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P.S. Shankar

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Former Director-General of World Health Organization (WHO), Gro Harlem Brundtland wrote that World has entered 21st century with hope but also with uncertainty. The remarkable gain in health, rapid economic growth and unprecedented scientific advances lead us to a new era of human progress. But the darker legacies from avoidable burden of disease and malnutrition bring uncertainty to this vision. The scientific advances have made enormous achievements. The things that seem routine today would have taxed imagination of even the most futuristic person just a few years ago. With scientific achievement human structure and function have reached a level of comprehension, which was unknown.

Health care system

Life is not merely living, but living in health. All our efforts are directed to maintain our health physically, mentally, socially, spiritually and intellectually in a proper way. The history of medicine is as old as the history of mankind. The health care system is in a constant stage of flux. Medicine is an art and science of promotion of health, prevention of disease, diagnosis, cure and rehabilitation. We have taken up this profession 'to cure sometimes, to relieve often and

to comfort all the times'. We have valued helping and serving others. The sea of changes in the concept and knowledge of medicine have come about due to major advances in diagnostic and therapeutic modalities. Despite that the values and ethics have always formed the underlying current of medical profession and have guided the physician about providing the best and most effective care to the patients.

Medical profession

Medicine is a profession. The persons pursuing it always put the needs of the patients before their own self-interests. They are neither perturbed at the unhealthy competition that has made the profession a business, by unscrupulous elements nor look at the financial benefits. They practice the profession following the ethical principles enunciated by Hippocrates, Charaka and Sushruta. These qualities of a professional man, in the words of Thomas Russel, Executive Director of American College of Surgeons, are multi-dimensional. It consists of competency and dedication to improve the skills, becoming a role model for future generation of medical men and placement of welfare of the patient above everything else. Sir William Osler, the renowned Physician of twentieth century said, that 'the

physician must be friend, philosopher, well-wisher and guide to the patient and the family under his care'. The physician has an obligation to practice his profession with conscience and dignity. Medicine is not only a science, but also the art of letting ourselves, individually interacting with the individuality of the patient. Our Vedas have looked the physician as a father in illness, as a friend in convalescence and as a guardian when health is restored.. According to Christopher Hufeland, the physician must generalize the disease, and individualize the patient. Francis Peabody has stressed that the treatment of a disease may be entirely impersonal, the care of patient must be completely personal.

Ideals of medical profession

From the times of Charaka, Sushruta and Hippocrates, the medical profession has formulated its own code of conduct explaining the physician's duties toward the patient and his obligation to the society. These have been modified in the recent years without altering the basic principles. The primary obligation of the physician is his professional duty. He must willingly 'consecrate his life to the service of humanity', and must practice the art and science of

medicine 'with conscience and dignity' and 'maintain the honour and noble tradition of his profession'. Medical ethics is an admirable attribute to be possessed by the physician and it forms an integral part of the healing art. It focuses the attention on patient care, protection of privileged information and maintenance of a special relationship between doctor-patient. Ever since the inception of medicine and medical practice, there has been a special relationship between a doctor and a patient. This complex and intricate entity is unique, without any parallel in any form of human behaviour or any facet of social field. R.K. Gandhi, noted Paediatric Surgeon said, existence of a sentimental bond, an emotional attachment, an irresistible affinity, an enticing attraction, unflinching faith, unflinching loyalty, mutual respect and reciprocity, compassion, and other such feelings and qualities have been intricately interwoven into this enigmatic entity.

Priceless possession

Doctor-patient relationship gives lot of dividends to the patients. It has the source of doctor's enormous empathy, his sincerity of service, his anxiety for the ailing, his consistent desire to allay the agony, his attention to patient's minutest comforts etc. By doing this work diligently the doctor lifts the medical profession high in the society, and brings nobility to the profession. He is respected, adored and worshipped by the grateful people. No other profession has

strived so hard to nurture such a bond. This relationship, being a priceless possession, must be preserved and protected. Apathy, indolence and inaction break this vital relationship. Consumer Protection Act (1986) has come due to a steady erosion of doctor-patient relationship in the recent decades. A physician must keep before him the high ideals of profession. While defining the ideals Charaka has told that 'one who desires to become a physician must previously become thoroughly knowledgeable and cultivate virtues by working hard. Then only he is in a position to restore the lost health of the patient and bring him back to normal life. Ethical values have become more important in these days. The ethical considerations have human orientation. The physician, according to Charaka, must develop the qualities of friendliness, compassion for the suffering patient and eagerness to do his best to relieve the suffering. The advice given by Charaka to the practitioners has all time relevance. It states, 'he who practices neither for money nor for caprice, but out of compassion for living beings is best among all physicians. The physician who set out to sell their skill like a commodity only, loses sight of the gold and acquires heap of dust. The physician who considers compassion for living brings as his highest religion fulfils his mission in life and obtains the highest satisfaction'.

Servant of the nature

Rudyard Kipling classified the mankind in the World into two

groups-doctors and patients. Since the times of Hippocrates and Galen, the relationship between the nature and physician has been appreciated. Hippocrates considered Natural forces as the healers of disease; and Galen stated that the physician is Nature's assistant. Physician works with the aim 'to prevent disease, to relieve suffering and to heal the sick'. Paracelsus considered physician as the servant of the Nature, not her master, thus it behooves medicine to follow the will of the Nature. He had emphasized, that the art of healing comes from Nature, not from physician. Therefore the physician must start from Nature, with an open mind. Thus a close co-operation exists between the Nature and physician. Homer Emerson summarized the fact as 'Nature heals, under the auspices of the medical profession'. Voltaire humourously said, the art of medicine consists in amusing the patient while nature cures the disease. Hans Krebs has said, 'you and your family must clearly understand that the great and ultimate healer is always Nature itself and that the drug, the physician, and the patient can do not more than assist Nature by providing the very best conditions for your body to defend and heal itself'. Anita Hesselgesser has written that the more man follows Nature and is obedient to her laws, the longer he will live; the further he deviates from these, the shorter will be his existence. Health is Nature's reward for getting into harmony with her laws.

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Leprosy is the classic example of an infectious neuritis. The initial lesion, an innocuous appearing skin macule or papule is hypopigmented and lacks sensation. This stage is often known as indeterminate leprosy, which can evolve in several ways, depending upon the resistance of the host. The bacilli may be locally invasive, producing a circumscribed granuloma that involves the cutaneous and subcutaneous nerves resulting in characteristic hypo-pigmented patch of superficial numbness and sensory loss (Tuberculoid leprosy). If a large nerve in the vicinity is invaded, a sensorimotor deficit in the distribution of that nerve is added to the patch of anaesthesia. In contrast, lack of resistance to organism leads to widespread invasion (Lepromatous leprosy) of cutaneous nerves and produces a symmetrical pattern of pain and temperature loss involving distal extremities- a distribution that is determined by relative coolness of these parts of skin. Extensive sensory loss is followed by impaired motor function owing to invasion of muscular nerves when they lie closest to the skin. There is loss of sweating in the affected parts, otherwise autonomic function is rarely affected.

Disability-When detected and treated early, primary impairments may be reversible. However, 11-51% of patients do not recover. In addition, 33-56% of newly registered patients already have clinically detectable impairments, often no longer amenable to drug treatment. Among new patients, 6-27% present with secondary impairments, such as wounds, contractures and shortening of digits. As the result of impairments, many people experience limitation of activities of daily living. As a result of these limitations, because of visible impairments, or simply because of the diagnosis 'Leprosy', many people are restricted in their participation in society¹. Leprosy interferes with the psychological and social life of the patients leading to their 'dehabilitation'. Therefore, it is necessary to assess the extent and direction of dehabilitation in order to make the treatment plan comprehensive and effective². Leprosy is curable now-a-days if initiatives are taken for prevention of disability along with complete and regular chemotherapy. Common disabilities encountered in leprosy are: Claw hand, foot drop, lagophthalmos, plantar ulcers and depressed bridge of nose Fig.(1). Objectives of prevention of

disabilities are preservation of nerve function, preservation of vision, to regain functional ability and self-esteem³.

Rehabilitation- Rehabilitation of the leprosy patient begins when the disease is diagnosed and continues through out the patient's lifetime. It must be stressed that the most important aspect of the rehabilitation program is the prevention of the anaesthesia and paralysis. This can be achieved by: (a) Early diagnosis, before the nerve is irreparably damaged. (b) Early treatment to forestall nerve damage, (c) Judicious treatment, with great vigilance of patient with borderline leprosy, (d) Prompt recognition and treatment of reactions involving nerves and eyes, and (e) Convincing the patient that he need not be disabled. The common modalities employed for patients with denervated extremities due to leprosy are: hydrotherapy, paraffin baths, oil massage, exercises, biofeedback techniques, electrical stimulation, splinting, sensory and motor re-education, foot care and specialized foot wear, and surgical reconstruction.

Hydrotherapy-Warm water baths: are used to clean and hydrate the skin. The extremity is immersed in warm water (temp

< 42°) for 10-15 minutes, during which it is gently massaged. This softens the keratin and makes the skin pliable.

- Paraffin bath: 9-minutes dip method of paraffin bath at 54° which delivers less heat than 20 minutes whirlpool treatment at 40° is employed. It restores suppleness and flexibility of the skin and is preferred over radiant heat/diathermy as they can cause burns.
- Oil massage: Effects of oil massage are similar to application of paraffin baths. Its added benefit is derived from the manipulation of the skin as it is rubbed (pincement), kneaded (petrissage), and tapped (tapotement).

Exercises-Both passive and active exercises are utilized. Exercises should be started as soon as symptoms of neuritis have subsided. Exercises are contraindicated in the presence of an acute infection, acute neuritic pain or bone and joint pathology. Role of exercises are can be divided in two phases:

- Expectant phase – When there is no sign of recovery. The role of therapy in this phase is to prevent contracture and retain full range of movement, avoid muscle atrophy, and to prevent overstretching of the muscles. This is achieved by passive movements through full range of motion twice daily Fig. (2).

- Active phase – when there is some return of nerve conduction. The aim is to regain as much power and range of movement as possible. This is done by carefully guided strengthening and practicing skilled co-ordinated movements.

Splinting-Splint or orthosis is an external device applied in order to prevent/correct a deformity, support unstable joint, relieve pain, or improve function. They are classified into static or dynamic splints:

- Static splint: It is a rigid device with no moving parts and does not permit movement Fig. (3). This type is more commonly employed in leprosy for following indications: (a) to allay acute pain of neuritis and discomfort following surgery (b) protect and support tissues during process of repair (c) maintain position and alignment during night (d) correction of deformities eg. ulnar drift, swan neck and mallet finger (e) to arrest the progress of contracture particularly of claw hand and (f) immobilize the wrist in “cock up” position so that hand can function more effectively.
- Dynamic splint: It possesses both rigid and mobile components allowing free movements. Dynamic splint is employed primarily during the day for the extremity that needs assistance and

possesses full range of motion Fig. (4). Indications for dynamic splints are: (a) to neutralize deforming forces resulting from muscle imbalance, eg. flexion assist or Wynn Parry splint, (b) strengthen weaker muscle, and (c) as counterforce to correct imbalance.

Sensory re-education- It is based on the presumption that during regeneration of nerves, the patient demonstrates less recovery than he is capable of achieving. This is attributed to the fact that the patient fails to interpret the altered profile of impulses discharged from the damaged nerves. The program is designed to help the patient interpret the altered impulses during the period of recovery by doing simple exercises. The exercises are simple and can be easily performed by the patient. The program is divided into two phases: early and late phase. In the early phase, a touch type of stimulus is applied to the distal phalanx. This serves to promote perception of slow adapting nerve fibers where as moving the object across a given area promotes perception of quickly adapting fibers. In the late phase, touch perception is re-established and the patient is taught to recognize shape by handling various objects.

Motor re-education-When the action of a muscle is weak or lost, it may be initiated by contraction of adjacent muscles that normally participate in the same movement. This principle

is utilized in the re-education after tendon transfers. The process of re-education is based on the ability of the cortical and sub-cortical centers to analyze sensory input and regulate the motor output. Re-education involves two distinct processes: cortical control and automated movements which are independent of cerebral control⁴. A transition from cortical directed movements to the sub conscious level occurs when motor actions are firmly established by repetition, practice co-ordination and skill.

Foot care- The problems of denervated feet are due to loss of sensory perception and motor impairment. Footprint presents a graphic representation of the problem by demonstrating areas of high pressure which are at risks for developing an ulcer. Footprint is taken on a specialized mat designed by Harris⁵ Fig.(5). The denervated foot can be classified into four categories⁶:

- Category I – Low risk foot: The foot is grossly normal, with diminished plantar sensation
- Category II – Moderate risk foot: The foot is grossly normal but has scarring on its plantar surface in addition to being anesthetic
- Category III – High risk foot: The foot posses a flexible deformity in addition to plantar scarring and loss of sensation
- Category IV – Disintegrated foot: The foot is deformed

structurally, is shortened or narrowed due to bone or joint absorption or to amputation.

Insoles of microcellular rubber (MCR) or polyethylene foam are reasonably successful in prevention of ulcers in first three categories of patients Fig. (6). However, they fail to distribute the weight bearing forces evenly, and hence have limited use. Even distribution of forces can be most efficiently be achieved by providing rigidity to the sole as demonstrated by the fact that most ulcers heal when the foot is immobilized in a walking plaster cast. Keeping in view of these factors, a molded plastic footwear was made consisting of hard wearing molded plastic sole and a microcellular sponge insole with a spring steel shank placed in between the two to give controlled rigidity. This foot wear is both acceptable and protective and is recommended for patients in category I, II, & III. The deformed foot of category IV requires custom made foot wear. The shoe must fit the contour of the deformed foot and this can best be done if it is made on a model of the foot. It must have a micro cellular sponge insole and a rigid steel shank running along the entire length of the shoe. These two requisites ensure that the weight of the body is applied evenly on the plantar surface. The disintegrated foot is very difficult to manage. If there is sub-talar destruction or disarticulation of the ankle, it can occasionally be repaired by arthrodesis. If a stable planti-grade foot

reasonably good sole can be obtained by surgery, the foot converts to category III. If not, there is no alternative to amputation.

