Journal of Postgraduate Medical Education, Training & Research

National Board of Examinations

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Considering the variations in the level of standards of post graduate and post doctoral examinations in our country and based on the recommendations of an Expert Group set up for maintaining uniform standards, the Ministry of Health & Family Welfare, Government of India, established the National Board of Examinations (NBE) in 1975, with its headquarters at New Delhi.

Objectives of NBE

Conduct postgraduate examinations in the disciplines of modern medicine at the national level.

Maintain a high standard of examination, so as to ensure that candidates have received adequate training and are competent in every way to practice as specialitists, in their respective fields.

Constitute Speciality Boards in which the examinations are to be conducted.

Formulate basic training requirements for eligibility to appear for the respective examinations.

Prescribe course curricula for postgraduate studies.

Organize postgraduate courses, workshops, seminars, symposia and training programmes of specialised nature.

Institute professorships, other faculty positions, fellowships, research cadre positions and scholarships etc. for realising the objectives of the Board.

Constitute an Accreditation Committee to approve centers for DNB courses.

Co-ordinate with national and international bodies, agencies, universities for the furtherance of the objectives of the Board.

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Want to be a doctor? Think before you leap!

B. M. Hegde

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Wait! While medicine is a wonderful profession if practiced ethically, it could be a curse if it is transformed into a business, like any other money making craft. Young as you are, at your age of 17-18, you are liable to be drawn by the medical claptrap that you witness daily in the media. The reality is far from what is seen in those advertisements. If your idea is to make a quick buck and "enjoy" life, please look for other easier avenues where your actions might not result in human misery. Medicine is not for those that are looking for a business. It is a long and arduous course in addition. Many of you, who have got very high ranks in the qualifying examinations, thanks to the multitude of coaching classes that teach the tricks of the trade in the present examination systems, might find it hard to master that enigma, the human body, which follows the non-linear mathematical rules and the science of chaos. You would not have studied those disciplines so far. Serious doctoring needs serious thinking and not parrot repeating the books as you might have done till now.

The one profession that gives overall job satisfaction still remains the profession of doctoring. That said, I must hasten to add that the pleasure comes only when one does not concentrate on the monetary returns. Money will come but, more than all that, it is the joy of seeing the gratitude in the eyes of a grateful patient who naturally thinks that you were his savior although, in effect, it is the human immune system that cures. As the common saying goes doctor just dresses the wound, it is God who heals the same. That brings us to the central focus of medical practice. A great physician of the 18th Century, Sir James Spence, once wrote that whole world of medicine revolves round two human beings. A human being who is ill or imagines being ill comes to seek the advice of another human being in whom the first has confidence (faith). It is this summit, the medical consultation, which is the most vital part. All else in medicine, like the medical school, the hospital, the laboratory and, the library should flow from this summit. Patient's faith in the doctor is the basis of the so called placebo effect, which stimulates the patient's immune system to cure the disease. It is otherwise called the Expectation Effect. (EE)

Therefore, you must remember that medicine could only be learnt on the bed side. Each patient is like a mini book. No two patients are alike even with the same disease as every disease presents through the personality of the patient. The habit of learning the books by heart, as you have been doing so far, does not work here. Books are necessary to keep in touch with the basis of medical practice. William Osler, the great medical teacher of the early 20 the century, had this to say about the books. "Learning medicine from the books alone is like swimming an uncharted sea, while learning medicine without books is not going to the sea at all." How very true? Every medical student should learn to listen to patients. Listening is more difficult than talking. Lord Platt, one of the greatest of teachers at the University College of Medicine in London, was of the firm opinion that "if you listen to your patient long enough s/he will tell you what is wrong with her/him." This statement, made in 1949, has been recently ratified by a very sophisticated study in London. I can vouch for the same with my nearly four and half decades of teaching medical students and managing patients experience.

Before you jump into the bandwagon ask a few relevant questions of your self. Can you smile at some one naturally? Can you touch some one naturally? Do you have an insight into another human being's problems?

If the answers to these three questions are in the affirmative, you are the ideal candidate for the medical course. If not, and be sure about that, you better avoid that profession. It is not a bad idea to have first hand experience of dealing with sick human beings before you finally take the plunge. Even while one is in the 10th class, the summer vacation could well be spent doing some volunteer work either in a hospital, nursing home or an old age home where you will see first hand the pain, suffering, the filth that might surround the sick, the smell of infected wounds, the moaning and groaning of the sick, tragedy of unexpected death and the resultant reaction of the near and the dear ones on their bereavement, as also the poverty that drives people to be sick in the first place. This could be experienced even now. If you are comfortable go ahead. If not do not suffer all your life as doing anything that does not make you internally happy is a great distress that might drive one crazy as also make one a patient even. Make your work your play and you enjoy life.

Many systems of medicine being practised in the world- Whereas modern medicine takes the cake in the field of medical care delivery, it need not be the only or the best. There are equally good systems of medical care that need to be scientifically authenticated. Efforts are on all over the world to do just that. Recent audits have shown that modern medicine, which uses the wrong linear mathematics as its base is slightly off balance like the Tower of Pisa. Prohibitive and ever increasing cost of medical care in the present corporate hospital set ups, the unacceptable dangerous side effects of the multitude of drugs used today and, the clear evidence of over diagnosis and over interventions have resulted in patients looking for avenues elsewhere. Unfortunately, as of now there are no guidelines for patients to use the complementary systems scientifically. Every system of medical care, of which there are plenty all over the world, led by the ancient Indian Ayurvedic system, seems to forget that they need to update their methods using the modern scientific methods of testing. Recent estimates of the very high budgets of complementary medicines in Europe and the USA have induced many players to come forward to authenticate those systems. Sooner than later it will have to fructify. The future of modern medicine might be confined to emergency care and corrective surgery at the most. Medical education in India is crying for reforms but the tardy pace of the governmental jaggarnaught moving and the money power of the vested interests are hampering those effort. It has to happen soon for the good of mankind and the profession if the latter has to survive in the present day world. Paternalism in medicine has to give way to partnership in medical care where the patient takes a keen interest in his/her treatment. Literate patients today could get access to most of medical information on the internet and the former have become vocal about their role. This is good augury. It becomes incumbent on the good doctor to transform those informations to knowledge and finally to medical wisdom to succeed. Novice will find it very difficult in this jungle of information to get at the rose wood. S/he needs a good guide to do that. That is the pivotal role of a teacher in the medical school. The down side of that is the defensive medicine that the star performers use today to save their skin. Defensive medicine turns out to be exceptionally expensive as all the tests are done for all patients irrespective of the need. Medical education must move from the four walls of the class room of a tertiary care hospital to the community where the student gets to see diseases naked. In the teaching hospitals the student gets to see the filtered lot of chronic, serious and incurable diseases. The latter are a microscopic minority of diseases in society. Lest the student should get a distorted version of the disease prevalence in society the teaching has to be in the community. Only one medical school in India does that and that is the MGM College in Wardha. They are also advanced in that they have seen that all out patients are first managed by the Community Medicine department in the hospital and the specialists come into the picture only when their services are needed, avoiding thereby unnecessary suffering for patients

with minor illness syndromes. The latter form the bulk of the sick population anywhere in the world. This kind of new approach is seen in many foreign medical schools. Surfeit of specialists is a bane of medical care arena as is clearly shown by a 14 industrialised countries survey of health status and medical care published in the leading American journal, JAMA (2000; 284: 483) This study, Is US medicine the best in the world will be an eye opener for all entrants to the medical college.

The biggest disease-Poverty is not only the basic cause of all diseases in the world ranging from common cold to cancer, poverty is the largest disease load in the world. Not knowing where one's next meal comes from is the greatest risk factor for killer diseases like heart attacks and caner! While there are about 39-40 million AIDS patients in this world, about whom you hear and read every day, there are 840 million who are malnourished and another 540 million live on a meagre income of less than a dollar a day. These are our real heroes looking for good doctors to do something about them. 6000 children die daily in India due to the complications resulting from malnutrition. India has the largest number of malnourished children in the world, a total of 67 million in all. This is much bigger than the total load of such children in the whole of Sub Saharan Africa! With all our governmental efforts in the last one decade India's load

of malnourished children fell from 47% in 1997 to 46.4% in 2007! What India needs for its health are the following: clean drinking water for all, three meals a day uncontaminated by human and /or animal excreta, avoidance of hookworm menace with toilets for all, avoidance of cooking smoke coming into the house in villages containing deadly carbon monoxide killing children and mothers in large numbers, economic empower-ment and education of women, nourishing mid day meal for all pregnant women to beget healthy children, good family welfare schemes, primary education for all, prohibiting alcohol and tobacco-the two deadliest enemies of good health and, employment opportunities for the able bodied. Unemployment is a big risk factor for killer diseases! While you would read about AIDS, heart diseases and diabetes daily in your news papers and you get to see illustrated medical star performers eloquently championing those disease managements, no one ever talks about the greatest disease-poverty. When you become a good doctor and a star performer please help the poor. God will bless you. Serve them and you will be rewarded. India needs a new medical care system which combines the best in emergency care and corrective surgery from modern medicine and the rest of the 90% patient population to be able to get well using the selected authenticated methods in the complementary

systems of medicine to do most good to most people most of the time. Ayurveda and homeopathy are also good systems if one wants to study them scientifically and then get into research to take knowledge forwards in those areas. You would be the pioneers. Your parents might not encourage you there as they would think that your social status might not be the same as modern medical doctors. Truth is otherwise, though. West is looking to Ayurveda and other systems to lessen their unnecessary financial burden of the top heavy modern medicine. Before you take your seat in the counseling table try and see the movie SICKO if you can. Good luck to you all. Try and get what you want but after that do all that you can to take knowledge forwards in that area for the good of mankind. What changes is progress and that which does not change becomes dogma. May the medial profession get its due place in society as in the past where doctors were looked up to as God incarnates!

