagnosed by clinical examination and MRI studies. The other tests such as EMG, nerve conduction study, diagnostic block, provocative discography and epiduroscopy.

Patient needs in depth assessment and role of medical team including rehabilitation, physiotherapist, and psychologist play important role.

Clinical history- Of the foremost importance one is the chronic disabling low back pain neurological symptoms, functional limitations and morbid psychological conditions. This requires detailed pain assessment, evaluation, type of pain, location, radiation, frequency, severity, association with movement and aggravating and relieving factors. For ex. pain resulting from spinal stenosis is relieved by forward flexion and aggravated by extension. Return of pain within months to years is result of fibrosis, aracnoiditis, facet joint disease, segmental instability, and recurrent disc herniation.

Physical Examination

The examination can be divided into musculoskeletal, neurological, functional and tension signs.

Musculoskeletal-Look for curvature of spine, and limitation of range of motion, for ex. repeated lumbar flexion is probably as a result of hyper mobility problem. Provocative tests can be secondary to stretch of the spinal cord or sciatic nerve or test of increased intrathecal pressure.

SLR- the pain must traverse typically below the knee to be called positive and test only provocative between 30-60 degrees. Reverse SLR or femoral stretch is specific for L2-3 radiculopahties.

Crossed leg test- more specific for radicular pain.

A thorough neurological examination is must. The "red flag" signs of cauda equine syndrome should be excluded. At times residual neurological effects such as foot drop or diminished deep tendon reflexes may linger despite surgical decompression.

Waddell designated five cardinal signs of non organic low back pain.

1- Superficial nonanatomic tenderness.
2- Stimulated pain of axial loading.
3- Distraction test (flip/ differential SLR).
4- Regional or light pinch with wide area of pain or weakness, or both and sensory loss.
5- Overreaction with disproportionate verbalization and facial expression.

On clinical examination any one of these signs is sufficient to establish an exaggeration of a physiologic state.

Allodynia and hyperalgesia secondary to central sensitization can present as superficial non anatomic tenderness.

Diagnostic Studies

Imaging Studies- Interpreting of imaging is more difficult in patients with FBSS because they may have superimposed anatomic abnormalities produced by primary surgery and instrumentation. MRI with gadolinium is helpful in diagnosing enhancing scar tissue and recurrent disc herniation or retained disc fragments. MRI has limitations in defining bony anatomy which can be visualized by CT and plain X-ray.

Electromyography- This method has a reported success rate ranging from 20%-90% for determining root compression. At this stage in patient evaluation, the original lumbar pathology, and surgical trauma or on going compression can yield positive EMG results there by making a negative study more useful.

Nerve Conduction Studies -These studies are useful in correlating clinical symptoms with noncom pressed nerve roots (from MRI).

Provocative Discography- is performed to determine the effect of mechanical loading of individual discs in patients with unremitting, chronic, discogenic low back pain. Elicitation of patient's characteristic pain is positive sign. Discography is mostly performed as a prelude to either Intradiscal Electothermal annuloplasyty (IDET) or fusion surgery.

Epiduroscopy-This diagnostic and therapeutic procedure is used for visualization of fibrosis, neovascularization, granulation tissue and entrapment of nerve roots. It is method of choice for epidural fibrosis and in the treatment of radiculitis by mobilization of adhesions around and entrapped and irritated nerve root (neuroplasty). At the same time corticosteroids, hylase and local anaesthetic can be injected.

Diagnostic Blocks -Facet block, sacroiliac joint block and selective nerve root block are useful in nonspecific spinal pain. The advantages of these blocks are as follows:-

1- They allow peripheral pain pathway to be localized.
2- Selectively blocking the specific nerves teases out that faction of the pain generator.
3- Relief of pain after local anaesthetic block is attributable solely to block of the target afferent neural pathway.

Facet joint pain is commonly associated with other causes of FBSS.