Reconstructive surgery- The most frequently involved parts of man's anatomy in leprosy are also his most vital tools and his contacts with the world about him, i.e. his hands, feet, and eyes. Every effort must be made to save these parts. Reconstructive surgical procedures can be divided into in to functional and cosmetic. Certain precautions must be undertaken before planning for any surgery: Patient should be eager to co-operate, adequate time should be given for a definitive trial of conservative management, clofazimine should be used at the time reactions as it reduces the need for surgery, and younger patients have better prognosis.

Conclusions- While the deformities of leprosy can be adequately dealt with, total rehabilitation of the patient with leprosy is often incomplete because it fails to treat the patient as a person. It is important to determine whether he is capable of performing activities of daily living, or can he solve problems of interpersonal relations and most important of all, whether he can return to a gainful occupation. A multidimensional approach that takes care of all these features simultaneously is mandatory for total rehabilitation of the patient.

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Figure 1 - Hand deformities

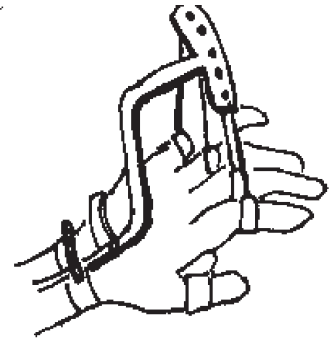


Figure 4 - Dynamic splint Wrist splint with finger loupes

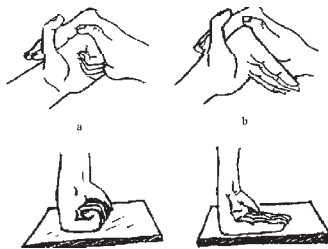


Figure 2 - Hand exercises



Figure 3 - Static splint : Ankle foot orthosis (AFO)

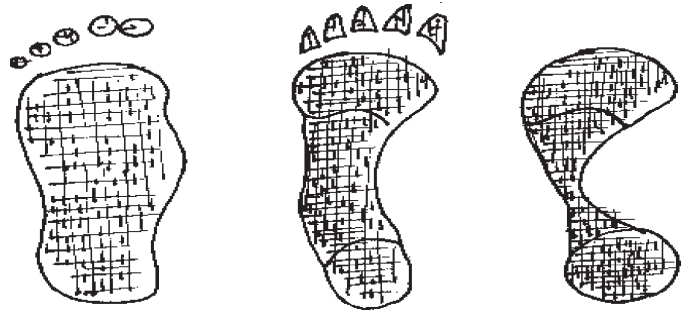


Figure 5 - Foot prints



Figure 6 - Specialized footwear

Basic principles of surgical treatment of Echinococcus

Granulosus infection (Hydatid cyst) of the liver

Commentary

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E. Granulosus or hydatid cyst of the liver is one of the common diseases of liver. The treatment of this benign disease is predominantly surgical. There are various surgical options available including conservative surgeries, radical surgeries and laparoscopic surgeries. There are technical modifications and sub-types in each of them. Indications and basic principles of each surgical option are variable. The decision-making process should consider each option and the best suited for a particular patient should be applied. Human being is an accidental intermediate host of Echinococcus Granulosus. Carnivorous animals (most commonly dog) are definitive host while herbivorous (Sheep, goat, cow etc.) are the natural intermediate hosts. Accidental ingestion of food contaminated by infected dog's faeces will lead to ingestion of eggs of the worm and they develop into larval form as a cyst, most commonly in the liver. This helminthic infestation is one of the common infestations in rural India and usually patients present with complain of dull aching pain or lump in the upper abdomen. In most of the cases the diagnosis is suspected clinically and confirmed by ultrasound of the abdomen.¹ CT scan of the abdomen should ideally be performed in each case to plan

the surgery. Though various serological tests are available they are seldom used clinically.² Surgery is the mainstay of therapy for the hydatid cyst though other conservative treatments like Drug therapies or Percutaneous Aspiration, Injection and Re-aspiration (PAIR) are described in literature and applied clinically in selected group of patients.^{3,4}

Here, we will review certain basic principles and decision-making points for the surgical treatment of hydatid cyst of the liver. This discussion does not include management of hydatid cyst in other organs or disease due to Echinococcus Multilocularis.

Surgical decision-making-The entire decision making process depends on two factors- condition of patient and characteristics of the cyst. Thus, the complete plan of management should be tailor-made for each individual patient.

Whether surgery is needed or not?-Surgery is either not required or not advisable in conditions such as-severe cardio-respiratory instability or other severe systemic disease, Less than 4 cm sized deep parenchymal single cyst and completely calcified cyst.

Which surgery should be performed-Surgery for liver hydatid can be broadly divided

into conservative surgeries or radical surgeries. Now, option of laparoscopy is also available, So decision-making process should also include it for the benefit of patient. (Figure 1)

Conservative surgery for liver hydatid - By definition, conservative surgery for hydatid cyst is a surgery in which cyst cavity entered electively while radical surgery is a surgery in which cyst cavity is ideally not entered. There are three basic aims of conservative surgery⁵: Cyst evacuation and prevention of peritoneal implantation, management of cyst biliary communication, and management of residual cyst cavity.

Cyst evacuation and prevention of peritoneal implantation-The abdomen is most commonly assessed through either sub-costal (Chevron) or sometimes midline incision. Abdominal cavity is thoroughly explored for unidentified cyst on preoperative imaging. The liver is bimanually palpated after complete careful mobilization. Intra-operative ultrasound may identify intra-hepatic cysts that are not identified on pre-operative imaging. It may show relation of the cyst with billio-vascular structures and hydatid debris in common bile duct (CBD). Wound and exposed peritoneal

surfaces are protected with green or blue coloured towels soaked in scolicidal solution to identify any spillage of daughter cyst. Cystic fluid is evacuated by large bore needle and suction tube. The cyst is then opened after taking stay sutures. Daughter cyst, laminated membrane and hydatid sand are all evacuated completely. Cavity is rinsed with 5% saline. Cyst roof is excised and edges are oversewn with absorbable sutures to prevent bleeding. Exocyst and daughter cysts in the crevices are carefully searched and excised. Cyst cavity is packed with white gauze to see for biliary staining. A variety of solutions are available as a scolicidal agent. At present 20% saline, 0.5% Cetrimide, 1% Povidone iodine, 10% Chlorhexidine or rarely 0.5% silver nitrate are used in practice.⁶ These solutions are more or less equal in efficacy and toxicity especially biliary injury. These solutions should not be injected blindly into the cyst cavity or if the cyst fluid is stained with bile.

Management of bile-duct communication-Preoperatively it is difficult to suspect bile duct communication unless there is history of recurrent cholangitis or ERCP proven hydatid debris in CBD. Intra-operatively bile stained fluid may suggest communication but sometimes it is difficult to identify it. Dead or calcified cyst may have bile duct communication. To search for bile duct communication the easiest way is to look meticulously and to pack the cavity by gauzes as described above. Other methods include intraoperative

cholangiography, Methylene Blue injection etc. On detection of such communication it must be repaired. The choice of repair depends on the level of biliary channel involvement as shown in Figure 2.

Management of residual cyst cavity-There are multiple options available to manage the residual cavity. Indications for each technique can be summarized as follows:

- Small, non-calcified, superficial cyst- suture close the edges after filling the cavity with normal saline.
- Large, shallow, superficial saucer like cavity- can be left open
- Deep cavity with redundant crevices- omentoplasty.
- Deep cavity and omentoplasty not possible- captonnage (imbricating the cyst wall into the depths of cavity).

Radical surgeries for liver hydatid⁷ - There are five types of radical surgeries in which cyst cavity is not entered. Multiple exogenous cysts or calcified cyst or suspected biliary communication can be treated by closed total cystopericystectomy in which a plane is developed between adventitia and liver parenchyma. This surgery is not possible when major vascular structures are abutting the cyst. If the adventitia is very thin or cyst ruptures during closed pericystectomy or a major vascular structure is abutting the cyst wall then the cyst is opened, debrided and pericystectomy is performed. Non-anatomical resections are reserved for

pedunculated peripheral cysts. Formal anatomical resection is performed only when no other surgical options are available such as recurrent disease, irreparable bile duct communication. Liver transplantation is very rarely indicated in patients in whom there is recurrent disease after multiple surgeries and has end stage liver failure.

Conservative surgery Vs Radical surgery^{8,9}-A general surgeon, in a relatively small center with less specialized equipments and skills can perform conservative surgeries for liver hydatid cyst. These surgeries are usually associated with less morbidity and mortality if carefully performed with acceptable recurrence rates. Limitations of conservative surgeries include need for entering cyst cavity and possible spillage, use of scolicidal agents and its potential hazards. Against these possible limitations of conservative surgeries, the radical surgery avoids spillage and use of scolicidal agents. Biliary communication can be managed well with radical surgeries and probably there are lesser chances of recurrences. The main limitation of radical surgeries is limited availability of surgical expertise and infrastructure especially in countries where hydatid cyst is more common. Morbidity and mortality may be little higher with radical surgeries in less experienced hands.

Laparoscopic surgery for hydatid cyst-With technological advancement in laparoscopy, it has become possible to manage hydatid cyst of liver by advanced

laparoscopic techniques.¹⁰ Selection of patient for the laparoscopic management is crucial. The guiding criteria for such selection are-Primary cyst, Superficial cyst, Less than 3 in number less than 15 cm in size and, No intra-biliary rupture, Proficiency of surgeon and set up for advanced laparoscopic surgery; At present only conservative surgeries can be performed by laparoscopy. Bile duct communication is not a contraindication to surgery.

Conclusion-E. Granulosus infection of liver is a common surgical disease. There are variety of surgical procedures and techniques available. Selection of proper technique and its proper implementation is crucial for long-term cure. The decision to choose a particular technique should be tailor-made for an individual patient.

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Osteoporosis is a significant public health problem. Vertebral fractures are the most common fractures in patients with osteoporosis, along with proximal femoral and wrist fractures. These fractures may cause acute or chronic pain, reduce the quality of life, and shorten life expectancy. Several medications are available that reduce the risk of fracture. Vertebroplasty may reduce or relieve pain in carefully selected patients. Osteoporosis is “a systemic skeletal disease characterized by low bone mass and micro-architectural deterioration with a consequent increase in bone fragility with susceptibility to fracture.” In simpler terms, it is a reduction in the quantity and quality of bone that leads to increased bone fragility and fractures. Osteoporosis is a silent disease — there are no symptoms or signs of low bone mass or bone loss. Typically, years or decades of bone loss are required before an individual becomes at high risk for bone fracture. Once the patient experiences a fracture, more fractures are likely to occur in the near future. Vertebral compression fractures are the most common fractures in patients with osteoporosis, after proximal femoral and wrist fractures. They are identified in nearly 25% of women aged

above 50 years. The incidence increases by about 15% with each passing decade. These fractures are nearly twice as common in females as compared with males. For reasons not clearly understood, only one third of spinal compression fractures are painful; most of these are refractory to medical management. A number of diseases and conditions predispose individuals to osteoporosis and secondary vertebral compression fracture. These include - Advanced age, Asthma, Cirrhosis, Diabetes Mellitus, Emphysema, Menopause, Oophorectomy, Bilateral Renal disease, Chronic Rheumatoid Arthritis, Transplants, Tumors, Parathyroid-related peptide Vitamin D deficiency. In addition, certain drugs are also associated with osteoporosis, such as - Anticonvulsants, Cytotoxic drugs, Alcohol, Thyroid replacement drugs, Steroids, Heparin

Patients with compression fractures typically present with a sudden onset of intense back pain, often after a relatively benign activity. Many patients refer to intractable pain after a sneeze or a cough. The pain tends to be debilitating. Patients find it difficult to find a comfortable position, and therefore, they have difficulty sleeping. Many patients

refer to sleep in a seated or semireclining position. Though majority of vertebral fractures eventually heal with conservative management some of them will fail to heal giving rise to chronic pain. This chronic pain is due to changes in spinal alignment, spasm of paraspinal muscles, and stretch on posterior spinal ligaments. Multiple compression fractures lead to kyphotic deformity (“dowager’s hump”), loss of height, crowding of internal organs, inactivity-induced physical deconditioning, and changes in self-image leading to a significant impact on self esteem and activities of daily living. Biomechanically with each vertebral fracture there is forward shift of weight bearing axis, leading to increased load on the anterior column of weak osteoporotic vertebrae predisposing them to further fractures. Relatively new procedure, vertebroplasty have been introduced for the management of unusually severe or persistent pain from vertebral fractures. It involves percutaneous injection of bone cement into one or more fractured vertebra. Pain relief has been reported in 60 to 100% of cases in which this procedure was performed.

Vertebroplasty-In 1984, vertebroplasty was first successfully performed in France

for the treatment of a cervical vertebral hemangioma (Deramond, 1998). Since then, the application of vertebroplasty have been expanded to include the treatment of the intense pain caused by vertebral compression fractures that is refractory to conventional therapies. Vertebroplasty involves the injection of acrylic cement under local anesthesia and either fluoroscopic guidance or, less commonly, CT guidance into fractured vertebra. Typically, the techniques are performed as an outpatient procedure and require approximately 40 minutes per level treated. Pain reduction or elimination is immediate, and the risk of complication is low. Vertebroplasty is a treatment for pain. Theoretically, 2 mechanisms may account for the pain reduction associated with the injection of methylmethacrylate. The first mechanism may be as a result of acrylic fusion of the fragments into a single block, preventing the painful motion of the individual fracture fragments against each other. The second mechanism of pain reduction may be related to the heat produced by the polymerization process as the acrylic hardens. An added benefit is that deposition of acrylic within the vertebra significantly strengthens osteoporotic bone, reducing the likelihood of repeat fracture.