"Life is a school. Those who learn to love and help others graduate with honors."
                                        Anon.

Setting up a Rural Hospital

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In today's world, high tech and 'state of the art' are metaphors usually associated with the setting up of hospitals in large cities. This, not withstanding the fact that "state of the art" technology invariably gets attached with a heavy price tag for the user and debars a large population of our country from availing any benefit out of such a facility. Even the doctors working in such hospitals are under constant pressure from the management to give a higher turnover of patients. And as a result, they are often forced to resort to unethical practices for their survival in such an atmosphere. On the other hand, peripheral government hospitals are often ill equipped and ill staffed mainly due to financial constraints, and the doctor here is often forced to take the patient outside for better quality of service again at the cost of his reputation or end up providing substandard care.Against this background, a number of doctors are today practising either through voluntary organizations or on their own setting up small hospitals with limited resources and backed up by their innovative skills. These are situated in periurban slums, small towns and semiurban areas and in rural settings providing appropriate care (both primary and secondary) to impoverished communities. This is across different specialities and needbased in nature. There is a high level of patient satisfaction in them. The doctors earn well and are highly respected in the community they serve. And it will not be any exaggeration to say that creation of such hospitals by committed and innovative health professionals across the country would be the most rational way of developing a viable and economically sustainable backbone of the healthcare services of the nation. As such, in our country, nearly 400million people have no access to what the WHO levels as "essential health care" today. And the percapita GNP of the countries from which we import surgical technology varies from 30,000 US dollars to 50,000 US dollars, while ours is around 750 US dollars only.

Summing up therefore, a "rural hospital" is a hospital which is need based, working with limited resources, and serving with appropriate care ,a population living in conditions prevailing in rural India. These institutions are sustained jointly by voluntary contribution of services of the healthcare providers and finances by the community. It may be in an urban slum, periphery of a small town or in a totally rural setting.

According to a survey, 50% of doctors working in such "rural" hospitals are in private practice and the rest are in government service[1]. This article is based on the experience of such doctors working across the country with success. After postgraduation in rural surgery, one has a choice, either to go into a voluntary organization, or be in government service, or to start his own individual practice. In any of these situations, he becomes an agent of change in healthcare development in his area of professional practice and in this article we propose to provide guidelines to help him in doing so. Let us take up the Non-government and government sectors one by one.

Non governmental sector (NGO or private)- It is always good to take some practical training in the various disciplines of the course before setting up one's own hospital. Initial setting depends on one's financial resources. A feasibility study must be made. Then, an initial master plan is necessary which can be implemented in parts, over a period of time. And having made the basic infrastructure, it has to start servicing the community.

Feasibility- First the needs of the local community must be studied. It is useless for instance to make a hospital next to a big

general hospital. It is also not possible (or very difficult) to start one in a remote area, where there is no proper communication channels like proper roads or a railway station nearby, Electricity is very important and so also is proper water supply. If local municipal water supply is insufficient, one may have to dig a tubewell. Pond water or water from an uncovered well is not desirable for hospital use at all.Shortfall of state electric supply is faced by rural surgeons in many suitable ways in their areas. Some are using solar energy. Others are using biogas. But the most common is the standby diesel generator. In our country, not more than 10% of rural population have any form toilets. And they always have many attendants with them, often travelling long distances. One may have to build a septic tank and make arrangements for garbage disposal for them. Various rural surgeons have made appropriate models suiting their areas of practice, of meeting their power and water needs and of sewerage disposal. Most people have made septic tanks and some form of burning of biological waste. A local engineer friend would be a very good support in planning in such matters. Even a pit latrine is good for attendants.

Summing up therefore, before starting to build a rural hospital, one has to consider the following things:

- Need of the community for a hospital

- Communication facilities
- Proper water supply
- Government electric supply, and
- Facility for waste disposal

Setting up the hospital - A building is like an almirah providing protection to its contents, in this case the patients, attendants and paramedics who take care of them. A good building is one which makes its users comfortable while working. One must remember that a hospital is a live structure bustling with activity 24 hours a day. The western educated public including health professionals are deeply committed psychologically, to the "white corridor" attitude of hospital construction. A pragmatic approach suiting the builder`s financial constraints, meeting the needs of our simple rural folk and while maintaining basic hygienic standards requires bold and radical thinking. For any given population, the WHO recommendation is 4 beds per thousand. While, in our country it is only 0`6 beds per thousand[2]. Norms laid down by western architects and government health directorates often becomes an impediment to private and voluntary hospital builders in solving this problem. Albeit one has to keep the law on the right side. In making a plan therefore, it is good to study the state legislations in this regard.Also, the rural surgeon must remember, that he is the main architect of the hospital. Whether it is by renovating an old building

or making a new one, and whatever advice the architect and civil engineer gives, he has to make the final decision. During the course of training, it is therefore good to visit some already established rural hospitals and meet the successful rural surgeons in the countryside. In the beginning, money should not be wasted in airconditioning etc.. They can come as "add ons" later.Cooking space and toilets have to be made. They should be of Indian style, which are easier to maintain. In a typical rural setting, a shed for patient`s relatives is sufficient for people coming from long distances. A canteen is preferable within or near the campus.

The initial master plan should include the following:

- Staff accommodation
- Sterilization area
- Pharmacy and medical store
- Library cum office room
- Administrative area
- Operation theatre including changing area
- Xray, darkroom,laboratory and wards
- If space is available, separate labour room. Albeit, for an initial small setup, operation theatre can double up for this purpose. Then proper precautions is a must before performing even minor clean surgery after a delivery.
- An outpatient clinic, with at least three consultation chambers, reception and waiting hall. Drinking water

has to be provided in hot climate.

- A shed or hall for patient's relatives.

The actual design will have to vary according to the available land etc. and the financial resources. Only a small part of the master plan may be made initially and as the clientele increases, and money flows in, additions may be made slowly over a period of time. Hence it is not proposed to provide a set plan of a building in this article.

Operation theatre- the operation theatre design needs to be discussed. This structure will have to be a pucca building. The floor and the walls upto a height of seven feet will have to be washable. This part will have to be of polished cement with stone chips, or stone or marble. Alternatively, the walls could be oilpainted. Ceramic tiles is also a good material. Painting should be done every year of the roof and the upper part of the walls. Windows should be sealed with white painted glass. This helps in using daylight inside the theatre. A good functioning exhaust must be fitted in the upper edge of a wall.Airconditioning is optional and should be used only if the power supply is sufficient. As such during a major surgery, at least one suction machine will be working, may be a diathermy, a steam sterilizer (boiler) and the operation theatre lights and an airconditioner will be an added burden on electric supply. Also, airconditioners are notorious for collecting dust particles and

associated bacteria and needs to be cleaned regularly. Many rural hospitals use wall fans in the theatre . The size of the operation should be such that there is enough space for circulation during major surgery of at least five people (surgeon, two assistants, anaesthetist and one OT assistant) and the instrument trolley, suction, diathermy, anaesthesia machine and at least one drug almirah. It should not be too large since then it would collect dust and cleaning and sterilization becomes a problem It has been our experience that the optimal space for the turnover of a small hospital would be around 300 sq.ft. A diagrammatic sketch is given below. For a bigger setup, two theatres can be made with a common scrubbing area, semisterile area for autoclave, changing etc.

After construction, the theatre needs to be furnished. Even if one has enough funds, one should beware of buying "state of the art" equipment under pressure of the sellers. Prioritisation needs to be done. A simple mechanical operation theatre table with a kidney bridge should be enough to start with (costing about six thousand rupees). A 200 watt lamp can be converted into an OT operating light by an innovative electrician and focusing standing table lamps could add up to this. One must have a foot suction as standby on the other hand and good standby torches to face sudden electricity failure. However no compromise

can be made in buying small instruments like artery forceps, mosquitoe forceps and needle holders etc.. Often trolleys and drip stands, hammers, screw drivers and such other equipment, can be made by the local blacksmith. Even hospital beds etc. Involving the local community in making the set up not only saves money, but also creates a goodwill amongst them. Then, as the clientele increases, more and more sophisticated gadgets can be added according to the need of the hospital. There are many rural surgeons today who, having started with small and simple facilities in this way, have finally ended up purchasing laparoscopes, endoscopes and other sophisticated equipments thus developing their standard of service.