Preventive Measures

Men over 40 years of age, overweight, obese and heavy smokers are at increased risk of chronic low back pain. The impact of smoking on micro vascular circulation of peripheral nerves and vascular network surrounding the intervertebral disc should not be underestimated. Smoking increases serum porteolytic activity which increases the intervertebral disk degeneration weakens spinal ligaments, causing spinal instability.

Goupille emphasized that the decision to operate should be based on factors such as:-

1- Clinical correlation on the patient symptoms with imaging findings.
2- Presence of radicular pain more than axial pain.
3- Absence of social or psychological deterrent factors.
4- Willingness for surgery.

Treatment Options

FBSS is multifactorial, complex problem and challenging one for pain physician. The following are the treatment options available which can be used singly or multidisciplinary depends upon an individual patient.

Role of Psychologist- A clinical psychologist can identify confounding behavioral problems that will continue to foster poor response to the treatment. He can also be helpful in evaluating the chronic pain and patient will be assessed for psychotherapy, biofeedback, relaxation techniques, psychopharmotherapy, and mind body chronic pain programs.

Role of Physiotherapy- The structural symmetries, decondtioning, on going pain and attitude of the patient of FBSS is a suitable candidate for rehabilitation. The goals of physiotherapy are to improve range of motion, enhance strength, increase daily activities, improve ergonomics, and prevent or decrease musculoskeletal injuries. In FBSS there is loss of normal lumbar lordosis, chronic muscle spasm of paravertebral muscles, tightening and shortening of glueteal, hamstring and calf muscle groups. The rehabilitation process is slow and challenging requires continuous education and reinforcement.

Pharmacological Management-

NSAIDS-Pain associated with musculoskeletal disorders. The side effects include GI disturbances, renal dysfunction, platelet inhibition, and water and sodium retention, allergic reactions. Recently the risk cardiovascular events are noticed with Cox2 inhibitors.

Systemic opioids-The use of oral opioids is increasing in long term non-malignant pain.

Adjuvant analgesic drugs-such as anticonvulsants are useful for neuropathic pain.

Muscle relaxants- carisoprodol, tizanidine, baclofen, are useful in relieving of muscle spasm.

Epidural Corticosteroids

Epidural steroids remain most common therapeutic modality in FBSS. The rationale is to reduce inflammation via phospholipase A2 inhibition. Reduced inflammation and direct C fibre inhibition reduces sensitivity of nociceptors to mechanical stimuli and tensile force.

Epidural injection of corticosteroids and hyaluronidase seem to benefit in some of FBSS patients. Injection of local anaesthetic in to the epidural space to silence sympathetic nervous system involvement of nociceptors is without any meaningful long term recovery.

Percutaneous adhesiolysis or Epidural neuroplasty- is done with Racz catheter is done with special catheter with stylet and maneuvered through adhesions at the affected site under fluoroscopy. Then normal saline with hyaluronidase is injected with force to perform adhesiolysis. The catheter can be kept in situ for 72 hours or more.

Epiduroscopy- The epiduroscope can be used for visualization of epidural fibrosis, granulation tissue, neovascularization. Under aseptic precaution and under fluoroscopic contorl epidursocope is introduced in to caudal epidural space through steerable catheter or introducer. The catheter then maneuvered closer to epidural scarring. An epidurogram is done to see filling defect indicating area of scarring or fibrosis. Thereafter hyaluronidase is injected with reasonable force to hydrolyze excess ground substance in epidural scar tissue thus allowing diffusion of local anaesthetic and corticosteroid to target area.

Facet Joint injection- Degenerative disease of facet joint is important cause of low back pain with radiation to the knee level in FBSS. Facet joint injection with local anaesthetic and corticosteroid provides good pain relief. Trigger point injection with physical therapy is helpful during rehabilitation program.

In some patients botulinum toxin A is injected in to Psoas, Quadrates lumborum, piriformis and paravertebral muscle can provide excellent pain relief.

Intradiscal Electrtheral Therapy (IDET) - Patient suffering from FBSS due to internal disruption of disc will be benefited with this technique. Studies evaluating IDET for patients with discogenic pain as suggested by history, physical examination, and discography have generated mixed results. However improved

patient selection, psychological testing and better technique will probably offer a select patient a nonoperative solution to discogenic pain.