Patient selection - A patient with painful osteoporotic compression fracture who has failed to respond to a fair and adequate trial of conservative treatment is the ideal candidate for vertebroplasty. Impending

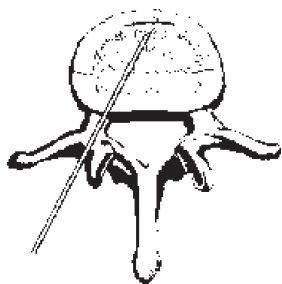
vertebral fracture due to localized osteoporosis following non-septic lesion such as hemangioma, multiple myeloma may be additional indications. Vertebroplasty may also be applied prophylactically to an at-risk vertebra between 2 other abnormal vertebrae. Inclusion criteria include -Fracture less than 12 months old, Pain localized to a fracture or tumor, and Pain refractory to medical management. The exclusion criteria include - Fracture extending to posterior vertebral cortex , Retropulsed fragment, Cord compression, Radiculopathy, Fever and/or sepsis, Coagulopathy.

Technique-Vertebroplasty is a day-care procedure that is performed on an outpatient basis. The procedure is not painful and requires only mild sedation and analgesia. Occasionally, patients report pain when the trocar reaches the fracture fragments and when the injection of acrylic cement is initiated. However this can be tolerated by most patients without much discomfort. In an occasional uncooperative patient general anaesthesia may be indicated. Lumbar vertebroplasty consists of the transpedicular placement of an 11-gauge bone biopsy needle into the affected vertebra under fluoroscopic or CT guidance. Thoracic vertebroplasty is performed via 13-gauge bone biopsy needle. Once positioned, methylmethacrylate is injected through the needle into the abnormal vertebral body. The acrylic is then prepared under sterile conditions for injection.

The dry, powdered polymer is mixed with a liquid monomer of methylmethacrylate to a consistency similar to that of toothpaste. The acrylic cement is then injected with either a 1-mL Luer-Lok syringe through the trocar. Injection is continued until complete opacification of the vertebral body is achieved or the first sign of extension into the epidural venous plexus appears. Opacification of the paraspinal veins is common. When opacification occurs, the injection of acrylic is suspended for approximately 1 minute to allow the cement to harden within the vein. Injection may then be resumed, and the acrylic follows a new path of lower resistance. Opacification of the vertebral body need not be complete for successful vertebroplasty. If the acrylic reaches both the superior and inferior endplates and extends across midline, approximately 80% of the load-bearing benefit of a completely opacified vertebral body is achieved (Belkoff et al). A small amount of methylmethacrylate is retained on the bench as a control. After approximately 10 minutes, the cement solidifies and becomes harder than the native bone. Once the control sample has solidified, the Patient is examined for neurological deficit before being transferred to a post-operative ward.

Risks -The risks of the procedure are low, but they potentially include infection, worsening of pain, and neurological problems such as weakness or pain in the legs. Occasionally, the acrylic may

extend into the epidural or paraspinous veins. Cement in the epidural venous plexus may lead to an ascending venous thrombosis or contribute to a spinal stenosis or cord or nerve root compression. Acrylic may extend from the paraspinous veins into the vena cava and may result in a pulmonary embolus. The risk of venous embolization increases if the operator cannot adequately identify when the cement begins to pass into the venous system. This risk is reduced by using angiographic equipment with the highest resolution available. The visibility of methylmethacrylate is further improved with the addition of fine metallic powders such as barium mixed with tantalum or tungsten.



Transpedicular placement of a trocar in the anterior third of the fractured vertebral body

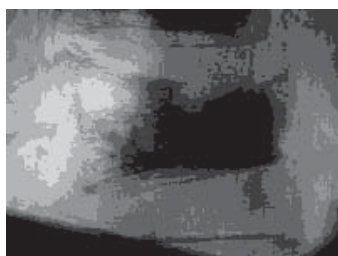


Lateral radiograph of fractured vertebra shows the initial placement of the trocar

Results -Approximately 85-90% of patients have rapid pain relief. This procedure is associated with a low morbidity rate. Fewer than 1% of patients with have morbidity. Morbidity may include local pain, rib pain, spinal stenosis, nerve root compression, and intravascular extension of acrylic. In approximately 90% of patients there is complete resolution of pain. Incisional and muscular pain may persist for the first few days after the procedure and it can be controlled with adequate oral anesgesics. Point tenderness that is noted before the procedure has not been noted in any of the patients after the procedure. Although pain is reduced or eliminated after the procedure, patients must exercise caution in subsequent activities



Injection of methylmethacrylate through the trocar in anterior one third of the vertebral body.



Compression fracture after vertebroplasty.

because other osteoporotic vertebral bodies may also be prone to fracture. Medical management of the underlying disorder that weakens the vertebral bodies should be initiated. This procedure does not eliminate the need for aggressive treatment of osteoporosis, without which other fractures may ensue.

Conclusions-Osteoporotic vertebral fractures are common in elderly. These fractures reduce a patient's quality of life and shorten life expectancy. Several available medications have been shown to reduce the risk of fracture. In patients with unhealed compression fractures vertebroplasty may reduce or relieve pain in selected patients.

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Development, structure and function of placenta, umbilical cord and amniotic fluid

Review Article

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The fetus is entirely dependent on its maternal support for growth and development. The link between the two is the indispensable placenta. The two arms of fetal-maternal communication system of human pregnancy are- Placental arm and Fetal membranes (amnion and chorion laeve). The placental arm of this system links the mother and fetus such that at all the sites of cell to cell contact, maternal tissues are juxtaposed to extra embryonic cells and not the embryonic or fetal cells. Before we discuss about the development of placenta it is important to mention about the endometrial preparation for receiving the developing embryo.

The decidua

Endometrium of pregnant uterus is decidua. It is entitled so because it is shed off at the time of delivery. The increased structural (vasculature) and secretory activity of the endometrium following implantation is known as decidual reaction which is most marked at the implantation site. The fibrous connective tissue of the stroma is converted to epithelia cells. The cells are enlarged and are placed back to back with clear vesicular cytoplasm. The glands are markedly enlarged and become increasingly tortuous. Small interstitial haemorrhages and leucocytic infiltration occurs, particularly at the implantation site. Decidua differentiates into

three layers-Superficial compact layer-compact mass of decidual cells, gland ducts and dilated capillaries; Intermediate spongy layer-dilated uterine glands, decidual cells and vessels. This layer forms the plane of cleavage when placental separation occurs; Basal layer-basal portion of glands. Regeneration of mucosa occurs from this layer.

Fertilization and cleavage

Fertilization of the ovum occurs in the fallopian tube. After fertilization, mature ovum becomes zygote-a diploid cell with 46 chromosomes. Zygote undergoes mitotic division yielding blastomeres while still inside fallopian tube and is surrounded by zona pellucida. Blastomeres continue to divide until solid ball of 12-16 cells (morula) is formed. Morula enters the uterine cavity about 3 days after fertilization. Zona pellucida persists even at 58- cell stage.¹ Fluid gradually accumulates in morula leading to formation of blastocyst. Compact mass of cells at one pole of blastocyst is called inner cell mass which later on forms embryo. The outer mass of cells forms trophoblasts.

Implantation

Implantation most commonly occurs on the upper part and the posterior wall of the uterus. Zona pellucida disappears and blastocyst touches and adheres to the endometrial surface at 108-256 cell stage. Trophoblasts

burrow into the stroma between endometrial cells till the blastocyst becomes encased within the endometrium. This type of implantation is called interstitial implantation. It starts on 6th day and is completed by 11th day. Process involves degradation of extracellular matrix by urokinase type plasminogen activator and metalloproteinases.²

Embryonic development

At around 7- 8 days of fertilization, inner cell mass differentiates into primitive ectoderm and underlying layer of endoderm. The embryonic mesenchyme first appears as isolated cells within the blastocyst and later completely line the cavity, forming chorionic vesicles. Its membrane is now called the chorion. Near the embryo mesenchymal cells condense to form the body stalk, which serves to connect the embryo to chorion. After implantation of blastocyst, the different parts of decidua become as follows: decidua basalis- in contact with base of blastocyst, decidua capsularis- thin superficial layer covering the blastocyst and Decidua parietalis- rest of the decidua lining the uterine cavity. its thickness gradually increases to 5-10mm at the end of second month and then regresses to 1mm by the end of 20 weeks. As the fetus grows, the deciduas capsularis fuses with the deciduas parietalis forming decidua vera. Deciduas capsularis is largely lost

by pressure and loss of blood supply.

Trophoblast

Trophoblast is the most important component of the placenta that is involved in invasion of blastocyst, immunological acceptance of the conceptus, nutritional support to the conceptus and producing hormones for the maintenance of pregnancy. Morphologically and functionally trophoblast differentiates into cellular and syncytial forms. During implantation some of the inner most cytotrophoblast contiguous with endometrium coalesce to form amorphous multinucleate membrane, the syncytium. This depends on protein synthesis, calcium dependent cell adhesion molecule E cadherin and desmosome formation.^{3, 4} Functionally, cytotrophoblast is the germinal layer and syncytium is the secretory layer.

Placental development

After adherence to the endometrium the cytotrophoblast towards the embryonic pole proliferate rapidly and invade decidua. As the blastocyst grows, the other pole of the mass extends towards endometrial cavity (abembryonic pole). As the trophoblast invade endometrium, small vacuoles appear in the syncytiotrophoblast which later coalesce to become lacunae.⁵ These lacunae get filled with the maternal blood from the endometrial arteries (spiral arteries) with invasion of maternal vessels.

Cytotrophoblast invasion of decidual vessels -

Cytotrophoblast initially invade the superficial capillaries of the

endometrium. Subsequently, the arterioles and then the spiral arteries are invaded. Cytotrophoblast can pass several centimeters along the vessels and have even been noticed in the myometrial portions of the vessels⁷ by around 16 weeks. Invading cytotrophoblasts form a lining within the endothelium. With invasion, degenerative changes occur in the vessel wall, most markedly affecting the smooth muscle cells. Gradually connections are established between lacunae and veins of endometrium. The lacunae join each other⁶ to form labyrinths around villi. This type of placentation is called hemochorioendothelial placentation.

Chorionic villi-Late in the second week of development, delicate columns of cytotrophoblasts extend out between adjacent blood filled lacunae in the syncytiotrophoblasts. These are known as primary villi. Initially, villi form over the entire surface of chorion, later only those on the most deeply implanted aspect of the conceptus (overlying deciduas basalis) persist. The chorionic villi on the deciduas capsularis gradually atrophy from pressure and become converted into chorion laeve by third month that lies between amnion and deciduas. Cells of the extra embryonic mesoderm lining the inner aspect of the trophoblast enter the primary villi and create a core, resulting in the formation of secondary villi (16th day). Each villus branches and attains complex form at the end of third week blood vessels develop within the core of the villus and

connect to the circulatory system of the embryo. With this forms the tertiary villus by 21st day of fertilization. Each villus has embryonic blood flowing inside it and maternal blood bathing its outer surface. Some of the tertiary villi extend completely such that cytotrophoblast in central core make contact with decidual cells, these are called anchoring villi. At the undersurface of endometrium, cytotrophoblastic shell is formed as the cytotrophoblast spread laterally from the end of each villus.

Structure of a terminal villus-from outside inwards each terminal villus has. Outer syncytiotrophoblast, Cytotrophoblast, Basement membrane, Central stroma with fetal capillaries, mesenchymal cells and connective tissue.

Placental barrier

Despite close proximity there is no mixing of the maternal and fetal blood because the two are separated by so called placental barrier. This comprises syncytiotrophoblasts, cytotrophoblasts, the basement membrane, stromal tissue, and endothelium of the fetal capillaries. Its thickness is approximately 0.002 to 0.006 mm.⁸

Placental cotyledon (Figure 2)

As the placenta matures, the stem villi branch repeatedly to form progressively finer subdivisions. Each mainstem alongwith its ramifications form placental cotyledon or lobe. Each of it is supplied with a branch of chorionic artery and is drained by one vein. These cotyledons

continue to grow till term but their number remains the same throughout gestation.⁹

Structure of placenta

Mature placenta is discoid shaped, measuring about 185mm in diameter and 23mm in thickness at centre, weighs about 500 gms and has average volume of 497 ml.¹⁰ Of the two surfaces of placenta, fetal surface is covered by amnion giving it smooth appearance, and has umbilical cord attached at or near centre. The placenta is limited internally by the amniotic membrane and chorionic plate, and externally by the basal plate. In between the two lie the villi and intervillous spaces.

Chorionic plate

It consists of primitive mesenchyme with branches of umbilical vessels, layer of cytotrophoblast and syncytiotrophoblast. It is formed by 8- 10 weeks.

BASAL PLATE: from outside inwards it consists of decidua basalis, Nitabuch's layer, cytotrophoblastic shell, and syncytiotrophoblast. Branches of umbilical vessels are visible beneath amnion as they radiate from cord insertion. On maternal surface cotyledons appear as slightly elevated areas called lobes, giving it rough and spongy appearance. These lobes are separated by grooves of variable depth occupied by decidual septae.

Placental circulation

Maternal circulation- it concerns with the circulation of maternal blood through the intervillous space. In mature placenta volume of blood in intervillous space is

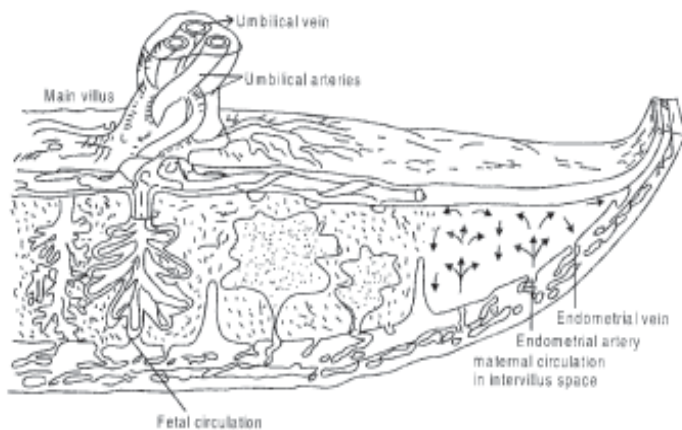
150ml and blood flow is 500- 600 ml per minute. Fetal homeostasis is dependent on an efficient maternal placental circulation. Maternal blood enters intervillous space via spiral arteries that enter the space by perforating basal plate. Blood is driven high up towards the chorionic plate by the head of maternal arterial pressure. Then it spreads laterally and bathes the external microvillus surface of chorionic villi. It is pushed by the pressure of continuous influx of arterial blood towards exits in the basal plate, from which it is drained by uterine veins.¹¹ Cytotrophoblastic invasion of vessels ultimately stops the circulation in some of these vessels and thus reducing the number of arterial openings. During uterine contractions, intervillous space distends with blood because venous outflow is reduced more than arterial inflow. Thus even at reduced rate of flow, increased volume of blood is available for exchange¹². So, uteroplacental circulation is regulated by arterial blood pressure, intrauterine pressure, and pattern of contractions.