Starting the service

Team building- Once the building and initial equipment is in place, the hospital is ready for service. No rural surgeon can function without a team of workers. This he has to build up. Many surgeons who are now practising in rural areas are married to doctors. This is a great support in developing a good practice in a remote setting. Hoping to get "qualified" nurses in a rural setting or even in a periurban slum is a rarity. The larger government institutions, the city corporate hospitals, and lucrative jobs in the middle east and Europe lure out almost all of them. One is lucky if one gets even one or two ANMs for one's rural hospital. The rural

surgeon therefore has to train up his own nursing staff. It is not a difficult job at all. And semi educated boys and girls from the local community do a great job in this position. This has been the experience of most practising rural surgeons. The number of staff will needs to be increased as the workload and the number of beds increase. And their capacity has to be build up by bedside teaching as also meetings and lectures in the local dialect (and also in English, specially for those who will have to handle drugs etc.) with them. Their welfare (leave, remuneration etc.)will also have to be looked after. In due course of time they become like members of a large family living together. Many rural surgeons are today using ayurvedic, unani and homeopathic doctors in their teams providing excellent service to the community. Thus not only the community is being served by the rural surgeon, but also he provides gainful employment to the local unemployed youth thus generating employment in their areas of practice. A detailed discussion on this "paramedic training " is beyond the scope of this article.

Sterilisation and maintenance of equipment - Suffice here to say that the surgeon has to learn the use of an autoclave and other sterilization equipment and teach it to his chosen paramedics. Initially a single drum autoclave might suffice but it is always preferable to buy a double drum one if finances permit. Sterilising

the theatre, packing of gauze, linen and instruments etc., has to be learnt by apprenticeship from established rural hospitals and then taught to the paramedics. Supervision has to continue. It is good to have a small workshop for maintenance.

Records, accounts and bookkeeping- These are very important functions. Both medical records, hospital equipment etc., and accounts. These the postgraduate student must learn during his posting in the rural hospital . If a clean slate is maintained from the beginning, one has the peace of mind to concentrate in his academic work. One has to understand that a rural surgeon is a team builder, manager and a research scientist all combined in one. Future development of appropriate technology in healthcare of our nation depends on the outcome of his practice.

Finances-no hospital serving the poorer sections can ever run with profit. It is always a loosing concern. Inspite of this fact, all rural surgeons as of today, are living a good life, providing the best of education to their children, having their holidays and saving for their old age. This is all because of innovative financial management. If the hospital is made to function under a trust, there is no dearth of funding from industrial houses, and funding agencies. If it is made into a corporate structure, a large clientele provides for them. It is good however to have a chartered

accountant friend to work for the surgeon from the beginning, albeit on payment.

This is in brief, guidelines to set up a rural hospital in the private sector.

Government Sector

After postgraduation, one could join a district or taluka hospital, a voluntary organization (eg.missionary) hospital, or a subdivisional hospital or even a CHC. One would here expect to have the following things:

- A building with a few beds with linen etc.

- An OPD, laboratory, oper-ation theatre with some furniture and equipment, colleagues of other specia-lities, and a lucky one, a colleague in the same speciality

- An administrative chief (the surgeon himself may be the one)

- The surgical, managerial and innovative skill of the surgeon himself

- Then there is no shortage of electricity and water (hope-fully).

The first thing is to fit in with the existing group of people and acquire their confidence. A new person in a senior position is always watched by others. And the surgeon's technical, manag-erial and leadership skills has to be the tools for him to succeed in developing a homogenous team for good service to the community. One has to check up the sterilization procedures in

practice in the institution as also the lab function, cleanliness and patient care. Today there is corruption in all areas. If the rural surgeon starts a clean practice himself, after initial resistance, others will slowly give in to his methods. And that is probably the only way he could improve in the functions of the hospital.

Conclusion - Our country is vast and the socioeconomic conditions vary enormously in different parts as also the language. The hospital has to suite the needs of the local communities. This is only a broad guideline for setting up a rural hospital in India. The rural surgeon's innovative faculties have to be sharp to succeed in providing appropriare care locally. Albeit this is not a difficult task at all as proved by previous entrepreneurs in the field of health care in the past.

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Arabian medical science

Arabian medical science forms an important chapter in the history of the development of medicine, not because it was especially productive but because it preserved Greek medical science with that of its most important representative Galen. It was, however, strongly influenced by oriental elements of later times. The adherents of the heretic Nestorius, who in 431 settled in Edessa, were the teachers of the Arabs. After the expulsion these Nestorians settled in Dschondisapor in 489, and there founded a medical school. After the conquest of Persia by the Arabs in 650, Greek culture was held in great esteem, and learned Nestorian, Jewish, and even Indian physicians worked diligently as translators of the Greek writings. In Arabian Spain conditions similarly developed from the seventh century. Among important physicians in the first period of Greek-Arabic medicine — the period of dependence and of translations — come first the Nestorian family Bachtischua of Syria, which flourished until the eleventh century; Abu Zakerijja Jahja ben Maseweih (d. 875), known as Joannes Damascenus, Mesue the Elder, a Christian who was a director of the hospital at Bagdad, did independent work, and supervised the translation of Greek authors, Abu Jusuf Jacub ben Ishak ben el-Subbah el-Kindi (Alkindus, 813-73), who wrote a work about compound drugs, and the Nestorian Abu Zeid Honein ben Ishak ben Soliman ben Ejjub el 'Ibadi (Joannitius, 809-about 873), a teacher in Baghdad who translated Hippocrates and Dioscurides, and whose work "Isagoge in artem parvam Galeni", early translated into Latin, was much read in the Middle Ages. Wide activity and independent observation — based, however, wholly upon the doctrine of Galen — were shown by Abu Bekr Muhammed ben Zakarijia er-Razi (Rhazes, about 850-923), whose chief work, however, "El-Hawi fi'l Tib" (Continens) is a rather unsystematic compilation. In the Middle Ages his "Ketaab altib Almansuri" (Liber medicinalis Almansoris) was well known and had many commentators. The most valuable of the thirty-six productions of Rhazes which have come down to us is "De variolis et morbillis", a book based upon personal experience. We ought also to mention the dietetic writer Abu Jakub Ishak ben Soleiman el-Israili (Isaac Judaeus, 830-about 932), an Egyptian Jew; the Persian, Ali ben el Abbas Ala ed-Din el-Madschhusi (Ali Abbas, d. 994) author of "El-Maliki" (Regalis dispositio, Pantegnum). Abu Dshafer Ahmed ben Ibrahim ben Abu Chalid Ihn el-Dshezzar (d. 1009) wrote about the causes of the plague in Egypt. A work on pharmaceutics was written by the physician in ordinary to the Spanish Caliph Hisham II (976-1013), Abu Daut Soleiman ben Hassan Ibn Dsholdschholl.

Seizures and Epilepsy

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Epilepsy is a disorder characterized by the occurrence of at least 2 unprovoked seizures atleast 24 hours apart. Seizures are the manifestation of abnormal hyper-synchronous discharges of cortical neurons. The clinical signs or symptoms depend on the location of the discharging cortical neurons.

Classification of epileptic seizures - In 1981, the International League Against Epilepsy (ILAE) developed a classification of epileptic seizures that divides seizures into 2 major classes- Partial-onset seizures begin in focal area of the cerebral cortex; Generalized - onset seizures have an onset simultaneously in both cerebral hemispheres; Some seizures are difficult to fit and they are considered unclassified seizures.

- **Partial-onset seizures -** Partial-onset seizures are further classified as simple partial seizures, complex partial seizures, or secondarily generalized tonic-clonic seizures;The defining element of SPC is a seizure with preserved consciousness; The many kinds of SPC include sensory, motor, autonomic, and psychic types; Consciousness is impaired during a complex partial seizure; Typically begins with behavioral arrest and is followed by staring, automatisms, and postictal confusion; Automatisms frequently consist of chewing, lip smacking, mumbling, and fumbling with the hands; Lasts about 60-90 seconds and is followed by brief post ictal confusion.

- **Generalized - onset seizures -** Generalized - onset seizures are classified into six major categories- absence seizures, tonic seizures, clonic seizures, myoclonic seizures, primary generalized tonic-clonic seizures, and atonic seizures.

- **Absence seizures-**Absence seizures are brief episodes of impaired consciousness with no aura or postictal confusion; Last less than 20 seconds and are accompanied by few or no automatisms; Facial automatisms are most common; Decreased perfor-mance in school or overall attention is a subtle manifestation of frequent absence seizures

- **Myoclonic seizures-** Myoclonic seizures consist of brief, arrhythmic, jerking, motor movements that last less than a second; Myoclonic seizures often cluster within a few minutes; If they evolve into rhythmic, jerking movements, they are classified as evolving into a clonic seizure.