Spinal Cord Stimulation (SCS) - SCS has been in use for FBSS since 1967 and has created basis for interventional pain management in neuropathic pain. The gate control theory by Melzack and Wall and central inhibitory mechanisms evoked by Abeta stimulation are commonly accepted mechanism of analgesia brought on by SCS. Long term studies have a success rate of 50%-70% for FBSS at 5 years. Dual lead placement in patients with FBSS allows for more programming operation to cover axial pain. SCS can lower medical costs by reducing office and emergency department visits. Medication requirements tend to stabilize or decrease activities of daily living increase and some patients are able to return to a meaningful employment. Overall, SCS is a viable option for patients who fail to obtain sufficient pain control.

Central Neuraxial Infusions

Recently the use of implantable pump for nonmalignant pain in particular intrathecal opiates for FBSS is increasing. It can reduce pain score by 25%-50% and improves coping skills in FBSS.

Reoperation

Reoperation should only considered if there is clear anatomic reason for worsening pain and conservative measures have failed. How ever it has a risk of epidural fibrosis.

Conclusion

In all studies of back pain 10% to 15% of patients account for 80%-90% of the total health care consumption for spine disorders, and 2% of patients who undergo spinal surgery are the most expensive group. At least 15% of patients who undergo spinal surgery develop FBSS. The lack of such satisfactory results in nonmalignant pain and represents the magnitude of the complexity of back pain in significant number of patients.

NEUROLYTIC SPLANCHNIC NERVE BLOCK FOR UPPER ABDOMINAL CANCER PAIN

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The mortality rate of cancer patients have been at the top for 35 years in Japan. One of the problems about advance cancer patients is that about 70% of these patients are to have pain during their survival periods and one half of them are reported to be have intractable pain. So, pain relief is the most important issue for the patients with not only advanced and also with early stage cancer.

Neurolytic splanchnic nerve block has been used for the management of pain caused by upper abdominal cancer. We have applied this procedure to more than two handred patients with upper abdominal cancer pain and have gotten good results immediately following the blocks in about 90% of the patients.

The aim of this investigation was to evaluate pain management after neurolytic splanchnic nerve blocks comparing it with patients without the blocks whose pain were treated with the conventional WHO methods.

We have treated two handred and fifteen patients with pain caused by upper abdominal cancer between 1987 and 2005. In these patients, 68 patients received neurolytic splanchnic nerve block, but 147 patients didn't because of the following reason. Poor general condition such as ascites, pleural effusions, hypo-protenemia, etc. in 92 patients. Multiple metastases in 32 patients, and controllable pain in 23.

We compared the results of pain management and the subsequent clinical courses in these two groups, that is , blocked proup of 68 patients and the non-blocked group of 147 patients.

The averaged age sex and primary sites of the cancers in each group were similar. Stomach, pancreas and liver cancers were the most prevalent cause of pain. The degree of pain expressed by the 11 points Visual Analogue Pain Scale (VAS) at the time of the first examination in our Pain Clinic and in the subsequent period after treatments.

The VAS value at the first examination in our each group was about 7, respectively, showing no statistical significance of difference. The VAS value in the time after treatments were about 1 in both groups. VAS value decreased significantly in each group meaning that both splanchnic nerve block and WHO methods were effective to treat upper abdominal cancer pain.

In the blocked group, we had 20 patients (30%) who became completely pain free, and 22 patients (32%) were treated further with Non-steroidal

Anti-inflammatory Drugs. Seven patients were prescribed codeine phosphate and 12 patients received strong opioids orally or intravenously. Epidural analgesia was used in 10 patients (patients, overlapped).

On the other hand, in the non-blocked group, no patients experienced complete pain relief. And 100 patients (68%) received strong opioids, 22 patients (15%): codine. 10 patients (7%) : NSAIDs and 15 : unknown. Further, larger dosage of strong oipids were used in the non-blocked group than blocked group.