Hemodynamics of placental circulation

Volume of blood in intervillous space	150 ml
Uterine artery pressure	70- 80 mmHg
Uterine vein pressure	8 mmHg
Pressure in intervillous space	
During contraction	30- 50 mmHg
During relaxation	10- 15 mmHg

Fetal circulation

Venous blood from fetus reaches placenta through two umbilical arteries. After traversing through the umbilical cord, umbilical vessels branch repeatedly beneath the amnion (surface or chorion vessels) and in the villi, finally forming capillaries. Immediately after entering the chorionic plate, the two umbilical arteries are joined by a transverse connection, the Hyrtl anastomosis. The truncal arteries are the perforating branches of the surface vessels that pass through the chorion plate. There is one truncal artery for each cotyledon. Maternal and fetal blood flow side by side but in opposite direction facilitating material exchange between the



two. The villus capillary pressure varies between 20- 40 mmHg. Oxygenated blood returns from placenta to fetus through single umbilical vein.

Placental ageing

As the pregnancy advances, placenta undergoes some degenerative changes. Ageing process involves both fetal and maternal aspects of placenta. This physiological process should be differentiated from the morbid changes that occur in some pathological conditions.

Syncytium thins out and at some places syncytial knots appear by aggregation of syncytium.

Volume of cytotrophoblasts reduces. Vessels become prominent and come close to the surface

Basement membrane of capillaries thickens. Few blood vessels are obliterated by the deposition of fibrin. There is decrease in stromal tissue 'Hofbauer cells' and fetal macrophages appear in the stroma. In the decidua, there is appearance of fibroid deposit called Nitabuch's membrane in the outer syncytiotrophoblast layer adjacent to the decidua. This membrane limits the further invasion of decidua by the trophoblast.

The syncytium covering the villi and extending into the intervillous space undergoes fibroid degeneration and form a mass entangling variable number of villi. These are called as white infarcts. Further, calcifications may occur on this.

Placental function

Apart from nidation and transfer of nutrients, placenta has

indispensable metabolic, endocrine and immunological roles to play.

Transfer of nutrients-The transfer function of placenta is dependent on physical properties of substance, integrity of placental membrane, rate of fetal and maternal blood flow.

Transfer of oxygen across placenta is blood flow limited and that of carbon dioxide is diffusion limited. Oxygen supply to the fetus is continuous.

Dynamics of gas exchange across placenta¹³

	Uterine artery	Uterine vein	Umbilical artery	Umbilical vein
pO ₂ mmHg	95	40	20-25 mmHg	30-40 mm Hg
pCO ₂ mmHg	32	40	48	43

Even at low pO₂, fetus compensates because of higher cardiac output, higher oxygen carrying capacity of fetal blood and more Hb level. Transfer of CO₂ is facilitated by high permeability of chorionic villi, less affinity of fetal blood for CO₂ and lower partial pressure of CO₂ in maternal blood due to pregnancy induced mild hyperventilation.

Excretory function- Waste products like urea, creatinine and uric acid are excreted to maternal blood by simple diffusion.

Nutritive function-Glucose which is the principal source of energy is transferred to the fetus by facilitated diffusion via GLUT 1 and GLUT 3 transport proteins^{14,15}. Free fatty acids cross the placenta by simple diffusion. Amino acids are transferred by active transport by enzymatic mechanism. Some proteins cross by the process of pinocytosis.

Iron is actively transported across placenta, rate of transfer steadily increasing with gestation.¹⁶

Barrier function-Fetal membranes act as a protective barrier against noxious agents in the maternal blood. However certain maternal infections and drugs used in pregnancy can cross the placental barrier.

Enzymatic function-Numerous enzymes are elaborated in the placenta like diamine oxidase (inactivates pressor amines),

oxyocinase (neutralizes oxytocin) and phospholipase A₂ (synthesizes arachidonic acid).

Endocrine function-The human placenta synthesizes an enormous amount of protein and peptide hormones like human placental lactogen, human chorionic gonadotrophin, ACTH, parathyroid hormone related protein, calcitonin, relaxin, thyrotropin releasing hormone, somatostatin, corticotrophin releasing hormone, inhibin, activin and atrial natriuretic peptide. Syncytiotrophoblast takes up maternal plasma low density lipoproteins (LDL) for progesterone biosynthesis.^{17, 18} The de novo synthesis of cholesterol in trophoblast is minimal. Hence it depends on maternal plasma for progesterone biosynthesis.¹⁹

Placenta cannot synthesize estrogens de novo because it lacks

17 hydroxylase/17-20 desmolase
It has high capacity to
convert C 19 steroid to estrone
and estradiol 17 β because of
placental aromatase enzyme.
20 Rich sulphatase activity
of placenta allows it
to convert maternal
dehydroepiandrosterone sulphate
to estradiol 17 β .²¹

Amnion and amniotic fluid

It is the innermost avascular fetal membrane. It provides tensile strength to the fetal membranes. Development -On 8th day of fertilization inner cell mass differentiates into bilaminar germ disc which consists of dorsal ectodermal layer and ventral endodermal layer. It is connected with the trophoblast by mesenchymal condensation called connecting stalk that later on forms umbilical cord ⁶. Two fluid filled spaces appear on each side of germ disc. The space on the dorsal aspect (between ectoderm and cytotrophoblast) is called amniotic cavity. The cavity which appears on the ventral aspect of bilaminar disc is known as yolk sac.⁶ It is lined externally by mesenchyme and internally by endodermal cells migrating from the germ disc. The epithelial cells lining the amniotic cavity are derived from fetal ectodermal cells. A layer of fibroblast like (mesenchymal) cell derived from embryonic mesoderm line the outer aspect of epithelial cells, thus constituting a two layered amnion. Simultaneously, interstitial collagens I, III and V are deposited between these two layers of cells. The amniotic sac expands to envelop the placenta and the chorion. Reflected amnion is fused to the chorion leave. The epithelial cells of

amnion replicate faster than mesenchymal cells. Also, there is progressive reduction in the compactness of mesenchymal cells. At term, the epithelial cells form a continuous uninterrupted lining but mesenchymal cells are widely dispersed.

Structure of amnion²²

Amnion consists of five layers

- Inner layer of cuboidal epithelial cells derived from ectoderm of embryonic disc
- Basement membrane
- Acellular layer of interstitial collagens
- Row of mesenchymal cells
- Outermost acellular zona spongiosa

Functions-Amnion is not merely an avascular layer. It is metabolically active and is involved in solute and water transport to maintain amniotic fluid homeostasis. Amnion epithelial cells are the cells that are usually referred to and studied most commonly in amniotic fluid. These are the site of transfer between amnion and amniotic fluid. They are metabolically active and are site of synthesis of tissue inhibitor of metalloproteinase- 1.23 Mesenchymal cells are responsible for majority of amniotic functions, like synthesis of interstitial collagens,²⁴ and synthesis of cytokines like IL-6, IL-8 and MCP-1.²⁵ Interstitial collagens are almost exclusively responsible for the tensile strength of amnion

Amniotic fluid

Origin of amniotic fluid- the precise origin still remains unsolved. It is thought to be of mixed fetal and maternal origin^{26,27}. During first half of pregnancy, amniotic fluid is mainly formed by transudation

across fetal membranes and fetal skin (before keratinization). In second half, significant contribution in regulating volume and composition is from fetal urine, tracheobronchial secretions and swallowing of amniotic fluid by fetus.

Volume-It is around 200 ml at 16 weeks, 1 litre at 28 weeks; thereafter it reduces such that at term it is 800 ml, and reduces to only 200 ml at 42 weeks.²⁸

Composition-Amniotic fluid is slightly alkaline with low specific gravity. In early pregnancy; the composition is similar to extracellular fluid. Later, it is altered by addition of fetal urinary metabolites. Water is the main constituent accounting for 98-99% of composition, rest 1- 2% is solid component. The solid components include proteins, glucose, urea, uric acid, creatinine, lipids and electrolytes. Various suspended particles include lanugo, exfoliated epithelial cells, vernix caseosa, cast off cells from respiratory tract etc.

Functions-Amniotic fluid acts as a shock absorber during pregnancy, maintains even temperature

Along with fetal membranes, it helps in cervical dilation during labour. It also acts as lubricant and gives freedom of movement to the fetus.

Umbilical cord

Development-At first embryo is interposed between amnion and yolk sac. The dorsal surface of the embryo grows faster than the ventral surface, the embryo bulges into the amniotic sac and the dorsal part of the yolk sac is incorporated into the embryo to form the gut. Later yolk sac becomes smaller. The allantois projects into the base of the body

stalk from the caudal part of yolk sac. In third month of pregnancy, the expanding amnion obliterates exocoelom, fuses with chorion, engulfs the embryo and covers the placenta and body stalk. Umbilical cord is developed from the body stalk. Initially it is attached to the caudal end of the embryonic disc but due to cephalocaudal folding of the embryo²⁹ and simultaneous enlargement of the amniotic cavity; it comes to lie on the ventral aspect of the fetus. As the amniotic cavity becomes filled with fluid, the embryo is carried more and more into the cavity with simultaneous elongation of body stalk, the future umbilical cord.

Structure-The constituents of umbilical cord are

- Epithelium -It is single layer of amniotic epithelium.
- Wharton's jelly-it consists of elongated cells in a gelatinous fluid formed by mucoid degeneration of the extra embryonic mesodermal cells.
- Blood vessels- Initially there are 4 vessels-2 arteries and 2 veins. The arteries are derived from the internal iliac arteries of the foetus and carry the venous blood from the foetus to the placenta. The right umbilical vein disappears by the 4th month. Presence of a single umbilical artery is often associated with foetal congenital malformation.
- Remnant of the umbilical vesicle and its vitelline duct-remnant of the yolk sac may be found as a small yellow body near the attachment of the cord to the placenta.
- Allantois-A blind tubular structure present near the fetal end.
- Obliterated extra embryonic

coelom.

Characteristics-The umbilical cord is 50 cm long with usual variation of 30-100 cm. Its thickness is not uniform at paces but present knot in its entire length. The umbilical arteries do not possess internal elastic lamina but have got well developed muscular coat. Both the arteries and vein do not possess vasavasorum.

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Anatomy of fetus
Anatomy of the fetus refers to the general structure of the body of the fetus. Clinical application: Knowledge of the fetal anatomy is essential for targeted ultrasounds for congenital anomalies. Transabdominal and transvaginal ultrasounds have given us an in-depth understanding of normal fetal anatomy during the various stages of development. Today ultrasound can achieve fetal anatomy assessment including visualization of the skull, brain, face, spine, four-chamber and three-vessel views of the heart, stomach, abdominal wall, kidneys, bladder and extremities. Various studies have shown that the best time for ruling out congenital anomalies is 10-14 weeks.^{1,2} This is especially important in high risk pregnancies with a previous anomaly in order to reassure the mother. However, the detection rate for structural anomalies in the first trimester varies from 33 to 64.7% while that in the second trimester is between 17 to 85%. Hence a first trimester scan cannot replace an anomaly scan between 18 to 24 weeks in low risk population³.

Fetal growth and development
Estimation of gestational age- Gestational age or menstrual age is the time elapsed since the first

Gestational age in weeks at which fetal organs can be visualized in >70% of fetuses by transabdominal and transvaginal ultrasound³

Fetal Structure	Transabdominal	Transvaginal
Cranium	11-13	11-12
Spine	12-13	11
Long Bones	12-13	10-11
Feet	12	13
Four-chamber Heart View	12-13	12
Kidneys	12-13	11-12
Bladder	12-13	13
Anterior Abdominal Wall	12-13	12
Face	12-13	12
Stomach	13	11-12

day of the last menstrual period. This is usually 2 weeks before ovulation and fertilization. Embryologists describe embryo fetal development in ovulation age or the time in day or weeks from ovulation. Gestational age can be calculated from the last menstrual period. Dating of pregnancy is required in high risk situations for proper assessment of growth and iatrogenic termination in maternal and fetal interest.

Gestational dating by mean sac diameter in the early first trimester⁴

Mean Sac Diameter (mm)	Gestational Age (wks)
2	5.0
3	5.1
4	5.2
5	5.4
6	5.5
7	5.6
8	5.7
9	5.9
10	6.0

Gestational dating by ultrasound in the first trimester⁴

Sonographic findings	Gestational Age (Weeks)
Gestational sac, no yolk sac, no embryo or heart beat	5
Gestational sac with yolk sac, no embryo or heart beat	5.5
Gestational sac with heart beat and embryo <5 mm in length	6
Embryo/Fetus ≥5 mm in length	Age based on crown-rump length

Approach to gestational age assignment by ultrasound on initial scan ⁴

State of pregnancy	Basis for GA	Accuracy (wks)
First trimester Early (5-6 wks) Mid-to-late (6-13 wks)	Sonographic milestones CRL	±0.5 ±0.5
Second trimester If OFD available If OFD not measurable	BPDc or HC BPD or FL	±1.2 (14-20 wks) ±1.9 (20-26 wks) ±1.4 (14-20 wks) ± 2.1-2.5 (20-26 wks)
Third trimester If OFD available If OFD not measurable	BPDc, HC or FL FL	±3.1-3.4 (26-32 wks) ±3.5-3.8 (32-42 wks) ±3.1 (26-32 wks) ±3.5 (36-42 wks)

OFD- occipito frontal diameter, HC – Head circumference, BPD- Bipareital diameter, FL – femur length, BPDc- corrected BPD

Fetal growth-Fetal growth has been divided into three consecutive cell growth phases ⁵. The initial phase of hyperplasia occurs during the first 16 weeks and is characterized by rapid increase in the cell number. It corresponds to a growth rate of 5 gm per day at 15 weeks. The second phase includes both cellular hyperplasia and hypertrophy and extends up to 32 weeks. The growth rate is 15- 20 grams / day at 4 weeks. After 32 weeks fetal growth occurs via cellular hypertrophy and growth rate is 30 to 35 gms per day at 34 weeks. Most fetal fat and glycogen deposition takes place during this phase. In early fetal life the major determinant of fetal growth is the fetal genome. Later in pregnancy environmental, nutritional and hormonal influences become increasingly important.