- **Tonic-clonic seizures-** Clonic seizures consist of rhythmic, motor, jerking movements with impairment of consciousness. involving the upper and lower extremities; Tonic seizures consist of sudden-onset tonic extension or flexion of the head, trunk, and/or extremities for several seconds. These seizures typically occur in relation to drowsiness. They are often associated with other neurologic abnormalities; Tonic-clonic seizures are commonly referred to as grand mal seizures. They consist of several motor behaviors, including generalized tonic extension of the extremities lasting for few seconds followed by clonic rhythmic movements and prolonged postictal confusion; Atonic seizures occur in people with clinically significant neurol-ogic abnormalities. These seizures consist of brief loss of postural tone, often resulting in falls and injuries

Classification of epileptic syndromes -In 1989, the ILAE

developed a classification of epileptic syndromes. At present, a task force is revising this syndromic classification. The current system comprises two major categories-localization-related syndromes and generalized-onset syndromes.

- **Localization-related epilepsies and syndromes** -Idiopathic with age-related onset; Benign childhood epilepsy with centrotemporal spikes; Childhood epilepsy with occipital paroxysms; Symptomatic.

- **Generalized epilepsies and syndromes-**Idiopathic with age-related onset; Benign neonatal familial convulsions; Benign neonatal convulsions; Benign myoclonic epilepsy of infancy; Childhood absence epilepsy (pyknolepsy); Juvenile absence epilepsy; Juvenile myoclonic epilepsy (JME); Epilepsy with grand mal seizures; Idiopathic and/or symptomatic infantile spasms; Lennox-Gastaut syndrome; Epilepsy with myoclonic astatic seizures; Epilepsy with myoclonic absences; Symptomatic

Management of a Child with Seizure

- **Ascertain is it a seizure-** There are conditions mimicking seizures like Benign positional vertigo, breath holding spells, cough syncope, involuntary movements, narcolepsy, night terrors, pseudoseizures, rage attacks, benign myoclonus of infancy and tics.If Initial seizure - yes, then do tests- RBS,Ca,metabolic studies, EEG , CSF, CT, MRI; Abnormal results-treat underlying cause, consider AED; Normal results– no family h/o seizures, close observation, no treatment. If EEG abnormal – Epilepsy? Then classify seizure type & follow up; Good control- AED levels, monitor toxicity, sos EEG; Poor control- prolonged EEG record &video monitor, readjust AED-compliance, dose, reinvestigate - CT MRI, reconsider underlying pathology.

- **Prescribe an Anti Epileptic Drug (AED)-**The goal of treatment is to achieve a seizure-free status without adverse effects. Monotherapy is important because it decreases the likelihood of adverse effects and avoids drug interactions. The type of seizure and the specific epileptic syndrome play a role in the selection of anticonvulsants .

Mechanism of action of drugs- Blockers of repetitive activation of sodium channel - Phenytoin, carbamazepine, oxcarbazepine; GABA enhancers - Phenobarbital, benzodiazepines; Glutamate modulators - Topiramate, lamotrigine, felbamate; T-calcium channel blockers - Ethosuximide, valproate; N- and L-calcium channel blockers - Lamotrigine, topiramate, zonisamide, valp-roate; H-current modulators - Gabapentin, lamotrigine; Blockers of unique binding sites - Gabapentin, levetiracetam; Carbonic anhydrase inhibitors - Topiramate, zonisamide

Recent Advances in Treatment of Seizures

- **Absence seizures -** If only absence seizures are present, most neurologists treat them with ethosuximide. If absence seizures are present with other types (eg. generalized tonic-clonic seizures, myoclonic seizures), the choices are valproic acid, lamotrigine, or topiramate.

- **Tonic or atonic seizures-** Tonic or atonic seizures typically indicate clinically significant brain injury. The Lennox-Gestaut syndrome is a common example of tonic seizures and best treated with broad-spectrum drugs (eg. valproic acid, lamotrigine, topiramate)

- **Myoclonic seizures-** This seizure has a bimodal distribution. Infants with myoclonic epilepsies usually have a poor prognosis. In adolescence, JME is typically a benign process that is treated easily. The best medications for JME and myoclonic seizures are valproic acid, lamotrigine, and topiramate.

- **Primary generalized tonic-clonic seizures-** This seizure type responds to valproic acid, topiramate, or lamotrigine.

- **Partial-onset seizures**-Carbamazepine is considered first-line therapy. However, in special populations, lamotrigine, oxcarbazepine, and topiramate might be better choices. Adjunctive therapy with levetiracetam, tiagabine, gabapentin, or pregabalin might be a choice if the first or second monotherapy trial with first line treatments failed.
- **Non-pharmacologic treatments -** Diet- The ketogenic diet has a role in children with severe epilepsy. One major problem is that <10% of patients continue the diet after a year. Furthermore, any small carbohydrate intake (eg. lollypop, piece of candy) resets ketone metabolism for 2 weeks, eliminating antiseizure efficacy; VNS- The VNS is a palliative device approved to treat medically refractory partial-onset epilepsy in adults.

Discontinuation of anticonvulsants - Seizure free for typically 2-5 years, physicians consider discontinuing the medication. Many patients outgrow many epileptic syndromes of childhood. A normal sleep-deprived EEG and normal brain MRI lower the risk of relapse. Gradually discontinued over 6-10 weeks, if they were used for a long period.

Recurrence risk is high if there is- Abnormal EEG; Abnormal brain MRI ; Several seizure types (worse if tonic or atonic); High number and frequency of seizures; Long duration of epilepsy before the seizures are controlled; Short duration of seizure freedom; About 75% of relapses occur in the first year, and at least 50% of patients do so in the first 3 months

Surgical Care –The two major kinds of brain surgeries for epilepsy are palliative and potentially curative. A few years ago, the most common palliative surgery was anterior callosotomy for patients with intractable atonic seizures.Several curative surgeries are possible, including lobectomy and lesionectomy. Outcomes of temporal-lobe surgeries are better than those for surgeries in other areas.

Activity-Driving, Water precautions, Climbing Heights, Fire, -should be with due care. Helmets to prevent head trauma while the patient is biking, skiing.

Status Epilepticus

It is a medical emergency, requiring organised & skillful approach to minimize morbidity & mortality.It is continous convulsion lasting more than 30 minutes or serial convulsions between which there is no return to consciousness. It has three subtypes-febrile , idiopathic and symptomatic status epilepticus

Management
General Measures
Early status (0-10 minutes), A ,B, C, D-Assess & assist airway; Adequacy of breathing give O2; Adequacy of circulation ,IVF, pressor therapy if necessary ; Drugs most important is 10% dextrose 5ml/kg as rapid infusion

Stage of established status (30-60 minutes)-Transfer to ICU; Regular monitoring of RBS, CBC, BUN, ABG, electrolytes & AED levels if necessary; Treat metabolic abnormality;CTscan if focal sign; LP if meningeal irritation; Metabolic workup if indicated; Identify & treat complications; Treat raised ICT: dexamethasone, mannitol, IPPV; Continuous EEG monitoring if indicated

Anti convulsants-Diazepam upto 0.3mg/kg IV in 2-6 minutes may be repeated; Lorazepam is also used at 0.05 -0.1 mg/kg IV. Less S/E like respiratory depression; if there is no IV line rectal diazepam in 0.5 mg/kg may be given or Sublingual Lorazepam may be given at 0.05-0.1mg/kg; Midazolam 0.15-0.3mg/kg IV may be used. May be repeated after 15 minutes

If no response then give- Phenytoin 10-30mg/kg in 10mg/kg increments @1mg/kg/min; Fosphenytoin-newer prodrug-150mg=100mg phenytoin. Well absorbed IM; Phenobarb 15-20mg/kg@2mg/kg/minute; Paraldehyde 150-200mg/kg iv slowly or4% at 0.1-0.3ml/kg IM; Valproic acid IV at 10-15mg/kg

If seizures are refractory - management is Elective intubation & assisted ventilation; Midazolam infusion-0.2mg/kg bolus; followed by 20-400 microgm/kg/hr infusion; Propofol 1-2mg/kg bolus 2-10mg/kg/hour; Thiopental 2-

4mg/kg bolus then titrated using EEG

Maintenance Therapy

- Phenytoin 3-9 mg/kg in 2divided doses begun 12-24 hours later
- Phenobarb 3-5 mg/kg in2 divided doses
- Lengthy period of ACT not necessary in febrile or idiopathic SE. Treat for 3months & stop if asymptomatic.
- Long term treatment in progressive neuro disorder, history of recurrent seizures.

Prognosis- Mortality rate 5% approximately.Highest incidence in symptomatic group. Long term sequelae more common in younger children &those having pre-morbid condition.

Febrile Seizures

Most common seizure disorder

- Age 3 months to -5years
- No evidence of intracranial / other cause
- Incidence 2-5% before 5 years
- Family h/o in 10% ?AD inheritance
- Recurrence rate 30-50%,mostly infants,
- +ve family h/o or complex febrile seizures
- Risk of epilepsy 9% in those with risk factors &1% in others.

Simple febrile seizures - rapid rise of temperature followed by generalized tonic-clonic seizure lasting few seconds to less than 10 min. Post ictal period brief. Single seizure

Atypical or complex - duration more than 15 min, repeated convulsions on same day, focal seizure or focal deficit postictally

Investigations- if in doubt CSF study, EEG& imaging in atypical seizures.