Although we do not want to emphasize that large dosages of strong opioids should be avoided to treat cancer pain, it is thought that lower dosages of medications would be more acceptable to patients with cancer pain.

In conclusion, we want to stress that neurolytic splanchnic nerve blocks make the management of pain easier in selected patients with upper abdominal cancer.

HISTORY OF PAIN MANAGEMENT IN BANGLADESH

Introduction:

Prof. John Bonica, an Anaesthesiologist and founder President of World Federation of Societies of Anaesthsiologists in USA first started multi-disciplinary pain clinic in 1950s. It became popular in different parts of the developed world. Ultimately, "International Association for Study of Pain" was formed in 1975, with members from various specialties working on pain management. There are now 51 chapters including Bangladesh actively functioning and 11 chapters are in formation.

History in Bangladesh:

Pain clinic was not in existence in Bangladesh. All medical practitioners used to treat pain, as it was the presenting criteria in most of the diseases. It was in 1977-78; Prof. M.Shafiqur Rasul first started Musical Mesmerism in Chittagong, though it was not popularly used. The beginning of Asian and Oceanic Society of Regional Anaesthesia- AOSRA (Later, named as Asian and Oceanic Society of Anaesthesia and Pain Management - AOSRA-PM) has an influence in development of pain management in South-East Asian Region including Bangladesh, which has a foundation member and director of AOSRA-PM. Major (Colonel) M.A. Zohur started treating pain patients exclusively in a chamber in Kalabagan, Dhaka in 1992. Simultaneously, Prof. S. N. Samad Choudhury also started practicing pain management in a clinic Dhanmondi, Dhaka, in 1992. Dr. Junaid Shafiq started treating pain patients in Japan Bangladesh Friendship Hospital in Gulshan, Dhaka in 1994. "Analgesia" a pain centre for treating out-patient pain patients was started in Kolabagan (later-on shifted to Dhanmondi), Dhaka. Dr. Shamsul Alam started pain centre in Khulna in 1996. Prof. Md. Shamsul Alam, Dr. Mahmudur Rahman and few others started pain management in various places

Academic development in Bangladesh:

A paper on "Scope of Pain Clinic" was first presented by Prof.K.M.Iqbal in a conference of Bangladesh Society of Anaesthesiologists in 1983. Two papers, one by Dr. Habib Ibrahim (Epidural Blocks for Abdominal Pain), and the other by Prof.S.N.Samad Choudhury (Pain management of Head-Neck Region due to Malignancy and Gasserian Ganglion Block for Trigeminal Neuralgia) were presented in a conference in Bangladesh College of Physicians & Surgeons, in early 1993. Prof. S.N.Samad Choudhury presented a paper on pain management due to lung cancer in Auckland, NewZeland in 1996 during International Congress on Regional Anaesthesia.

Greatest impact was created on development of pain management in Bangladesh during the First Conference of South Asian Confederation of Anaesthesiologists (SACA) held in October, 1993 while a big group of Pain Specialists joined the congress from abroad esp. from Japan and presented many papers on pain management. Pain Specialists from Japan offered to co-operate in developing this specialty in Bangladesh.

The Department of Anaesthesiology, IPGM&R (now BSMMU) first started "Pain Clinic" for out-patient in co-operation with Neuromedicine, Neurosurgery, Psychiatry and Physical Medicine in 1994 under the guidance of Prof. K.M.Iqbal. Collaboration between this Department and Japanese Pain specialists started in 1994. Prof. Kosaka of Shimane University, Japan, through the initiation of Dr. Lutful Aziz and Dr. Shamsul Alam, engineered the exchange program to train the Anaesthesiologists from Bangladesh in the pain management. Dr. Manjurul Hoq Laskar and Dr. Mahmudur Rahman were the first two Anaesthesiologists who were trained in this program from Shimane University in 1994. Later-on, Dr. Kenziro Dan initiated the training of Dr. Moinul Hussain from Fukuaka University. Dr. Abdul Khaleque Beg and Dr. Jahangir Kabir were trained in the same program.