Fetal Nutrition-Maternal diet is translated into storage forms that are made available to meet the demands for energy, tissue repair and new growth including maternal needs for pregnancy.

Glucose: Glucose is a major nutrient for fetal growth and energy. Human Placental lactogen, a hormone present in abundance in the mother blocks peripheral uptake of glucose while promoting the mobilization and use of free fatty acids by maternal tissues. This ensures glucose supply to the fetus. D-glucose is transferred across cell membranes through carrier mediated, stereo specific, non concentrating process of facilitated diffusion. Of the glucose transport proteins (GLUT) GLUT -1 and GLUT -3 are prominent in the plasma membrane of the microvilli of syncytiotrophoblast. **Clinical**

application: Fetal macrosomia is associated with a hyperinsulinemic state with increased levels of selected growth factors and increased expression of GLUT proteins in syncytiotrophoblast. Lactate is also transported across the placenta by facilitated diffusion. It is co-transported with Hydrogen ions as lactic acid. Most free acids are transported cross the placenta by simple diffusion and are also synthesized in the placenta. The Low Density Lipoprotein Particles from maternal plasma bind to specific LDL receptors in coated pit regions of microvilli on maternal-facing side of syncytiotrophoblast. It is then taken up by a process of receptor mediated endocytosis.

Amino acids : Amino acids are concentrated in syncytiotrophoblast and then transferred to the fetal side by diffusion. Usually there is limited transfer of large proteins across the placenta. Those which are ,like IgG and retinol binding protein, are transported by receptor mediated transport. Iodine and zinc are transferred across the placenta by an energy requiring ,carrier mediated active process. The heavy metal binding protein, Metallothionein – 1 is expressed in human syncytiotrophoblast. This binds and sequesters a host of heavy metals , including zinc ,copper, lead and cadmium. Thus calcium and phosphorus are actively transported across the placenta. Vitamin A is transported across the syncytium bound to Retinol binding protein. Vitamin C

(ascorbic acid) Is transported across the placenta by an energy mediated carrier dependant mechanism.

Fetal development - Fetal development is the process in which a fetus develops during gestation, from the times of conception until birth. Fetal development consists of 3 stages.

Pre-implantation

Also known as “ all or none “ period. Toxic exposures may cause fetal death but do not cause developmental defects

Week 1 (3rd week after L.M.P)

-Fertilization of the ovum to form a zygote which undergoes mitotic cellular division, but does not increase in size. A hollow cavity forms marking the blastocyst stage. The blastocyst contains only a thin rim of trophoblast cells and a clump of cells at one end known as the “embryonic pole” which include embryonic stem cells. The blastocyst hatches from its protein shell (zona pellucida) and implants onto the endometrial lining of the mother’s uterus by the 11th -12th day. If the zygote is going to separate into identical twins, the outcome depends on when the division occurs. If division occurs within the first three days, the inner cell mass is yet to form and the outer layer of blastocyst has not yet committed to become chorion. Thus a monozygotic diamniotic, dichorionic twin pregnancy develops.⁶

If division occurs between 4th and 8th day, the inner cell mass has formed and cells destined to become chorion have already differentiated, but those of the

amnion have not. This gives rise to monozygotic , diamniotic monochorionic twin pregnancy.⁶

Week 2 (4th week after L.M.P)

- Trophoblast cells surrounding the embryonic cells proliferate and invade deeper into the uterine lining. They will eventually form the placenta and embryonic membranes. Formation of the yolk sac. The embryonic cells flatten into a disk, two-cells thick. If the zygote is going to separate into twins and the division takes place after 8 days, a monozygotic , monoamniotic monochorionic twin pregnancy results. This is because by then the chorion and amnion have already differentiated. If division is initiated even later , that is after the embryonic disc has formed , cleavage is incomplete and conjoined twins result.⁶

Embryonic Period

This is the period from the 2nd to the 8th week following fertilization . It encompasses organogenesis and is thus the most crucial period with regard to structural malformations. Toxic exposures often cause major congenital malformations

Week 3 (5th week after L.M.P

- first missed menstrual period) - A notochord forms in the center of the embryonic disk. A neural groove (future spinal cord) forms over the notochord with a brain bulge at one end. Heart tubes begin to fuse.

Week 4 (6th week after LMP)

-The embryo measures 4 mm (1/8 inch) in length and begins to curve into a C shape. Somites, the divisions of the future vertebra, form. The heart bulges, further

develops, and begins to beat in a regular rhythm on day 21. Branchial arches, grooves which will form structures of the face and neck, form. The neural tube closes. The ears begin to form as otic pits. Arm buds and a tail are visible.

Week 5 (7th week of preg-

nancy) - The embryo measures 8 mm in length. Lens pits and optic cups form the start of the developing eye. A primitive mouth and nasal pits form. The brain divides into 5 vesicles, including the early telencephalon. Leg buds form and hands form as flat paddles on the arms. Rudimentary blood moves through primitive vessels connecting to the yolk sac and chori-ionic membranes.

Week 6 (8th week of preg-

nancy) - The embryo measures 13 mm in length. Lungs begin to form. The brain continues to develop. Arms and legs have lengthened with foot and hand areas distinguishable. The hands and feet have digits, but may still be webbed.

Week 7 (9th week of preg-

nancy) - The embryo measures 22-24 mm in length. Nipples and hair follicles begin to form. Location of the elbows and toes are visible. Spontaneous limb movements may be detected by ultrasound. All essential organs have at least begun formation.

Week 8 (10th week of pregnancy)

- E m b r y o measures 40 mm in length. Intestines rotate. Facial features continue to develop. The eyelids are more developed. The external

features of the ear begin to take their final shape.

Fetal Period

During the fetal period, toxic exposures often cause physiological abnormalities or minor congenital malformation **Weeks 9 to 12 (11th to 14th week of pregnancy)** - The fetus reaches a length of 6-7 cms. The head comprises nearly half of the fetus' size. The face is well formed and develops a human appearance. Centers of ossification appear in most fetal bones. The eyelids close and will not reopen until about the 28th week. Tooth buds, which will form the baby teeth, appear. The limbs are long and thin. The fetus can make a fist with its fingers. Genitals appear well differentiated. Red blood cells are produced in the liver.

Weeks 13 to 16 (15th to 18th week of pregnancy) -The fetus reaches a length of about 15 cm (CRL – 12 cms). A fine hair called lanugo develops on the head.Fetal skin is almost transparent. More muscle tissue and bones have developed, and the bones become harder.The fetus makes active movements.Sucking motions are made with the mouth.Meconium is made in the intestinal tract.The liver and pancreas produce fluid secretions. Gender can be determined by inspection of the external genitalia by 14 weeks.

Week 18 (20th week of pregnancy) - The fetus reaches a length of 20 cm .Lanugo covers the entire body.Eyebrows and eyelashes appear.Nails appear on fingers and toes.The fetus is more active with increased muscle

development."Quickening" usually occurs (the mother can feel the fetus moving).The fetal heart-beat can be heard with a stethoscope.

Week 22 (24th week of pregnancy) - The fetus reaches a length of 28 cm .The fetus weighs about 725 g .Eyebrows and eyelashes are well formed.All of the eye components are developed.The fetus has a hand and startle reflex.Footprints and fingerprints continue forming. Alveoli are forming in lungs.

Weeks 23 to 26 (25th to 28th week of pregnancy) -The fetus reaches a length of 38 cm (CRL 25 cms).The fetus weighs about 1.2 kg .The brain develops rapidly.The nervous system develops enough to control some body functions.The eyelids open and close. The respiratory system, while immature, has developed to the point where gas exchange is possible. A baby born prematurely at this time may survive, but the possibilities for complications and death remain high.

Weeks 27 to 31 (29th to 33rd week of pregnancy) -The fetus reaches a length of about 38-43 cm (CRL 28 cms). The fetus weighs about 2 kg. The amount of body fat rapidly increases. Rhythmic breathing movements occur, but lungs are not fully mature. Thalamic brain connections, which mediate sensory input, form. Bones are fully developed, but are still soft and pliable.The fetus begins storing iron, calcium, and phosphorus.

Week 34 (36th week of pregnancy) -The fetus reaches a

length of about 40-48 cm The fetus weighs about 2.5 to 3 kg . Lanugo begins to disappear. Body fat increases. Fingernails reach the end of the fingertips. A baby born at 36 weeks has a high chance of survival, but may require medical interventions.

Weeks 35 to 38 (37th to 40th week of pregnancy) - The fetus is considered full-term at the 37th week of pregnancy. It may be 48 to 53 cms in length.The lanugo is gone except on the upper arms and shoulders. Fingernails extend beyond fingertips. Small breast buds are present on both sexes.Head hair is now coarse and thicker

Clinical Application-For an agent to cause teratogenicity the fetus must be exposed to the agent during a critical development period. Major effects occurring from drug exposure within the first 8 weeks result in an embryopathy, after 8 weeks a fetopathy results.

Fetal physiology

Amniotic fluid - In early pregnancy , amniotic fluid is an ultrafiltrate of maternal plasma. By the second trimester, it consists largely of extracellular fluid that diffuses through the fetal skin. After 20 weeks, however, the cornification of fetal skin prevents this diffusion, and amniotic fluid is composed largely of fetal urine. Pulmonary fluid accounts for a small proportion of the amniotic volume, and fluid filtering through the placenta accounts for the rest. The volume of amniotic fluid increases progressively and then falls slightly at term (200 ml at 16weeks, 1000 ml at 28 weeks,

900ml and 800 ml at 36 and 40 weeks respectively.)

Clinical applications

- Abnormal amniotic fluid volume : Diminished fluid volume is termed oligohydramnios (amniotic fluid index of 5 cms or less.)
- Increased amniotic fluid or polyhydramnios is defined by an AFI of greater than 25 cms. This condition may result from congenital anomalies (anencephaly and other open neural tube defects, esophageal atresia), maternal diabetes mellitus, Hydrops fetalis etc.
- Decreased amniotic fluid volume may be associated with fetal growth retardation and this requires close fetal surveillance. If fetal compromise is confirmed by Doppler studies, Termination of pregnancy may be indicated. It may also be associated with fetal Chromosomal anomalies (Triploidy, Trisomy 18, Turner syndrome) or congenital anomalies (Fallot tetralogy, Septal defects, Renal agenesis or dysplasia, Urethral obstruction, Hypothyroidism etc.)
- Amniocentesis for genetic diagnosis is usually performed between 14 – 20 weeks . It has more than 99 percent diagnostic accuracy for Down syndrome. Amniotic fluid AFP levels are measured if a Neural Tube Defect is suspected on ultrasound and maternal serum AFP is raised. Amniotic cells can be used

for karyotyping for diagnosis of aneuploidy and other chromosomal disorders. Early amniocentesis is performed between 11 and 14 weeks of pregnancy.

Placental Circulation-Chorionic villi are first seen around the 12th day after fertilization. Mesenchymal cords, derived from extra embryonic mesoderm, invade the primary villi forming secondary villi. After angiogenesis these are called tertiary villi. Maternal venous sinuses are tapped early in implantation but maternal arterial blood enters the intervillous space by about 14th-15th day after fertilization. By about the 17th day, fetal blood vessels are functional and the placental circulation is established.

Fetal circulation-The major differences in fetal circulation as compared to that in an adult are the presence of umbilical-placental circulation and the absence of significant pulmonary circulation.

Oxygenated blood from placenta comes through the umbilical vein, which joins the left branch of the portal vein. The greater part of this passes directly to the Inferior Vena Cava (IVC) through the ductus venosus. The oxygen rich blood reaching the right atrium through the IVC tends to course along the medial aspect of IVC and is directed through the valves of the IVC towards the foramen ovale. Here, it is divided into two portions by the lower edge of the septum secundum (crista dividens).

a) Most of it passes to the left atrium through the foramen ovale

b) The rest gets mixed with deoxygenated blood from Superior Vena Cava(SVC) and passes into the right ventricle.

The high venous return from the placenta maintains the right to left shunt through the foramen ovale and delivers most of the oxygenated blood to the brain and the heart. From the right ventricle, the blood enters the pulmonary circulation. Most of it bypasses to the systemic circulation via the ductus arteriosus and reaches the descending aorta. A high pulmonary vascular resistance maintains the right to left shunt through the ductus arteriosus. Most of the oxygenated blood from the left atrium reaches the left ventricle and then supplies the brain and the upper extremities through the aorta and the great vessels. Rest of it gets mixed with poorly oxygenated blood from the ductus arteriosus. Much of the blood in the descending aorta is carried by the umbilical arteries to the placenta, where it is again oxygenated and returned to the heart.