Treatment-General measures to find & treat cause of fever; Prolonged anticonvulsant prophylaxis no role as risk of drug does not justify it's use; Intermittent prophylaxis with diazepam 0.3mg/kg 8 hourly for 2-3 days when parents anxious regarding seizure; Reassurance of parents of the benign nature & that child will outgrow it.

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Percutaneous Endoscopic Gastrostomy, 1979

When patients have difficulty swallowing, as a result of disease or injury, a feeding tube can be inserted to provide the nutrients to sustain life. In 1979, Micheal Gauderer, a pediatrician from University Hospitals of Cleveland, and Jeffrey Ponsky, a University Hospitals endoscopist, devised Percutaneous Endoscopic Gastronomy (PEG) to insert these feeding tubes that was both inexpensive and low risk. This procedure comprised an attractive alternative to laparotomy, a surgical incision of the abdomen, and soon became a widespread indication for therapeutic endoscopy. The placement of a PEG tube involves but a few ingenious steps. First, a cannula containing a suture is inserted through the skin into the abdomen of the patient. An endoscope is inserted down the esophagus to the stomach and the endoscopist snares the suture from the cannula. The suture is then pulled up through the esophagus and out of the mouth where it is tied to the enteral feeding tube. The tube is then pulled back down through the hole in the stomach wall and skin until it is partly out of the body. The mushroom tip on the internal end of the tube keeps it in the stomach. After the tube is inserted, nutrients may be fed directly into the stomach via syringe after twenty-four hours. PEG could be performed either as an inpatient or outpatient surgery. The procedure eliminated risks associated with laparotomy, including anesthesia complications, infection, and organ rupture. In their review of 150 cases published in the *Archives of Surgery* (August,1983), Gauderer and Ponsky found no deaths as a result of the procedure, and complications (in only ten percent of cases) were minor and easily treated. The apparatus seen here is the PEG feeding tube and a syringe that would be used to administer the nutrients.

Put Feet First—Prevent Amputation by Managing Peripheral Vascular Disease in Diabetes **Commentary**

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Peripheral vascular disease is a narrowing of the arteries. It mainly occurs in leg arteries in diabetes. The main symptom is pain in the legs when you walk. Treatment usually includes: stopping smoking (if you smoke), regular exercise, medication to lower your cholesterol level, a daily aspirin, and lowering blood pressure if it is high. Surgery is a last resort in severe cases.

What is peripheral vascular disease (PVD)?

Peripheral vascular disease (PVD) is narrowing of one or more arteries (Blood vessel). It mainly affects arteries that take blood to the legs. (Arteries to the arms are rarely affected and are not dealt with further in this leaflet.) The condition is also known as 'peripheral arterial disease'. It is also sometimes called 'hardening of the arteries' of the legs.

Who gets peripheral vascular disease?

In the UK, at least 1 in 20 people over the age of 55 have some degree of PVD. It is more common with increasing age.

What causes peripheral vascular disease?

The narrowing of the arteries is caused by atheroma. Atheroma is like fatty patches or 'plaques' that develop within the inside lining of arteries. A patch of atheroma starts quite small, and causes no problems at first. Over the years, a patch of atheroma can become thicker. (It is a bit like scale that forms on the inside of water pipes.)

A thick patch of atheroma makes the artery narrower. This reduces the flow of blood through the affected section of artery. Tissue 'downstream' have a reduced blood supply, which can lead to symptoms and problems.

Atheroma can develop in any artery but the common arteries affected are:

- Arteries taking blood to the heart which may lead to angina or heart attack.
- Arteries taking blood to the brain – which may eventually lead to a stroke.
- Arteries taking blood to the legs – which may lead to PVD.

What causes atheroma?

Many people have some atheroma in various arteries as they become older. It is often a small amount and causes no symptoms. However, there are certain 'risk factors' which increases the chance of atheroma becoming worse and causing problems. Risk factors include:

- Smoking
- Having diabetes
- High fat or cholesterol level in the blood
- Inactivity (little physical exercise)
- High blood pressure
- Obesity.

Smoking and having diabetes are the most serious risk factor for PVD. If you have PVD your doctor is likely to advise that you have a blood test to see if you also have diabetes.

What are the symptoms of peripheral vascular disease?

The typical symptom is pain which develops in one or both calves when you walk. This is called 'intermittent claudication'. It is due to narrowing of the femoral artery—the most common site for atheroma to develop in blood and oxygen supply. The narrowed artery can not deliver the extra blood, and so pain occurs from the oxygen-starved muscles. The pain soon goes when you slow down or stop. The pain comes on more rapidly when you walk up a hill or stairs than when you walk on the flat .If an artery higher 'upstream' is narrowed, such as the iliac artery or aorta, then you may develop pain in your thighs or buttocks when you walk. If the blood supply to the legs becomes

worse, the following may be found by a doctor who examines you:

- Poor hair growth below the knee, and poor toenail growth
- Cool feet
- No pulses in the arteries of feet

Severe cases

If the blood supply is very much reduced, then you may develop pain even at rest, particularly at night when the legs are raised in bed. Ulcers (sores) may develop on the skin of the lower leg if the blood supply to the skin is poor. In a small number of cases, gangrene (death of tissue) of the foot may result. This is usually preventable (see below).

What is the outlook (prognosis) for the peripheral vascular disease?

Studies that have followed-up people with PVD have shown that :

- Symptoms remain stable or improve in about 15 out of 20 cases.
- Symptoms gradually become worse in about 4 out of 20 cases.
- Symptoms become severe in about 1 out of 20 cases.

So, in most cases, the outlook for the legs is quite good. However, if you have PVD is means that you are more likely to form atheroma in other arteries. So, you have a higher than average chance of developing heart disease (such as angina, heart attack) and strokes.

The chances of developing severe PVD (and heart disease, or a stroke) is much reduced by the self help measures and treatment described below.

What 'self-help' measures can I do?

Stop Smoking-If you smoke, stopping smoking is the single most effective treatment. Stopping smoking, increases walking distance by two or three hold in over 8 out of 10 people with PVD. (Stopping smoking also greatly reduces your risk of having a heart attack or stroke).See your practice nurse for help if you find it difficult to stop smoking. Nicotine gum or tablets to help you stop may be an option. There is also a medicine called bupropion which can help you to stop smoking.

Exercise regularly-Regular exercise encourages other smaller arteries in the legs to enlarge and improve the blood supply. If you exercise regularly, there is a good chance that symptoms will improve, and the distance that you can walk before pain develops will increase.Walking is the best exercise if you have PVD. Regular exercise means a walk everyday, or on most days. Walk until the pain develops, then rest for a few minutes. Carry on walking when the pain has eased. Keep this up for at least 30 minutes each day, and preferably for an hour a day. The pain is not damaging to the muscles.Other exercises such as cycling and swimming will also help you to become fit, and are good for the heart. But, these should be done

in addition to walking has been shown to be the best exercise to improve symptoms of PVD.Research studies have shown that – if you stop smoking, and exercise regularly, then symptoms of PVD are unlikely to become worse, and they often improve. Your risk of developing heart disease or a stroke will also be reduced.

Lose weight if you are overweight -Losing weight reduces and demands on the heart and leg muscles.

You should eat a healthy diet-This is the same as advised to prevent heart disease. This reduces the chance of atheroma forming. A practice nurse or dietician may advice you on how to eat a healthy diet. Also, another leaflet in this series called 'Healthy Eating' gives details. Briefly, a healthy diet means:

- Atleast five portions of a variety of fruit and vegetables per day.
- The bulk of most meals should be starch-based foods (such as cereals, wholegrain bread, potatoes, rice, pasta), plus fruit and vegetables.
- Not much fatty food such as fatty meats, cheeses, full-cream milk, fried food, butter etc. Use low-fat, mono or poly unsaturated spreads.
- Include 2-3 portions of fish per week. At least one of which should be 'oily' (such as herring, mackerel, sardines, kippers, pilchards, salmon, or fresh tuna).

- If you eat meat it is best to eat lean meat, or poultry such as chicken

- If you do fry, choose a vegetable oil such as sunflower, rapeseed or olive oil

- Try not to add salts to food, and avoid foods which are salty

Take care of your feet-Try not to injure your feet (Injury may lead to an ulcer or infection developing more easily if the blood supply to the feet is reduced). Do not wear tight shoes or socks which may reduce blood supply. Trim your toenail's straight' across (rather than 'round')

Tell your doctor if you have any foot injury, pain in your feet when you are resting, or any marked change in skin colour or temperature of your feet.

What are the treatments for peripheral vascular disease?

The self-help measures above are the most important part of treatment. In addition, medication is often advised. Surgery is only needed in a small number of cases.

Medicines-Aspirin is usually advised. A daily low dose (75-15 mg) is usual. This dose is not help with symptoms of PVD, but helps to prevent blood clots (thrombosis) forming in arteries. This is an uncommon complication of PVD. However, as mentioned, people with PVD have a higher than average risk of developing heart disease or stroke. A daily low dose of aspirin reduces this risk too.