Prof. Kosaka of Shimane, Prof. K. Dan of Fukuaka and Prof. S. Ogawa of Tokyo contributed equipments for the development of "Pain Clinic" in BSMMU.

Formation BSSP

Bangladesh Society for Study of Pain (BSSP) was formed in February 1997 with 11 (eleven) working pain specialists, which has become an affiliated Chapter of International Association of Study of Pain (IASP). Now the membership has increased to many.

The collaboration program continued with Japan and BSSP (in place of BSMMU). In this program Dr Zerzina Rahman was trained in Shimane University. Prof. Satsuro Ogawa organized training of Dr.A. B. M. Maksudur Rahman, Dr.A.K.M. Akhteruzaman .Lt.Col.Delwar Hussain, Dr. Hasibul Hussain and Dr. Shahnaz Begum in Nihon University, Tokyo, Japan.

BSSP organizes bimonthly Seminars on different topics of pain regularly. The First Annual Conference was held in March, 1998; Second one was held during Silver Jubilee of Bangladesh Society of Anaesthesiologists (BSA) in February, 1999; Third held in February, 2000; Fourth in February, 2001; Fifth was held along with SACA Congress in 2003.

BSSP participated in the World Congress of Pain held in San Diego, USA in 2002.

Formation of SARPS:

BSSP initiated the formation of South Asian Regional Pain Society (SARPS) during the Conference held in Dhaka with representative from all SAARC members.

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MYOFACIAL PAIN VS FIBROMYALGIA PROGRESS TOWARDS UNDERSTANDING

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This lecture presents the definition of myofacial pain (MP) and fibromyolgia (FM), the possible pathophysiology for better understanding of pain mechanisms and the common differentiating characters of both. The main aim of this paper is to review available data and current hypothesis concerning pathophysiology of myofacial (MP) and fibromylgia (FM) to see progress towards understanding of possible pain mechanism.

Recent years added new information to our understanding about etio-pathophysiological factors of these common clinical problems. Researches on genetics, biogenic amines, neuro-transmitters, oxidative stress and mechanism of pain modulation, central sensitization and autonomic function revealed various abnormalities indicating that multiple factors and mechanisms are involved in the pathogenesis of chronic muscular disorder.

These two medical conditions,Mp & FM are not similar however they often occur together. MP is known to be very common as mentioned in famous "Nuprin report". According to him 53% of all American report muscle pain. In contrast the reported incidence of FM is low & around 2-4%.

Pathophysiological explanations for both conditions are hypothetical. Trauma or acute overload is an inciting factor for MP. No histopathological finding have found and despite the lack of microscopic findings various theories have been presented. Chronic local ischemia, metabolic disturbances, central nervous system sensitization and residual adhesion from previous injuries are the acceptable theories. The factors like sex hormone, sleep deprivation, neurotransmitter imbalance, depression, genetic involvement and oxidative stress and nitric oxide have been identified for pathogenesis of FM . Overall recurring injury and bio-mechanical imbalances perpetuate muscle strain.

The diagnosis of both condition are made by history and clinical examination. No specific diagnostic laboratory test is recommended. Various major and minor clinical criteria have been in practice.

Some predictions about future development are ventured based on current state of knowledge & and considering those for MP and FM , future research should be focused on oxidative stress, nitric oxide and anti-oxidant treatment to enhance better understanding of both common clinical conditions .

References

1. Linakar CH, Walker - Bone K, Palmar K. frequency and impact of regional musculoskeletal disorder. Baillvers Best Pract Res Clin Rheumatol 1999; 13:197-215
2. Simons DJ. Myo-facial trigger point : A possible explanation. pain 1981; 10:106

3. Melzack R. Myo-facial trigger points : relation to acupuncture and mechanism of pain. Arch Phy Med Rehabil 1981; 62:114
4. Parziale JP, Chan JJ. Fibromyalgia. Me Health RI 1996; 79:188
5. Genetics of fibromyalgia. Curr pain headache Rep 2005 Oct; 9(5):313-5
6. Sogut S et al. changes in nitric oxide levels and antioxidant enzyme activities. Pathophysical mechanism. Chin chim acta 2003; 331:111-117.
7. BannetR. Fibromyalgia:Present to future. Curr Rheumatol.2005;7(5):371-6.