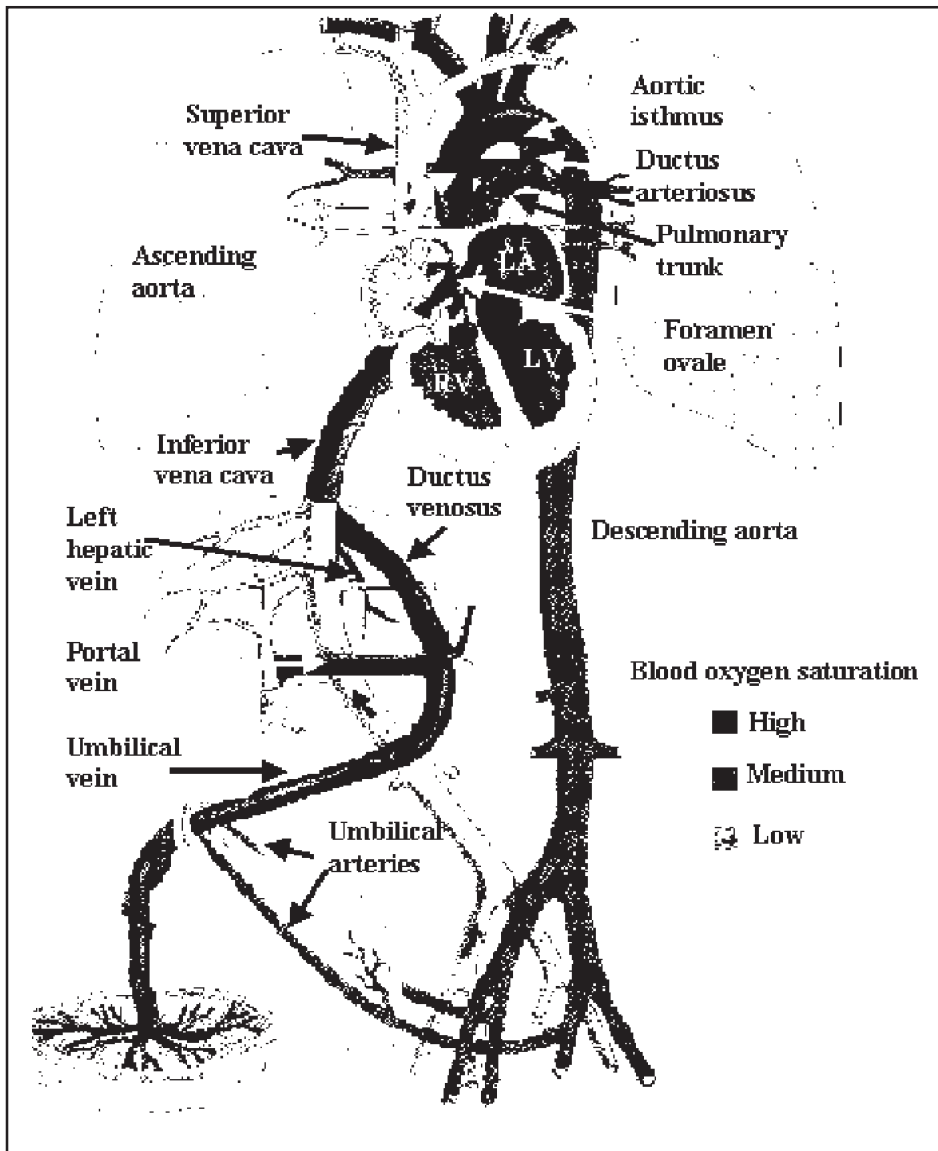
Fetal blood

Hemopoiesis-In early embryonic life hemopoiesis is demonstrable first in the yolk sac. The next major site is the liver , and finally the bone marrow. Haemoglobin content of fetal blood rises to about 12 mg/dl at midpregnancy and at term it is about 18 gms / dl.

Erythropoiesis - This is controlled in the fetus by fetal erythropoietin produced most likely by the fetal liver. The production of erythropoietin is influenced by testosterone,

estrogen , prostaglandins, thyroid hormone and lipoproteins .

Fetal hemoglobin - Most haemoglobin in the fetus is HbF , which has two g chains (a - 2 , g- 2) in place of the adult haemoglobin HbA (a - 2 , b - 2) and HbA₂ (a-2 , d -2) . b,g and d genes lie on chromosome 11. HbF is resistant to denaturation by acid and alkali. It has a higher affinity for oxygen than adult



Modified from Moore KL, Persaud TVN: Before We Are Born: Essentials of Embryology and Birth Defects, 5th ed. Philadelphia, W.B. Saunders, 1998). RA and LA=right and left atria, RV and LV=right and left ventricles.

haemoglobin. The major reason for this is that HbA binds 2,3 -diphosphoglycerate (2,3 DPG) more avidly than HbF , and this lowers the affinity of haemoglobin A for oxygen. 90% of fetal haemoglobin is

HbF between 10 and 28 weeks of gestation. From 28 to 34 weeks a switch from HbF to HbA begins. By term HbF is three fourths the total fetal hemoglobin, and by 6 months after birth only 1 % of the total haemoglobin is HbF.

Fetal Coagulation factors - with the exception of fibrinogen the fetus starts producing normal, adult type, procoagulant, fibrinolytic and anticoagulant proteins by about 12 weeks. However the levels are reduced. At birth factors II, VII, IX, X, XI, XII, XIII and fibrinogen are low in cord blood. Without prophylactic treatment, the vitamin K dependant coagulation factors usually decrease even further during the first five days after birth. This decrease is amplified in breast fed infants and may lead to hemorrhage in the newborn. Fetal fibrinogen, which appears as early as 5 weeks, has the same amino acid composition as adult fibrinogen but forms a less compressible clot, and the fibrin monomer has a lower degree of aggregation.

Despite this relative reduction in procoagulants, the fetus seems to be well protected from hemorrhage. Even after invasive procedures such as cordocentesis, excessive bleeding usually does not occur. Amniotic fluid thromboplastins and some factor in Wharton jelly combine to facilitate coagulation at the umbilical cord puncture site.

Fetal immunocompetence- In the absence of antigenic stimulus the fetal immunoglobulins consist almost entirely of maternal immunoglobulin G (IgG)

transferred across the placenta. Transfer of maternal IgG begins at about 16 weeks and the maximum takes place in last 4 weeks of pregnancy. Hence the relative deficiency in preterm infants. In certain situations like isoimmunisation resulting in hemolytic disease of the newborn, transfer of maternal IgG can be harmful to the fetus. Maternal IgM is not transported across the placenta to the fetus. Also, the IgM response is dominant in the fetus and remains so for weeks to months in the newborn. Serum IgM levels in umbilical cord blood and identification of specific antibodies may be useful in the diagnosis of intrauterine infection. IgA ingested with amniotic fluid before delivery and that ingested in colostrum after delivery provide mucosal protection. Newborn responds poorly to immunization.

Nervous system- The spinal cord extends along the entire length of the vertebral column in the embryo, but after that it grows more slowly. By 24 weeks it extends to S1, by birth to L3, and in the adult to L1. Movements of the fetus begin at 7 weeks, become co-ordinated only by 12 weeks. Respiration is evident by 14-16 weeks. The fetus can apparently hear sounds in utero by 24 - 26 weeks and by 28 weeks the eye is sensitive to light. Clinical application - A definitive negative correlation exists between fetal hypoxemia and fetal movements. This can be assessed by the biophysical profile. Thoracic movements, inappropriately called breathing

movements, disappear first, followed by the movements of fetal extremities, limb girdles, and finally fetal trunk and spine.

Gastrointestinal system - Swallowing begins at 10-12 weeks. Later in pregnancy the volume of amniotic fluid appears to be regulated substantially by fetal swallowing, for when swallowing is inhibited, hydramnios is common (anencephaly and esophageal atresia) Term fetuses swallow between 200-760 ml per day.

Meconium-The fetal bowel contents consist of various secretions, such as glycerophospholipids from the lungs, desquamated fetal cells, lanugo, scalp hair and vernix. It also contains undigested debris from swallowed amniotic fluid. The dark greenish - black appearance is caused by pigments, especially biliverdin. Meconium passage can result from normal bowel peristalsis in the mature fetus or from vagal stimulation. It can also occur when hypoxia stimulates arginine vasopressin (AVP) release from the fetal pituitary. AVP stimulates the smooth muscle of the colon to contract, resulting in intramniotic defecation.

Liver-The fetal liver has limited capacity to convert free bilirubin to bilirubin diglucuronoside. This is more so in an immature fetus. Normally unconjugated bilirubin from the fetal circulation is excreted into the amniotic fluid after 12 weeks and is then transferred across the placenta. This transport is however bidirectional. Hence

unconjugated bilirubin from a mother suffering from hemolytic anaemia will readily cross the placenta.

Gall bladder -In the third month gall bladder contains bile salts and bile acids.

Blood from umbilical cord of normal term infants contains an average 1.4 mg of bilirubin (0.2 – 2.6 mg). In hemolytic disease of the newborn it may be upto 30 – 4- mg / 100 ml.

Pancreas - Insulin containing granules can be identified in the human fetal pancreas by 9 – 10 weeks, and insulin in fetal plasma is detectable at 12 weeks. Fetal growth is determined by the amounts of nutrients from the mother with anabolism through the action of fetal insulin. Serum insulin levels are high in newborns of diabetic mothers and in other large – for – gestational age infants, but insulin levels are low in infants who are small for age.

Urinary system - The fetal kidneys start producing urine at 12 weeks . by 18 weeks they are producing 7-14 ml / day and at term this increases to 27 ml / hour or 650 ml / day. Uteroplacental insufficiency is known to decrease fetal urine formation.

Pulmonary system - In order for the lungs to function properly at birth it is important that both anatomical and morphological development and its capacity for surfactant formation are present. Anatomical development of the lung cannot be hastened by antenatal or neonatal therapy. There are three essential stages of lung development.

- Pseudoglandular stage : growth of the intrasegmental bronchial tree between the 5th and the 17th weeks. The lung looks like a microscopic gland.
- Canalicular stage : his extends from the 16 to 25 weeks. During this phase the bronchial cartilage plates extend peripherally. Each terminal bronchiole gives rise to several respiratory bronchioles, and each of these in turn divides into multiple saccular ducts.
- Terminal sac stage : the alveoli give rise to primitive pulmonary alveoli. An extensive capillary and lymphatic network develop and type II cells begin to produce surfactant.

Various insults can upset this process . With renal agenesis, no amniotic fluid is present from the beginning of lung growth, and major defects occur in all three stages. In fetuses with membrane rupture and oligohydramnios before 20 weeks, there is normal bronchial branching but immature alveoli. Membrane rupture occurring after 24 weeks has little effect on pulmonary structure.

Surfactant - It is a chemical made up of 90% lipids and 10 % proteins and is produced by type II Pneumocytes. The principal active component of surfactant is a specific lecithin – dipalmitoylphosphatidylcholine (DPPC) which accounts for 50 % and phosphatidylglycerine which accounts for 8-15% .Surfactant spreads to line the alveolus at the time of the first breath and

prevents alveolar collapse at the time of expiration. Role of corticosteroids in fetal lung maturation: Fetal cortisol is believed to be the natral stimulus for lung maturation and augmented surfactant synthesis. Thus lung maturation is delayed and respiratory distress syndrome common in fetuses with limited cortisol production. These are anencephaly , adrenal hypoplasia or congenital adrenal hyperplasia. Thus exogenously administered corticosteroids significantly reduce the incidence of respiratory distress syndrome.

Endocrine glands

Pituitary glands- The pituitary develops from two different sources. The adenohipophysis develops from the oral ectoderm – Rathke pouch and the neurohypophysis develops from the neuroectoderm. The fetal pituitary is responsive to hormones and is capable of secreting these hormones from early in fetal life. Adenocorticotrophic hormone is first detected in the fetal pituitary gland by 7 weeks and Growth formone and Leutenising hormone by 13 weeks. The posterior pituitary gland is well developed by 10- 12 weeks when oxytocin as well as arginine and vasopressin can be found in fetal blood. Oxytocin as well as AVP probably function at the level of the lung and placenta to conserve water in the fetus. AVP in fetal blood is increased during fetal distress. There is a well developed intermediate lobe in the fetal pituitary gland which disappears before term and so is absent in the adult. The cells of this lobe

produce a melanocyte stimulating hormone and Bendorphin.

Thyroid gland - The pituitary thyroid axis is functional by the end of the first trimester. The thyroid gland is able to synthesize hormones by 10-12 weeks. The placenta actively concentrates iodide on the fetal side and from 12 weeks onwards, the fetal thyroid concentrates iodide more avidly than maternal thyroid. Hence maternal administration of any form of iodides is potentially hazardous to the fetus. The thyroid hormone levels increase throughout pregnancy. The fetal pituitary is not sensitive to feedback until very late in pregnancy. Fetal thyroid hormones have a critical role in the development of the fetal brain, among other tissues. Placental tissues and membranes prevent passage of maternal thyroid hormones to a large extent by rapidly de-iodinating maternal T₃ and T₄ to reverse T₃, which is relatively inactive. Antithyroid antibodies however can cross the placenta. Congenital hypothyroidism results in many neonatal problems like neurological abnormalities, respiratory difficulties, dysmorphic facies, lethargy and hypotonia. These can be avoided with prompt thyroid replacement after birth. Immediately after birth, atmospheric cooling evokes sudden and marked increase in thyrotropin release which leads to a marked increase in T₄ levels, which are maximum 24 – 36 hours after birth.

Adrenal glands- The glands, which are much larger in the fetus

than in the adult, are made up largely of the inner or fetal zone of the adrenal cortex. The fetal adrenal glands also synthesize aldosterone.

Sexual differentiation of the fetus

Chromosomal gender: Genetic gender-XX or XY is established at the time of fertilization. For the first six weeks of development the male and female embryo are morphologically indistinguishable. **Gonadal gender:** If the fetus carries a Y chromosome the indifferent gonad develops into a testis at about six weeks after conception. Testes development is directed by a gene located on the short arm of Y chromosome. This testis-determining factor (TDF) or sex-determining region (SRY) is specific to the Y chromosome and is expressed in the single cell zygote immediately after ovum fertilization. In addition, testes development requires a dose dependent sex reversal (DDS) region on the X chromosome, as well as certain unidentified autosomal genes. **Phenotypic gender:** Male phenotypic sexual differentiation is directed by the function of the fetal testis. In the absence of a testis, female differentiation takes place. The fetal testis secretes a proteinaceous substance called *mullerian* inhibiting substance. It acts as a paracrine factor to cause regression of the mullerian duct preventing the development of uterus, fallopian tubes and upper vagina. The fetal testes also secrete testosterone, which causes virilization of the external and internal genitalia. In the external genitalia, testosterone

is converted to 5- α dihydrotestosterone, which amplifies the androgenic action of testosterone.

Genital ambiguity of the newborn

Ambiguity of the neonatal genitalia is the result of either excessive androgen action in a fetus destined to be female, or inadequate androgens in one destined to be a male. Such abnormalities can be assigned to one of four clinically defined categories. These are -

Female pseudohermaphroditism

- Mullerian inhibiting substance is not produced,
- Androgen exposure of the embryo-fetus is excessive, but variable for a fetus genetically predestined to be a female,
- The karyotype is 46 XX
- Ovaries are present.