Clopidogrel is an alternative to aspirin which helps to prevent blood clots. It is usually used in people who are not able to take aspirin. A 'statin' medicine is usually advised to lower the cholesterol level. This helps to prevent a build-up of atheroma.If you have diabetes then good control of the blood glucose level will help to prevent PVD from getting worse.If you have high blood pressure then you will normally be advised to take medication to lower it. Normal blood pressure is less than 140/90 mmHg. (However, if you have diabetes you should aim to have a level less than 140/80 mmHg). Other medicines are sometimes used to try to 'open up' the arteries, for example, niftidrofuryl. These are usually quite disappointing in treating PVD. So, they are not commonly used. But, there is no point in continuing with these medicines if you do not notice an improvement within a few weeks. However, a newer medicine called cilostazol has recently been shown to improve walking distance in people with PVD. It has several actions which seem to improve the blood flow in the lower leg. The role of this medicine is not fully established, but it is likely to be used in people who have quite severe symptoms which do not improve with the measures described above.

Surgery -Most people with PVD do not need surgery. Your GP may refer you to a surgeon if symptoms of PVD become severe, particularly if you have pain when you are resting. Surgery is considered a last resort. Surgery is not easy and not without possible complications. There are 3 main types of operations for PVD. Angioplasty is where a tiny 'balloon' is inserted into the artery and 'blown up' at the section that is narrowed. This widens the affected segment of artery. This is only suitable if a short segment of artery is narrowed. Bypass surgery is where is graft (like a flexible pipe) is connected to the artery above and below a narrowed section. The blood is then diverted around the narrowed section. Amputation of a foot, or lower leg, is needed in a small number of cases. It is needed when severe PVD develops, and a foot becomes gangrenous due to a very poor blood supply.

Thyroid Gland- Anesthetic Implications

Commentary

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Although the term thyroid is derived from the Greek word meaning shield, the gland is most commonly described as 'butterfly' shaped. The thyroid gland lies in the neck related to the anterior and lateral parts of the larynx and trachea. Anteriorly, its surface is convex; posteriorly, it is concave. It is composed of two lobes joined by an isthmus. The isthmus lies across the trachea anteriorly just below the level of the cricoid cartilage. The lateral lobes extend along either side of the larynx as roughly conical projections reaching the level of the middle of the thyroid cartilage. Their upper extremities are known as the upper poles of the gland. Similarly, the lower extremities of the lateral lobes are known as the lower poles. The gland is brownish- red due to a rich blood supply. Dietary iodide is concentrated by the thyroid gland and is oxidized by the enzyme peroxidase, in the follicle cells, to iodine. This is inhibited by thiocynate & perchlorate. The iodine is linked to tyrosine molecules to form monoiodotyrosine & diiodoty-rosine, latter are coupled to form tri-iodothyronine (T^3) and tetraiodotyronine [thyroxine (T^4)] which are then released into the circulation. This coupling is inhibited by thioureas. Anti-thyroid drugs also block the synthesis of T^3 and T^4 by interfering with various steps of this process. All the steps in the synthesis of thyroid hormones are stimulated by thyroid stimulating hormone (TSH) secreted from the anterior pituitary gland. The major fraction of circulating is bound to thyroid- binding glob-ulin(TBG), with a smaller fraction bound to albumin and thyroid-binding prealbumin. Less than 0.1% is present as free, unbound hormone. T^4 is transported in the blood bound to plasma proteins, mainly thyroglobulin and little amount are bound to prealbumin. T^4 is metabolized by monodeio-dination to T^3 or reverse T^3 (rT^3). T^3 is biologically active where as rT^3 is inactive. The half-life of T^4 is 7 days and the half life of T^3 is 24 -30 hrs. TRH from Hypoth-alamus stimulates synthesis and release of TSH from the Pitutary. Circulating TSH stimulates thyroid gland to produce and secrete T^4 (about 80%) and smaller amounts of T^3 (about 20%). Remainder of T^3 is produced in extra thyroid tissue (liver/kidney). T T^3 feedback on pituitary to inhibit production of TSH.

Thyroid Swelling - Case Presentation

History

- Age - graves disease (thyrotoxicosis) seen in 20-40 years of age while hypothy-roidism is more common in elderly females.

- Sex- more common in females; incidence of hypoth-yroidism in general is 3- 5 %

- Occupation - thyrotoxicosis occurs more in persons working under stress

- Residence - endemic areas

- Swelling -onset, duration, rate of growth, whether asso-ciated with pain, any sudden increase in size with pain

- Sleep disturbance - like complaints of sleeplessness as in primary thyrotoxicosis

- Pain - more common in inflammatory conditions and anaplastic carcinoma.

- Pressure Effects - dyspnoea, dysphagia, hoarseness of voice, stridor

- Symptoms of Primary Thy-rotoxicosis - loss of weight, preference for cold and intolerance to heat and excessive sweating, CNS involvement - nervous excita-bility, irritability, insomnia, tremor of hands and weakness of muscles, Cardiovascular symptoms- palpitation, tachycardia. and dyspnoea on exertion.

- Symptoms of Secondary Thyrotoxicosis - more of cardiovascular system inv-olvement - palpitations, irregular heart beats (ectopics), dyspnoea on exertion, chestpain, signs of CCF like ankle swelling, oliguria.

- Symptoms of Hypothyroidism- increase in weight despite poor appetite, somnolence, intolerance to cold, dryness of skin, facial puffiness dull expression, loss of hair, muscle fatigue and lethargy, failing memory and hoarseness of voice, Constipation and Oligomenorrhoea.

- Past history - Course of treatment and its effect on the swelling history of ingestion of goitrogenic drug e.g. PAS or sulphonylureas or antithyroid drugs, which are goitrogenic, history of of surgery, external neck radiation(for hypothyroidism).

- Personal History - Dietary habits, Goitrogenic Vegetables e.g. cabbage.

- Family History - History of similar occurrences in family, endemic goiter.

Clinical Examination

Physical examination-Build and state of Nutrition; Facies: hypothyroid patients may have mask like facies; Mental state and Intelligence; Pulse Rate- rate, rhythm, regularity, Sleeping Pulse Rate; Skin — hot and moist or dry and inelastic; Tremor

Inspection

- Pizzillo's Method: Hands are placed behind the head and the patient is asked to push her head backwards against her clasped hands on the occiput.

- Diffuse Enlargement

- Nodular Enlargement

- Movement with Deglutition -ask patient to swallow

- Movement on protrusion of tongue

- Check for Retrostenal goiter -Any dilated subcutaneous veins over the upper anterior part of the thorax; Determine lower border of swelling on deglutition; Pemberton's sign; Position of Trachea

Palpation

- Palpation of the entire gland - from front and from behind

- Palpation of each lobe— Lahey's method.

- Crile's method

- During palpation the following points to be noted: Whether the whole thyroid gland is enlarged - note its surface, its consistency; In localized swellings-note position, size, shape, extent

and its consistency; Mobility of the swelling; Ability to get below the thyroid gland

- Position of the larynx and the trachea should be noted

- If pressure on trachea is suspected elicit Kocher's test

- Feel for Carotid pulse

- Palpation of cervical lymph nodes

Percussion - Over the manubrium sterni to exclude retrosternal goiter

Auscultation -Any systolic bruit over thyroid - as in primary toxic goiter due to increased vascularity

General Examination-Look for causes, signs and symptoms of hyperthyroidism(excessive amount of circulating thyroid hormone) and hypothyroidism(inadequate circulating levels of T^4 or T^3 or both).

Hyperthyroidism	Hypothyroidism
CausesGraves disease (TSI - ?LATS), Paranodular graves, Multinodular goitre, Toxic nodule, Hypersecretion of TSH, iatrogenic due to thyroid hormone replacement& iodine therapy. Pregnancy, trophoblastic tumors, TSH secreting pituitary adenomas, thyroid carcinoma, Jodbasedow phenomenon: antiarrythmic agent : amiodarone which is iodine rich	Primary failure of thyroid gland refers to decrease production of thyroid hormone despite adequate Tsh production & accounts for 95% of thyroid dysfunction. causes being Idiopathic, Iatrogenic - Surgery, Radioactive iodine external neck radiation. Antithyroid R$_x$ Iodine deficiency. Hashimotos, De-Quiervan's, Reidals thyro-iditis Drugs - Li, cholest-ryramine, SNP, Amiodarone, phenytoin, phenobarbitone Secondary or Tertiary Hypothy-roidism is caused by hypoth-alamic or pituitary disease