CAUDAL EPIDURAL BLOCK IN PAEDIATRIC PATIENTS- EFFECTS OF BUPIVACAINE, NEOSTIGMINE AND FENTANYL

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Objective

Bupivacaine is the usual drug injected caudally for analgesia in children in a dose of 2-2.5 mg/kg, action of which lasts for 2-4 hrs. Most of the children undergoing subumbilical operation require further analgesia during postoperative period. This study was done to assess the intensity and duration of postoperative analgesic efficacy of neostigmine and fentanyl in combination with bupivacaine through caudal epidural route and to assess associated nausea and vomiting with fentanyl and neostigmine as an adjunct to bupivacaine.

Method

Sixty children aged 2-6 yrs of ASA physical status 1 and 2 undergoing subumbillical operations was randomly divided into three groups of 20 children each and allocated to receive one of three solutions for caudal epidural block. No premedication was given, and general anesthesia was induced with nitrous oxide, oxygen, and halothane. Children in group -1 received a caudal injection of 0.25% bupivacaine 1 ml/ kg for each.Group-2 received an identical local anesthetic doses mixed with neostigmine 2 ╡g/kg. Group-3 received bupivacaine, neostigmine and fentanyl 1mg/kg to a total volume of 1 ml/ kg for each children . Caudal epidural block was performed under general anesthesia. Surgical intervention started 10 to 15 minutes after caudal injection of analgesic medication. Postoperative pain was assessed using pain score. Side effects mainly nausea and vomiting was also recorded.

Results

Our study shows that caudal neostigmine along with bupivacaine significantly prolongs the postoperative analgesic action. Addition of fentanyl also prolongs the analgesic efficacy as comparable to bupivacaine with neostigmine group. This study also shows that incidence of nausea and vomiting is more with addition of neostigmine and fentanyl.

Conclusion

We can conclude that neostigmine or neostigmine with fentanyl improves the quality of analgesic effect of bupivacaine in terms of intensity and prolongation of duration of analgesia in children under going subumbilical operations. It is also found that both drugs are associated with increased incidence of nausea and vomiting.

LIGNOCAINE VS LIGNOCAINE-KETAMINE MIXTURE IN BIER'S BLOCK: A COMPARATIVE STUDY

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Bier's block is generally a safe technique but tourniquet pain is considerable. Clonidine or ketamine in combination with lignocaine reduces the tourniquet pain. On the other hand recommended dose of lignocaine in Bier's block is unsafe in terms of cardiac toxicity when tourniquet released earlier or at tourniquet failure. Ketamine has a potent analgesic property. Unpleasant side effects can be abolished or controlled by pharmacological adjuvant. We have taken up this study for the experience of low dose lignocaine with low dose ketamine mixture as an anaesthetic solution in intravenous regional anaesthesia (IVRA) in this randomized clinical study. Method: A total 60 of ASA-I & ASA-II patients were included for the study of scheduled upper extremity surgery. Patients in control group (Gr-L) received 40 ml solution of 0.5% lignocaine (200 mg) and the trial group (Gr-LK) received 40 ml solution of 0.15% lignocaine (60 mg) with 0.3% ketamine (150 mg) as an anaesthetic solution. Rubber tourniquet was used in absence of pneumatic one and at least 20 minutes were allowed to establish anaesthesia as well as to ensure safety for the change or removal of tourniquet. Pulse, blood pressure, sedation score, respiration, tourniquet and surgical pain in VRS scale rescue pethidine and complications were recorded 60 minutes postoperatively. Result: Tourniquet pain was significantly less in Gr-LK ($p<0.000$) than the Gr-L and inadequate analgesia was also significantly less in Gr-LK ($p<0.000$) than the Gr-L. Postoperative analgesia was significantly prolonged (76.40 ▒ 11.78 min) in Gr-LK. Nausea vomiting was not significantly different. Conclusion: This study demonstrates that low doses of lignocaine and ketamine mixture in IVRA produces comparable anaesthesia during the procedure as well as significant analgesia in post operative period. The mixture (Gr-LK) is a more potent anaesthetic solution, reduced the tourniquet pain and the total amount of lignocaine used is safer irrespective of the study time in terms of cardiac toxicity than the lignocaine (Gr-L) alone.