Male pseudohermaphroditism

- There is production of mullerian inhibiting substance,
- Incomplete but variable androgenic representation for a fetus destined to be male,
- A 46,XY karyotype,
- The presence of testes or no gonad.

Dysgenetic gonads, including true hermaphroditism

- Mullerian inhibiting substance is not produced,
- Fetal androgen exposure is variable,
- Karyotype is variable, mostly abnormal,
- Neither normal ovaries nor testes are present.

True hermaphroditism

- In addition to all characteristics of dysgenetic

gonads, these subjects had both ovarian and testicular tissues.

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Anatomical Manikins

The anatomical manikin pictured here is a rarity among the cased sets of surgical instruments, amputation kits, and other medical items that make up the artifact collection of the University Archives. Often these manikins are confused with Chinese diagnostic dolls or “doctor’s ladies”. In Chinese culture, modesty forbade a woman from undergoing a physical examination or even mentioning parts of her body to a male physician. To circumvent this situation, during a house call the doctor brought along the diagnostic doll. By marking the section giving her discomfort, the woman could communicate her problems to the physician. While both anatomical and diagnostic manikins were somewhat similar in appearance, the craftsmen fashioned anatomical manikins with much more detail. Sometimes produced in male and female pairs, it was far more common to create only the female figure and always in an advanced state of pregnancy. Medical history contains little information on the origin or intended use of the manikins. Since early anatomists had few subjects

available for dissection, most anatomy studies focused on two-dimensional drawings. Historians surmise the models provided a means during the 17th and 18th centuries to study anatomy with a three-dimensional object or teach pelvic anatomy to midwives. K.F. Russell proposed in his 1970 study that while detailed, the structure of the manikins did not provide enough accurate information for serious study. Instead the models were probably used to educate the lay public on the differences between the sexes and the physiology of pregnancy. Since neither the manikins or diagnostic dolls possess dates, signatures, or any information about their creators, dating and attribution is difficult to make. Russell studied 98 manikins from several different medical libraries and his observations allowed him to separate the manikins into several different groupings. All the figures Russell studied were from Germany, Italy, or France. None were found from Great Britain, the US, or other European countries. According to his grouping, the anatomical manikin in the University Archives is most likely from Germany.

Review Article

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Breech Presentation the most common obstetric malpresentation complicates approx 4% of deliveries at term, though incidence is 7% at 32 weeks and 25% of pregnancies less than 28 weeks duration. Furthermore, because of the multitude of problems and potential problems associated with it, breech presentation has become, in recent years, the subject of significant controversy. Three types of breech presentation are recognized. Frank breech is most common type accounting for 60-65% of breech presentations and it is more common at term. Complete breech is least common type, accounting for about 5% of breech presentation. Incomplete breech is more common among premature fetuses and accounts for 25% to 35% of breech presentations. The diagnosis of breech presentation may be made by abdominal palpation and vaginal examination and confirmed by ultrasound. Prematurity, fetal malformation, mullerian anomalies, hydramnios, oligohydramnios and polar presentation are commonly observed causative factors. Congenital abnormalities of the fetus were identified in 6.3 percent of breech deliveries compared with 2.4 % of non breech deliveries. High rates of

breech presentation are noted in certain fetal genetic disorders, including trisomies 13, 18 and 21, Potters syndrome and myotonic dystrophy. Thus, conditions that alter fetal muscular tone and mobility also increase the frequency of breech presentation. In more than 50% of cases, however, no causative factor for breech presentation can be identified.

There is no major difficulty about the mechanism of breech delivery except that the largest and least compressible part of the baby (head) comes last. The irregular outline of the breech means that spontaneous rupture of the membranes may be followed by cord prolapse. The breech being comparatively soft is less likely than the head to compress the cord but still acute fetal hypoxia might occur if cord goes into spasm. Patients also have tardy labor because of ill fitting breech especially in multiparas. The most dreaded complication is entrapment of after coming head when body is pushed through partially dilated cervix which causes acute fetal hypoxia when delivery has to be affected through Dührssen's incisions. A study has shown the danger of occipital diastasis and possibility of intracranial hemorrhage when un moulded fetal head descends through pelvis.

Management

Vaginal deliveries for breech presentation have long been a topic of debate. Cesarean delivery is commonly but not exclusively used in following circumstances – a large fetus (wt > 3.8 kg), any degree of contraction or unfavorable shape of pelvis, a hyper extended head, when delivery is indicated in the absence of spontaneous labor (Some clinicians used oxytocin), Uterine dysfunction (some use oxytocin augmentation), Incomplete or footling breech presentation, An apparently healthy and viable preterm fetus with the mother in either active labor or in whom delivery is indicated, severe fetal growth restriction, previous perinatal death or children suffering from birth trauma, a request of sterilization, lack of an experienced operator, previous cesarean section.

Cesarean section

Management practice varies among different institutions and even among different institutions and even among different clinicians in the same institution. The decision to perform cesarean delivery is often based on personal experience and fear of litigation. Some clinicians have recommended a policy of cesarean section for breech presentation at term based on results of non-randomized

studies, anecdotal experiences, and medico legal concerns. However, these studies were not randomized and hence potentially biased. Otherwise also, the clinician experience, as well as, strict criteria for inclusion considered relatively safe for vaginal delivery was not compared. A recent randomized multinational trial of planned vaginal versus planned cesarean delivery for woman with a term breech presentation was undertaken. A total of 2083 women were randomly assigned to either group. The trial published in 2000, confirmed that neonatal risks associated with term breech births are much higher among planned vaginal deliveries and implied that cesarean deliveries should be systematically planned for all such women. Serious maternal complications are similar between the groups. In the later publication secondary analysis of term breech trial was done to determine factors associated with adverse perinatal outcome and it was concluded that the breech infants at term are best delivered by prelabor cesarean section. In view of the above mentioned trial which demonstrated that overall risk of perinatal death for the term frank / complete breech fetus with planned cesarean section was reduced by 75%, RCOG in its guidelines has recommended that the best method of delivering a term frank or complete singleton breech is planned cesarean section. Similarly, the ACOG Committee opinion no. 34 has concluded that the persistent term singleton breech

presentation should undergo a planned cesarean delivery. However, the decision regarding mode of delivery should depend on the attending obstetrician under hospital specific protocol guidelines and vaginal breech delivery should only be planned after documented informed consent from patient. Moreover long terms outcomes for mother and child have not been evaluated in term breech trial. Many issues were raised regarding the flaws and outcome of the trial in subsequently published correspondence in the Lancet and the British Medical Journal.

Various studies have shown convincingly that cesarean section is associated with significant morbidity, subsequent compromise of re-productive function and negative emotional effects. Also, numerous recent studies that applied a relatively wide spread policy of planned vaginal delivery in various practice conditions, did not observe the excess risk as in term breech trial. It is accordingly essential to assess a management policy in a population (like ours in India) under conditions of every day practice and planned vaginal delivery remains a clinical option that can be offered to women after providing them with clear, objective and complete information.

For preterm fetuses who are at increased risk of entrapment of after coming head through a partially dilated cervix, no randomized studies regarding vaginal delivery are present. Although most deaths in low birth weight breech group are due

to prematurity or lethal anomalies. Cesarean delivery after correcting perinatal mortality in the weight group of less than 1500 grams show improved survival compared with that in similar sized vertex presentation. Also the policy for cesarean section of preterm fetuses needs to be based on the survival rates of neonatal center of the hospital for that gestation and weight of the baby.

Even if a policy of liberal cesarean section for breech presentation is followed, the attending physicians should be prepared and trained for imminent delivery in an emergency. Otherwise also, a vaginal breech delivery should be allowed only in centers where provisions for emergency cesarean section are present, continuous fetal heart monitoring is used, delivery is attended by trained obstetrician and pediatrician.

Complicated vaginal breech deliveries are associated with increased maternal risks. Manual manipulations within the birth canal increase the risk of infection and subsequent post partum hemorrhage. Delivery of the after coming head through an incompletely dilated cervix may cause rupture of the uterus, lacerations of cervix or both. Manipulations might also lead to extensions of the episiotomy and deep perineal tears. Fetal injuries include fracture of humerus, clavicle and femur which may occur in both vaginal and cesarean deliveries. Neonatal perineal tears and testicular injuries have been reported.

Hematomas of the sternocleidomastoid muscles occasionally develop after delivery but resolve spontaneously. Brachial plexus injury leading to paralysis of arms as well as forcible extraction of fetus through a contracted pelvis might lead to spoon shaped depressions or fractures of skull.

External cephalic version (ECV)

External cephalic version (ECV) is another option in a persistent breech presentation beyond 36 weeks period of gestation. This gestation has been chosen since spontaneous reversion to breech decreases at this gestation. Also, iatrogenic prematurity in case of complications can be avoided. Surprisingly studies have shown that, women potentially suitable for ECV were not made aware of this option. Focus should be on increasing the rate of offering ECV, increasing its uptake and also its success. Reported success with external version varies from 60 to 75 percent, with a similar percentage of this remaining vertex at the time of labor. Application of a policy of external cephalic version has been associated with reductions in the caesarean delivery rate at various institutions. Version should not be attempted when there is a contra indication to vaginal delivery. A prior uterine incision is a relative contra indication. Success of ECV is less with nulliparity, anterior, lateral or cornual placenta, decreased liquor, low birth weight, descent of breech into pelvis, obesity, posteriorly located fetal spine, extended legs and firm abdominal muscles. Attempts at external version should be done

in the labor and delivery suite, and a non-stress test should be reactive prior to procedure. During ECV, the fetal position and heart rate should be monitored with real-time ultrasonography. A forward roll of the fetus is generally attempted first. If unsuccessful, backward flip is tried. Discontinuation of attempt is done in cases of discomfort, persistently abnormal fetal heart rate, or after multiple failed attempts. The non-stress test is repeated after version until a normal test result is obtained since it is commonly observed that non-reactive tracings are obtained for 20-40 minutes after the procedure. Temporary bradycardia and risk of placental abruption is also present. Routine tocolysis (IV Ritodrine, S.C. Terbutaline) appears to reduce the failure of ECV in primis but not in parous women. Various other approaches suggested for spontaneous version like knee chest position, acoustic stimulation of fetus, Moxibustion, a traditional Chinese acupressure technique are still of uncertain value. D-immunoglobulin is given if indicated after attempted version.

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Postpartum hemorrhage (PPH) or excessive blood loss after child birth is a major cause of obstetric morbidity and mortality in both developed and developing countries. It is estimated that world wide , one women dies every 4 minutes due to PPH with an average yearly incidence of about 140,000 women deaths .¹ PPH has been defined as an estimated blood loss in excess of 500 ml following a vaginal birth or 1000 ml following a caesarean birth. However, estimation of blood loss at delivery is usually inaccurate in order to clearly define PPH. Similarly, decline in haematocrit level of 10% has often been used to define PPH but determination of hemoglobin or packed cell volume may not accurately reflect the current hematologic status in women with PPH. Features like hypotension, dizziness, pallor and oliguria often regarded as markers of PPH usually do not appear until blood loss has been substantial i.e. 10% or more of total blood volume.² Etiology of PPH varies according to its time of onset and may be associated with many underlying predisposing risk factors. Although anticipation of PPH in a particular clinical setting and consequent prevention is the most rewarding management strategy but PPH

often occurs without warning in many patients.

Etiology of PPH.- Postpartum hemorrhage is conveniently divided into primary and secondary according to its time of onset in relation to parturition.^{3,4}

Primary (within first 24 hrs of delivery)- Due to -Uterine atony, Retained placenta especially placenta accrete, Defects in coagulation, and Uterine inversion

Secondary (after 24 hrs of delivery)- this due to subinvolution of placental site Retained products of conception, Infection and Inherited coagulation defects

Risk factors for PPH- Postpartum hemorrhage is often associated with several risk factors⁵, recognition of which always helps in taking appropriate and timely preventive measures for PPH. These include- Prolonged labor, Augmented labor, Rapid labor, Past history of PPH, Episiotomy (especially mediolateral), Pre-eclampsia, Over distended uterus (macrosomia, multiple pregnancy, polyhydramnios), Operative delivery, Asia and Hispanic ethnicity, Chorioamnionitis

Management-Management of PPH varies in an individual patient depending on its etiology,

availability of different treatment options and patient's desire for future fertility. Prevention of PPH through anticipation in high risk and predisposed patients is most important part of management. However, an established and intractable PPH may defy all attempts of conservative management through relatively less invasive method, often requiring hysterectomy for preservation of life. At times immediate modern intervention techniques like uterine artery embolisation may be live saving with relative preservation of fertility if general condition of the patient allows the same.

Prevention of Post partum hemorrhage- Prevention of PPH is one of the prime objectives of active management of third stage of labor which is usually implemented as a "package" including- Early cord clamping; Placenta delivery by controlled cord traction, Following signs of placental separation, Uterine massage

Cochrane review (2003) of active versus expectant management of 3rd stage of labor included meta-analysis of five randomized controlled trials and found that active management of labor decreased the incidence of PPH (both 500-1000 ml and > 1000ml), shortened the third stage of labor, decreased the need for additional therapeutic

uterotonics agents and need for blood transfusion for all women, including women deemed to be at low risk for post partum hemorrhage. The incidence of PPH, 500 ml or more was reduced in the actively managed group. The implementation of active management of labor has been controversial because of lack of consensus regarding the order of actual steps, appropriate uterotonics for low resource centers and which route is to be used. Despite lack of clarity there are recommendations to offer active management of labor to all women because on two grounds , i) The presence of risk factors can not be used to predict PPH and ii) Active management has proved to reduce the incidence of PPH, the quantity of blood loss and blood transfusions ⁶.

Oxytocins with or without ergometrine has been agent of choice as uterotonics for last several decades; both are unstable at room temperature and need to be given intramuscularly. However, misoprostol, a newer oral preparation of PGE-1 analogue is fast becoming a prime candidate for its uterotonic properties. Misoprostol can be used through oral, vaginal or rectal route, has a relative low cost and is generally stable at room temperatures ⁷. Review of misoprostol use in third stage of labor; found that misoprostol is better than placebo in reducing the need for additional uterotonics but inferior to oxytocin in preventing PPH.⁸

Treatment of PPH- Treatment of PPH involves some nonspecific measures irrespective

of the cause of PPH or till etiology becomes obvious followed immediately by the specific management of the underlying cause of PPH.