Symptoms Fatigue, Weight loss, Increased appetite, Heat intolerance, nervousness Palpitations, sweating, anxiety, irritability, diarrhoea, Oligo/ Amenorrhoea	Weight gain, Anorexia, Cold intolerance, fatigue, constipation, Menorrhagia, Dry skin, Hair loss
Signs General Thin, anxious pt, warm moist skin, palmar erythema, telangectasia, fine soft skin, hair, nails, clubbing (acropachy), pretibial myxoedema	Hypothermia, Puffy face, Periorbital odema, large tongue, coarse dry skin, carotenaemia, hoarseness, deafness, slow mentation, confusion, coarse sparse hair, loss of outer third of eyebrows
Cardiaorespiratory SVT with atrial fibrillation Resting pulse > 90bpm (sinus tachycardia), High output failure ,tremors	Sinus bradycardia, prolonged QT, low cardiac output, CCF pleura effusion and cholesterol rich pericardial effusion producing low voltage ECG, hypoventilation, ventilatory responsiveness to hypoxia and hypercapnia is depressed which is potentiated by sedative, opoids & GA
Peripheral Fine static tremor, Proximal myopathy leading to muscle weakness and stiffness	Carpal tunnel syn. Pendular reflexes (delayed deep tendon reflexes)
Eye signs Lid Retraction ; Exophthalmos-Von Greene's sign, Joffroy's sign, Stellwag's sign, Moebisign, Dalrympte's sign; Ophthalmoplegia; Chemosis	
Other Osteoporosis, myopathy	Megaloblastic anaemia, sleep apnea, myopathy ,concomitant adrenal insufficiency, constipation, urinary retention due to decreased motility, impaired renal free water clearance with hyponaterimia

Investigations

Thyroid Function Tests

- Serum Protein Bound Iodine- $\bar{}$ in thyrotoxicosis, pregnancy; $\bar{}$ in hypothyroidism

- Hormone assay- T^4, T^3, Free T^4, Free T^3 are all raised in hyperthyroidism and are good indicator of latter.

- T^3Resin Uptake Test, **T^3RU**- uptake inversely proportional to conc. of unoccupied sites on thyroglobulin. $\bar{}$ in hyperthyroidism , $\bar{}$ in hypothyroidism

- FTI- T^4 x T^3RU (% of control), $\bar{}$ in hyperthyroidism , $\bar{}$ in hypothyroidism

- Serum Thyroid Stimulating Hormone (TSH): increased in hypothyroidism & is the best indicator. sub clinical hypothyroidism is a condition characterized by elevated TSH and normalT^4

- RAIU- used to confirm hyperthyroidism in which it is raised.

- Urinary RAI excretion- increased in hypothyroidism

- Thyroid Scan- delineates active thyroid tissue and diagnoses retrosternal extension.

- Miscellaneous Test- BMR(after 12 hrs fasting), CBC, S.electrolyte, S.cortisol, ABG, S.cholesterol, S.creatine, Measurement of tendon reflexes, ECG

- Radiology of upper airway- CXR to diagnose position of trachea and for pleural/ pericardial effusion; STN - AP and Lateral view for

radiology of airway, CT scan , ultrasound

- Indirect laryngoscopy

Differential Diagnosis - Clinching points of a thyroid swelling- Its position, shape and upward movement during deglutition

Classification of goiter

- Non-toxic goiter-1. Diffuse parenchymatous 2. Colloid 3.Multinodular 4. Solitary nodular

- Toxic goiter-1, Diffuse (Grave's Disease) 2.Multinodular 3.Toxic nodule

- Neoplastic-.Benign; Malignant. Functioning (hot) thyroid is rarely malignant. non functioning(cold) tissue may be malignant or benign.

- Thyroiditis -Acute bacterial; Granulomatou ; Autoimmune;Riedel's

Medical Management (for hyperthyroidism)

Beta-blockade- most rapid method of reversing symptoms by blocking beta catecholamine receptors effective within 12 - 24 hrs; doesn't inhibit hormones synthesis but impairs peripheral conversion of T4 to T3 over 1-2 weeks; Beta-blockade combined with iodide (or lithium) can achieve euthyroid state in 1 -2 weeks but cardiac effects take longer to resolve; usually only used to tide over while other therapies take effect; should not be used routinely in patients with symptoms of congestive heart failure and bronchospasm.

Antithyroid drugs- antithyroid thyroid drugs are thiourea derivatives that inhibit the synthesis of thyroid hormone but if given alone take atleast 6- 8 weeks to achieve euthyroid state.

- **Methimazole / Carbimazole**- carbimazole is the prodrug of methimazole; iodinated molecule blocks iodination of tyrosine residues; effects seen after 3 - 4 weeks; can be used as the sole therapy for hyperthyroidism: given for a period of 12 -18 months but relapse rate >50%; S/E – skin rash, arthralgia, N&V , fever, hepatitis & lupus like syndrome; agranulocytosis: reversible

- **Propylthiouracil** - mechanism of action: as for carbimazole and blocks peripheral conversion of T4 to T3; faster onset of action of carbimazole; S/E - same as carbimazole; can convert from one drug to the other if SFx a problem

- **Ablative Therapy - Radioactive Iodine (I^{131})** - I^{131} concentrates in the thyroid and destroys functioning cells; takes 6 -10 weeks for clinical effects; repeat doses often necessary ; pregnancy an absolute contraindication because it crosses the placenta and may destroy fetal thyroid ; hypothyroidism can occur up to years after therapy; 10- 60% of cases occurs in the first yr of therapy and additional 2% occur per year thereafter; no evidence for inherited genetic damage in babies if mother has had therapy in the past ;

- **Inorganic Iodide**-inhibits iodide organification and thyroid hormone release; Effective in reducing size of hyperplastic gland and helps in the preparation of patient for emergency thyroid surgery

- **Glucocorticoids**-dexa methasone (8-12 mg) per day used in the management of severe throtoxicosis; it reduces thyroid hormone secretion and reduces the peripheral conversion of T^4 to T^3

Surgery-Due to I^{131}, surgery for hyperthyroidism(subtotal thyroidectomy) is less commonly required now than in the past. Complications include-hypo- (or occasionally hyper-) thyroidism ; hypoparathyroidism

Medical Management (for hypothyroidism)

Medical Therapy - Thyroxine 50 - 200 ug/day ; IV T^3 in emergency; Caution with thyroxine replacement if IHD because Acute MI has been precipitated by treatment of hypothyroidism: add beta-blocker early and keep to low dose of thyroxine; Caution with digoxin dose in CCF as increased contractility may provoke ischeamia

Surgery- Subtotal or total thyroidectomy

Anaesthetic Implications

Hyperthyroidism

- Except for absolute emergency surgery, all patients should be clinically euthyroid prior to being exposed to the stress of surgery

- Pharmacological stabilisation of hyperthyroid patient requires at least 6 -8

weeks Beta - blockade combined with iodide (or lithium) can achieve euthyroid state in 1 -2 weeks but cardiac effects take longer to resolve..

- All antithyroid medications are continued through the morning of surgery

- Goal of intraoperative management in the hyperthyroid patient is to achieve a depth of anaesthesia that prevents an exaggerated sympathetic response to surgical stimulation, while avoiding the administration that stimulates the sympathetic nervous system

- Ketamine to be avoided even if the patient is euthyroid

- Induction: agent of choice thiopentone sodium as it has anti thyroid activity due to thiourea molecule.

- Hypotension during surgery is best treated direct acting vasopressor rather than a medication that provokes the release of cathecohlamine

- Appropriate NM blocking drugs should be selected; pancuronium having the ability to increase the heart rate should be avoided; muscle relaxant providing greater CVS stability (VECURONIUM, ATRACURIUM) should be used.

- Incidence if myasthenia gravis is increased in hyperthyroidism ; thus the initial dose of muscle relaxant should be reduced and a twitch monitor should be used to guide all subsequent administration of NM blocking agents

- MAC of inhalational anaesthetic agents is not increased but the requirement of inhalational anaesthetic agents as such is increased due to - cardiac output, - temperature & ? CNS excitation

- Intraoperatively HR, Temp, IBP, ETCO2, SpO2, ABGs should be monitored

- care of exophthalmic eyes to be taken intraoperatively / tarsorrhaphy

- Regional anaesthesia is an excellent alternative when appropriate : however epinephrine containing solutions should be avoided.