RECOVERY AFTER COMBINED EPIDURAL - GENERAL ANAESTHESIA : COMPARISON WITH GENERAL ANAESTHESIA

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Postoperative problems regarding anaesthesia and surgery depend on quality of recovery. Recently combined general - epidural anaesthesia is applied instead of general or epidural anaesthesia for upper abdominal surgery presuming better recovery and reducing postoperative morbidity and mortality. This study was objected at evaluating the efficacy of combined epidural - general anaesthesia on patient recovery and its different aspects.

In our study, patients of ASA physical states I & II and of different baseline characteristics having insignificant differences (P-values>0.05) were randomly selected into two groups considering their inclusion and exclusion criteria to receive either general anaesthesia (Control Group) or combined general - epidural anaesthesia (Experimental Group) in their elective upper abdominal surgery to see and compare immediate recovery status.

The cardiovascular parameters like heart rate, systolic, diastolic and mean blood pressure were found to be significantly different with the Experimental Group having better rates than the Control Group within first two hours after extubation but became almost insignificant within 2 - 4 hours of extubation.

Oxygen saturation of the Experimental Group was found significantly higher than the Control Group (P-value was always <0.05) for up to six hours after extubation. Considering P-value, the difference between the two groups' respiratory rates was not insignificant until 6 hours after extubation. Taking the nature of respiration into account, majority in the Control Group were dyspnic comparing to the Experimental Group, none of whom showed any dyspnoea at 15 and 30 minutes interval.

The difference of recovery activities between the two groups remained significant all over the observation period as well as the consciousness level parameters.

During postoperative period, recovery from the individual general anaesthesia application is noticeably more stressful and lengthier process than the combined epidural-general anaesthesia, which can be substantiated by Modified Aldrete Recovery Score.

Combined epidural - general anaesthesia application, therefore, ensures smooth recovery with better haemodynamic stability without major side-affects in contrary to the general anaesthesia use.

INTRATHECAL LOW DOSE PETHIDINE AND FENTANYL WITH HYPERBARIC BUPIVACAINE FOR ELECTIVE CAESAREAN SECTION - A COMPARATIVE STUDY.