Non-specific measures

Uterine massage- Uterine atony is the single most common cause of PPH hence a bimanual pelvic examination and emptying the bladder combined with uterine massage can diminish bleeding, expel blood and blood clots and allow time for other measures to be implemented.

Visual assessment- If bleeding persists, rule out traumatic PPH. Lacerations need careful assessment and repair. Satisfactory repair may require transfer to a well equipped operating room and examination under general anaesthesia.

Placental expulsion- Manual removal or curettage by blunt instrument may identify succenturiate lobe or retained bits of the placenta as a cause of PPH. Spontaneous expulsion of placenta, apparent structural integrity on inspection and the lack of a history of previous uterine surgery make a diagnosis of retained placental bits less likely.

Additional uterotonics - Continuous intravenous infusion of oxytocin 40 units (maximum 100 milli units / minute) with or without methyl ergometrine 0.2 mg intramuscular every 2-4 hourly may be required to control PPH. In absence of a desirable response 15 methyl PGF₂α (Carboprost) 250 microgram intramuscular every 15-90 minute (8 doses maximum) or PGE-1 analogue (Misoprostol) 800-1000

mg per rectally, may be given. Various studies using 800 & 1000ugm of rectal misoprostol in the treatment of PPH have concluded that rectal misoprostol is a relatively easy, non invasive and potent treatment for PPH. It can be added to oxytocin and ergometrine as a first lime agent in the therapeutic drill in the steps taken to treat PPH⁹. A recent Cochrane review also concluded that 800mcg of rectal misoprostol could be a useful 'first line' drug for the treatment of severe PPH¹⁰.

Clotting abnormality- Abruptio placentae, HELLP syndrome, prolonged IUD, sepsis and amniotic fluid embolism are often associated with clotting abnormalities which may be caused or perpetuated by PPH. Baseline studies include complete haemogram, PT, APTT, fibrinogen and blood grouping and cross matching. The clotting time provides a simple measure of fibrinogen which can be assessed using 5 ml of blood to clot within 8-10 minutes and will remain intact normally. If fibrinogen concentration is low the blood will not clot or if it does, it will undergo partial/ complete dissolution in 30-60 minutes. In such cases a blood product replacement may be necessary to control PPH.

Uterine tamponade- Packing uterine cavity with linen gauze rolls, Foley's catheter with a large bulb, condom catheter, Sensgstacken Blakemore tube etc. helps reducing atonic PPH. Although uterine packing is generally regarded as temporary method, the majority may not

require further surgical intervention to control PPH.

Arterial embolization- Patients with stable vital signs and persistent bleeding especially if the rate of loss is not excessive are ideal candidates for arterial embolization. Radiographic visualization of bleeding vessel allows embolization with gel foam particles or coil. Embolization can be a useful alternative to hysterectomy to preserve fertility or at times may also be required to manage intractable bleeding persisting after hysterectomy.

Surgical techniques -Failure of prompt medical and other non specific measures warrants surgical intervention often depending both upon general condition of the patient and technical expertise of the obstetrician. Various surgical techniques have their own advantages and limitations. ¹¹ (Table 1)

Blood transfusion-Blood transfusion is necessary, whenever extent of blood loss is significant and ongoing, particularly if vital signs are unstable. To avoid dilutional coagulopathy concurrent replacement with coagulation factors and platelets may be necessary. Autologous transfusion (donation, storage and retransfusion) has been shown to be safe in pregnancy. It requires anticipation of the need as well as a minimal haematocrit concentration often above that of a normal pregnant woman ¹². A good knowledge of blood component therapies (Table – 2) is of utmost importance in

instituting correct replacement of deficient component of blood.

Specific measures

PPH due to ruptured uterus- Uterine rupture during labour can frequently occur spontaneously or at site of a previous caesarean section scar or any other surgical procedure involving the uterine wall, from intrauterine manipulation, trauma or from congenital malformation like rudimentary horn. Rarely, an abnormal labor, operative delivery, and placenta accreta can also lead to uterine wall rupture. Surgical repair is required, with the specific approach tailored to reconstruct the uterus, if possible. Care depends on the extent and site of rupture, the patient current clinical condition and her desire for future child bearing. Regardless of the patient wishes for the avoidance of hysterectomy, this procedure may be necessary in a life threatening situation.

PPH due to suspected placental accrete-Abnormal attachment of placenta to the inner uterine wall (placenta accreta) can cause massive hemorrhage. In fact, placenta accreta and uterine atony are the two most common reasons for post partum hysterectomy. Several factors predisposing for occurrence of placenta accreta include -Placenta praevia with or without previous uterine surgery; Prior myomectomy; Prior caesarean; Asherman's syndrome; Submucosal leiomyoma; Maternal age >35 years. In patients with placenta praevia in the current

pregnancy, the risk of placenta accreta in subsequent pregnancies has been estimated to increase progressively to 3%, 11%, 40%, 60% and 67% with each pregnancy respectively for those undergoing caesarian delivery ¹³. In the presence of predisposing factors like placenta praevia or a history of caesarian section, the obstetric care provider must keep a high index of clinical suspicion for placenta accreta and take appropriate precautions. Uterine conserving options may work in focal placenta accreta, but hysterectomy usually is the most definitive treatment of PPH due to placenta accreta. Following measures may help managing a suspected case of placenta accrete-Ultrasonography and color Doppler may be helpful although not diagnostic;The patient should be counseled about the likelihood of hysterectomy and blood transfusion; Blood product and clotting factors should be available; Cell saver technology should be considered; Delivery should be planned to allow access to adequate surgical personnel and equipment; A pre-operative anaesthesia assessment should be obtained.

PPH due to an inverted uterus- Uterine inversion, in which the uterine corpus descends to, and some times through the uterine cervix, is associated with marked hemorrhage. On bimanual examination, the finding of a firm mass below or near cervix, with absence of uterus on abdominal examination, suggests

inversion. Replacement of uterine corpus involves placing the palm of the hand against the fundus (now inverted and lower most at or though the cervix) and exerting upward pressure circumferentially with fingertips. Manual replacement with or without uterine relaxant usually is successful. In the unusual circumstances abdominal procedure may be required. The Huntington procedure involves progressive upward traction on the inverted corpus using Babcock or Allis forceps¹⁴. The Haultain procedure involves incising the cervix posteriorly, allowing for digital repositioning of the inverted uterus with subsequent repair of the incision¹⁵.

PPH due to secondary PPH- PPH occurring after first 24 hours of delivery occurs in 1% of pregnancies. Uterine atony with or without infection, retained placental tissue and bleeding diathesis¹⁶ are the common causes of secondary PPH. Ultrasonographic evaluations help in identifying intra uterine tissue or subinvolution of the placental site. Uterotonic agents, antibiotics, and dilatation and or curettage are usually sufficient to treat secondary PPH. Patient should be counseled about the likely possibility of hysterectomy before initiating any procedures.

Miscellaneous measures

Treatment of PPH in low resource settings involves several promising techniques for treatment of PPH that are relatively simple can be used by personnel with limited skills and

training in remote and /on rural areas of less developed countries¹⁷. These include-Universal use of active management of labor. Use of oxytocin in the form of prefilled sterile injection (uniject). Use of oral or rectal misoprostol for prevention and treatment of PPH. The non-inflatable antishock garment (NI-ASG) for stabilization and resuscitation of hypovolemic shock secondary to PPH The Hydrostatic condom balloon catheter to control intractable PPH secondary to uterine atony.

Oxytocin Uniject Device¹⁸- It is a sterile packet containing prefilled, non refillable, sterile, easy to use device with a fixed needle that can be activated for use after opening the sterile packet. Study conducted in Indonesia over 2200 home births comparing the oxytocin uniject device with oxytocin in a standard syringe concluded that unsafe reuse of syringes ceased, dosage accuracy increased with usage of uniject. Uniject was found to be less painful than regular syringe injections.

Non-inflatable antishock garment (NI-ASG)^{19, 20}-The non-inflatable antishock garment (NI-ASG) is a neoprene garment, much like the bottom half of a wet suit, designed in horizontal segments. Using the elasticity of the neoprene and Velcro fasteners, the garment can apply 30 to 50 mm Hg of pressure to the lower body pressure. The NI-ASG is a refinement of the pneumatic military (or medical) antishock trousers. Both devices provide circumferential counter pressure to the lower body as a

means of resuscitation from hypovolemic shock and hemostasis for bleeding in the lower body. The medical applications of the military antishock garment are described in previous reviews. A single provider can place this garment and the counter pressure of NIASG will cause a transfer of blood from the lower body and abdomen to the central circulation (Brain, heart, lungs & kidneys). It provides tamponade to bleeding sites below the level of diaphragm and diminishes further hemorrhage. Various studies document rapid resuscitation from hypovolemic shock and an extended period of stabilization for patients. Contradictions to use of NI-ASG are heart failure, stenotic heart valves, pulmonary edema, bleeding above the diaphragm.

Hydrostatic balloon condom catheter-It is a sterile rubber catheter fitted with a condom placed into the uterus through the vagina and inflated with 250-500 ml of saline. To keep catheter in place vagina must be packed with gauze. In a recent study^{21, 22} conducted in Bangladesh, atonic PPH was controlled within 15 minutes following use of condom catheter along with uterotonics. The catheter was removed after 24-48 hours, depending on the initial amount of blood loss. Even if surgery would eventually be required NIASG and condom catheter can gain useful time for transport to a hospital where definitive therapy would be available.

Conclusion-Post partum haemorrhage remains a leading cause of maternal mortality. Effectiveness of active management of third stage of labour in preventing PPH has been established in the literature. Anticipation and step wise approach in managing PPH may avoid serious morbidities and mortality.

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Table 1

Technique	Advantages & limitations
Bilateral uterine artery ligation	Quicker, easy to perform & similar sutures within utero-ovarian ligament.
Internal iliac artery ligation	Difficult technique, generally reserved for the experienced gynecologist & less successful than considered.
B lynch suture*	Exerts even pressure to compress uterine cavity and decreases bleeding.
Multiple square suturing*	Eliminates uterine cavity space by suturing both walls.

* New techniques for which experiences are limited.

Table 2

Product	Volume (ml per unit)	Contents	Effect (per unit)
Packed red cells	240	RBCs, WBCs, plasma	Increases haematocrit (3%) & Hb (1gm.dl)
Platelets	50	Platelets, RBCs, WBCs, plasma	Increase platelet count (5000-10000.cu.mm/unit)
Fresh frozen plasma	250	Fibrinogen, antithrombin III, factors V & VIII	Increase fibrinogen (10 mg/dl)
Cryoprecipitate	40	Fibrinogen, factors VIII & XIII, Von-Willebrand factor	Increase fibrinogen (10mg/dl)

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Injuries to genital tract are common both in assisted as well as spontaneous deliveries. It is estimated that over 85% of women who have spontaneous vaginal birth will sustain same form of perineal trauma and of these 60 – 70% will require repair. RCOG 2000 reports International variation in episiotomy rate namely 8% in Netherlands, 20% in England and Wales, 50% in USA and 99% in Eastern European countries. Anal sphincter injuries are reported in 11% of vaginal deliveries with midline episiotomy 2.5% with mediolateral episiotomy but 33% of women sustain occult anal sphincter injury (Sultan 1997). If the perineal laceration are not appropriately repaired due to incorrect classification long term complication can occur. Sultan (1999) has proposed the following classification which has been incorporated in guidelines of Royal College of Obstetricians & Gynaecologists and International classification (Norton et al 2002).

- *First degree* - Laceration involving vaginal epithelium or perineal skin only.
- *Second degree* - Involvement of perineal muscles but not anal sphincter.
- *Third degree* - Disruption of anal sphincter muscles and this is further classified into - *Grade 3a* - <50% thickness of external sphincter torn; *Grade*

3b - >50% of thickness of external sphincter torn; *Grade 3c* - Internal sphincter also torn

- *Fourth degree* - A third degree tear with disruption of anal epithelium.

An isolated rectal tear without involvement of the anal sphincter is rare and should not be included in the above classification. Any injury to the anterior part of the labia, anterior vagina, urethra or clitoris is classified as an anterior perineal tear.

This classification enables to identify full extent of injury to perform an appropriate repair. It has prognostic significance from evidences that repair of isolated internal anal sphincter defects in patients with fecal incontinence is associated with favourable outcome.

Risk factors for 3rd degree perineal repair include

- Fetal macrosomia OR (Odd ratio) 1.9
- Failure of labour to progress in 2nd stage OR 10.8
- Induction of labour (OR 1.9)
- Non reassuring fetal heart pattern OR 11.7
- Mediolateral epis 2.8%
- Vacuum extraction 8.2%
- Forceps deliveries 26.7%

Episiotomy

Episiotomy is a surgical incision made in perineum and cuts the vaginal mucosa, perineal muscles and skin to enlarge the vaginal orifice during the last part of the

second stage of labour. It is the commonest operation requiring repair in the obstetrics. It is of three types

Median - Starts in the midline of the fourchette and extend in the middle of the perineum. Drawbacks is extension to anal sphincter is common. Advantage is associated with less bleeding, pain and wound breakdown.

Mediolateral - Starts at the midpoint of the lower vulvar rim and should be inclined to left or right the side of midline by about 15° - 20°. Main advantage is that it avoids the risk of potential extension of the incision to the external and sphincter and may reduce the likelihood of a severe perineal tear (Riskin – Mashiah et al 2000). It is associated with more bleeding and pain.

J-shaped - Incision is initially directed downward towards the anal sphincter but terminal part of the incision is directed laterally. Aim is to combine the advantage of both medial and medio lateral episiotomy.

Episiotomy was popular in 70s and 80s and rational behind the routine episiotomy was that it cause less pain, heals better, have less chances of extending to 3rd or 4th degree tear and less dyspareuria compared to ragged perineal tear. However, evidence based studies showed it episiotomy cause more haemorrhage, equal pain and