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Caesarean section is one of the common operations in the child bearing age of a woman. Regional anaesthesia is the technique of choice for elective caesarean section. Advantage of regional techniques include: less neonatal exposure to potentially depressant drugs, a decreased risk of maternal pulmonary aspiration, an awake mother at the birth of her child.
. Spinal anaesthesia has rapid onset and it does not produce serious systemic drug toxicity because of the smaller dose of local anaesthetic. Although a local anaesthetic solution may be used alone for induction of spinal anaesthesia, opioids are commonly added. When lipophilic opioids fentanyl and sufentanil were added to local anaesthetic, early postoperative analgesia was prolonged compared with local anaesthetic alone. Pethidine is an opioid of intermediate lipid solubility and is unique in having significant local anaesthetic property. It has been used as a sole agent during spinal anaesthesia for caesarian section.
So far literature reviewed, low dose pethidine with hyperbaric bupivacaine has been used as Epidural post operative analgesia after caesarean section small dose of opioids administered directly into the cerebrospinal fluid (CSF) have been found to be very effective in controlling symptoms of pain in many clinical situations. Although recent works suggest fentanyl as drug of choice for intrathecal use, pethidine has not been compared in terms its efficacy. This is needed as pethidine still remains as the readily available cheap agent in the country. Therefore, the purpose of this study was to investigate the effect of adding pethidine or fentanyl to hyperbaric bupivacaine in patient undergoing elective caesarian section under spinal anaesthesia. Selection criteria - Patients of elective caesarian section. Patients of ASA-I and ASA-II Exclusion criteriaPIH, Eclampsia, Placenta previa. Group A: Control group - Hyperbaric bupivacaine 0.5% of 2 ml + 0.2 ml distilled water intrathecally at L2-3 or L3-4 space. Group B : Fentanyl group - Hyperbaric bupivacain 0.5% of 2 ml + 0.2 ml fentanyl (10 microgram) intrathecally at L2-3 or L3-4 space. Group c: Pethidine group - Hyperbaric bupivacaine 0.5% of 2 ml + 0.2 ml of 5% pethidine (10 mg) intrathecally at L2-3 or L3-4 space.
It was a prospective comparative study of 90 parturient undergoing elective caesarean section under spinal anaesthesia. Parturient was randomly allocated by card sampling into three groups (30 in each group). Patients were premedicated with a regime consisiting of inj. Prochloparazine and ranitidine. And 20 mLKg-1 Lactated Ringer's solution was given as preload. Standard monitoring was applied, including continuous pulse oxymetry and ECG. Noninvasive arterial blood pressure was recorded.. Hypotension, define as a decrease in systolic arterial pressure to less than 90 mmHg or a decrease of 25% from base line, treated with boluses of IV Ephedrine 10 mg as required. Apgar score of the baby was recorded at I and 5 minute. Intraoperative pain and pruritus was assessed according to a three point scale (O=no symptom, 1=mild symptom with with no treatment required, 2-symptoms are such that treatment is required on parturient request). Any instances of respiratory depression (defined as a respiratory rate of less than 12 breaths/min), shivering or nausea or vomiting was recorded. Nausea and vomiting was treated In the Postoperative ward the parturient was monitored in terms of sedation, respiratory rate, pulse oxymetry and noninvasive arterial pressure every 15 minutes for 6 hour and then at hourly until parturients were discharge after 24 hours. The results was compiled and analyzed statistically using Anova-test with a alpha value of 0.05. It was seen that the parturient was haemodynamically stable causes effective prolong post operative analgesia without affecting the outcome of the baby compared with bupivacaine fentanyl group and bupivacaine alone.

COMBINED SPINAL EPIDURAL FOR LABOUR ANALGESIA: A COMPARISION BETWEEN BUPIVACAINE-FENTANYL WITH BUPIVACAINE-TRAMADOL

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Objective: The labour pain represents the most common form of acute severe pain in adult life. Regional anaesthetic techniques are preferred for management of labour pain. Recently, the combined spinal epidural (CSE) technique has gained popularity as an approach to labour analgesia. But pain relief in labour is still in infancy in our country. This study was designed to see the effectiveness, satisfaction and safety of the drugs regime.

Study Methods: Forty parturient patients were enrolled in this study that was randomly divided into two groups of twenty parturients each by blind envelope method. Analgesia was induced in Group A, Intrathecal-Bupivacaine 1.25mg + Fentanyl 20 ┤g. and Epidural infusion- Bupivacaine 0.0625% + Fentanyl 2┤g/ ml at the rate of 10-15 ml/ hr. In Group B, Intrathecal- Bupivacaine 1.25mg + Tramadol 10 mg and Epidural infusion- Bupivacaine 0.0625% + Tramadol 1mg/ ml at the rate of 10-15 ml/ hr.

Results: The result shows both the regimes produce effective and satisfactory analgesia. No significant difference was observed in haemodynamic parameters, quality of anaesthesia and analgesia. In this technique the added benefits were rapid onset and less motor block. That allows the parturient to ambulate during the period of labour and immediately after delivery. But complications like vomiting and pruritus were seen in fentanyl group. Apgar Score of the baby at delivery was in between 7-10.

Conclusion: Combined spinal epidural technique is a worldwide acceptable technique for the management of labour pain. So, it can be recommended that labour pain can be effectively treated by the bupivacaine-tramadol regimen which is a good choice for labour analgesia by combined spinal epidural technique.