Thyroid Storm

- Life-threatening exacerbation of hyperthyroidism that may occur during or immediately after surgery; provoked by operating on an acutely hyper thyroid patient; it is probably not due mechanical release of hormone

- Onset is usually abrupt and precipitated by stress of surgery or non thyroid illness in an undiagnosed hyperthyroid patient

Clinical features

- fever, '!CO2 production, acidosis, hyperventilation

- CVS: tachycardia, dysrhythmia, CCF, shock

- CNS: agitation, tremor, delerium, coma

- GIT: diarrhoea, abdo. pain, vomiting

- No lab test is diagnostic although free T^4 levels are often markedly elevated management

- 02, active cooling (aspirin to be avoided as it displaces T4 from TBG)

- intravascular volume should be restored; PA catheter & arterial catheter is especially useful in guiding the treatment of patients with left ventricular dysfunction

- Beta-blockade: I.V. Propanolol 1-5 mg promptly treats fever, tachycardia, tremor; does not reduce O2 consumption

- Glucocorticoid Rx often recommended due to possible "adrenocortical exhaustion"

- Iodide IV (as KI 60mg bd or NaI 1.0 - 2.5 g) rapidly controls thyrotoxicosis

- Can use lithium if allergic to iodide

- Large doses of propylthiouracil should be commenced immediately

- removing or treating the precipitating event is essential DDx

- Phaeochromocytoma (Beta-blockade alone contraindicated)

- Malignant hyperthermia

- Light anaesthesia

Emergency Surgery in hyperthyroid patient

- b adrenergic blockade should be administered to achieve a heart rate dH 90 bpm
- Glucocorticoids such as dexamethasone (8-12 mg) per day used in the management of severe throtoxicosis because they reduce thyroid hormone secretion and reducethe peripheral conversion of T^4 to T^3
- Start anti-thyroid Rx as soon as diagnosis made (in conjunction with specialist endocrinologist)
- preoperative sedation with anxiolytics
- in the days before medical stabilisation preop was rigidly adhered to, they talked about "stealing" the patient from the ward: unknown to the patient, a barbituate was added to their IV fluids and the drowsy patient was then stolen away to the operating theatre! **Thyroid Steal**

Hypothyroidism

- Controversy remains regarding the preoperative anesthetic management of the hypothyroid patient with regard to all hypothyroid surgical candidates being restored to a euthyroid state before surgery
- Thyroid replacement therapy is indicated for patients with severe hypothyroidism or myxoedema coma & for pregnant patients who are hypothyroid. Untreated hypothyroidism in pregnant patients is associated with an increased incidence of spontaneous abortion and mental and physical abnormalities in the offspring.
- There appears to be little reason to postpone elective surgery in patients who have mild or moderate hypothyroidism
- Regarding management of hypothyroid patients with symptomatic coronary artery disease, the need for thyroid hormone replacement therapy must be weighed against the risk of precipitating myocardial ischemia.
- In symptomatic and unstable patient with cardiac ischemia, thyroid replacement should prob. be delayed until after coronary revascularization

Anaesthetic Considerations

- possibly increased sensitivity to sedatives - usually little or no sedative premed is needed
- increased risk of hypothermia, hypoglycemia, hyponatreamia, anaemia, hypovolaemia, adrenocortical insufficiency
- gastric paresis may be there, therefore always consider Rapid Sequence Induction and aspiration prophylaxis
- thiopentone is used as the induction agent although ketamine has been proposed as the ideal induction agent
- maintenance of anaesthesia is safely achieved with either IV or inhaled anaesthetics, there is little if any decrease in the MAC for volatile agents; intraoperativaly ventilation should be supported and intravascular volume should be well maintained to avoid any hypotension
- due to skeletal muscle weakness NM blocking agents should be used cautiously under NM monitoring; NM blockade must be always reversed
- monitoring is directed towards the early recognition of hypotension, CCF and hypothermia; scrupulous attention should be paid to maintaining normal body temperature
- postop ventilatory failure requiring prolong ventilation is rarely seen in hypothyroid patients in the absence of coexisting lung disease, obesity, myxoedema coma
- possibility of prolonged recovery
- monitor ventilation in postoperative period
- regional anaesthesia is a good choice provided that the intravascular volume is well maintained

Myxoedema Coma

- is decompensated hypothyroidism ;life threatening complication of severe hypothyroidism characterized by a decreased level of consciousness, even coma Management
- Thyroid hormone replacement; IV T^3 & T^4 under strict ECG monitoring to be given as oral absorption is unreliable
- General supportive measures: IV fluids to restore intravascular volume, passive warming (never active),

mechanical ventilation, treat any seizures, use sedatives cautiously, Hydrocortisone 100mg 8 hourly till evalu. of hypothalm-pit- adr axis

- Cardiovascular supportive measures: inotropes after restoring intravascular volume, monitor for arrhythmia, monitor for MI which may be precipitated by thyroid hormone replacement therapy

Thyroid Surgery

- subtotal thyroidectomy as an alternative to prolong medical therapy is used less frequently than in the past
- airway obstruction is a potential problem in the patient with a large substernal goiter, although rarely a problem with goiters exclusively in the neck
- usually performed under general endotracheal anaesthesia although the use of Laryngeal mask airway(LMA) is increasing
- use of LMA allows real time visualization of vocal cord function because the patient is allowed to breath spontaneously.
- complications after subtotal thyroidectomy include recurrent laryngeal nerve damage, tracheal compression secondary to hematoma or tracheomalacia and hypoparathyroidism. Latter is most frequently seen after total thyroidectomy
- postop hematoma formation requires immediate evacuation
- hypocalcaemia typically develops 24 -96 hrs after surgery manifested by laryngeal stridor progressing

to laryngospasm; IV Calcium chloride and gluconate given immediately if clinically detectable hypocalcaemia is present, magnesium levels should be monitored corrected if low

- bilateral recurrent laryngeal nerve injury is an extremely rare injury(1/30,000) causes aphonia and necessitates reintubation
- unilateral recurrent laryngeal nerve damage is more common and is often transient and is characterized by hoarseness and paralyzed vocal cords; vocal cords function should be evaluated before and after surgery by laryngoscopy or by asking the patient to phonate by saying the letter "E"
- postop extubation of the trachea should be perfomed under optimal conditions; intraoperative recurrent laryngeal nerve injury or collapse of tracheal rings from previous weakening may mandate emergency reintubation

Hippocrates contribution to medicine

Tradition knows seven physicians named Hippocrates, of whom the second is regarded as the most famous. Of his life we know but little. He was born at Cos in 460 or 459 B.C., and died at Larissa about 379. How great his fame was during his lifetime is shown by the fact that Plato compares him with the artists Polycletus and Phidias. Later he was called "the Great" or "the Divine". The historical

kernel is probably as follows: a famous physician of this name from Cos flourished in the days of Pericles, and subsequently many things, which his ancestors or his descendants or his school accomplished, were attributed to him as the hero of medical science. The same was true of his writings. What is now known under the title of "Hippocratis Opera" represents the work, not of an individual, but of several persons of different periods and of different schools. It has thus become customary to designate the writings ascribed to Hippocrates by the general title of the "Hippocratic Collection" (Corpus Hippocraticum), and to divide them according to their origin into the works of the schools of Cnidus and of Cos, and of the Sophists. How difficult it is, however, to determine their genuineness is shown that even in the third century before Christ the Alexandrian librarians, who for the first time collected the anonymous scrolls scattered through Hellas, could not reach a definite conclusion. For the development of medical science it is of little consequence who composed the works of the school of Cos for they are more or less permeated by the spirit of one great master. The secret of his immortality rests on the fact that he pointed out the means whereby medicine became a science. His first rule was the observation of individual patients, individualizing in contradistinction to the schematizing of the school of Cnidus.

Portal Hypertension

Commentary

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Normal portal venous pressure is approximately 4-8 mm Hg. Increase in portal venous pressure above normal is portal hypertension. The portal vein is formed by the confluence of splenic and superior mesenteric veins. Its flow rate normally averages about 1-1.2 L/min.

Sites of portal obstruction- Portal pressure is the product of portal blood flow and intrahepatic resistance. PH= Portal blood flow x intrahepatic resistance.The Portal flow is increased in post-surgical or post-traumatic and in Splenic arteriovenous fistula

Causes of portal hypertension

- **Extrahepatic postsinus-oidal -** Budd Chiari Syndrome
- **Intrahepatic postsinus-oidal-**Veno-occlusive disease
- **Sinusoidal-**Cirrhosis, Cystic liver disease, Metastasis malignant disease
- **Intrahepatic presin-usoidal-**Schistos-omiasis, Sarcoidosis, Congenital hepatic fibrosis, Vinylch-loride, Drugs
- **Extrahepatic presin - usoidal -** Portal vein thrombosis, Abdominal trauma, Pancreatic malign-ancy, Congenital

History to assess cause of Portal Hypertension

- **Past History -** Jaundice, Blood transfusion, IV drug abuse (Hepatitis B and C), Pruritus – PBC, Hereditary liver disease (Haemoc-hromatosis), Wilson's disease
- **History Suggestive of Complications of Portal Hypertension-** Haemetemesis or Melena, Mental status (encepha-lopathy), Increasing abdo-minal girth (ascites), Abdominal pain and fever (SBP), Hematochezia (blee-ding from portal colopathy)

Physical Signs-Signs of porto-systemic collaterals-Anterior abdominal wall dilated veins – (umbilical epigastric vein); Venous pattern on the flanks (portal – parietal peritoneal shunting); Caput medusa (tortuous collaterals around the umbilicus); Rectal hemorrhoids; Para-umbilical hernia

Signs of Liver Disease-Ascites, Jaundice, Spider angioma, Asterixis, Testicular atrophy, loss of libido and hair loss, Gynaecomastia, Dupuytren contracture, Muscle wasting, Splenomegaly, Hemorrhagic tendency: bruises, purpura, epistaxis, menorrhagia. Other features: Pigmentation, clubbing and low grade fever

Signs of Hyperdynamic Circulatory State-Bounding pulses, Warm well perfused extremities, Arterial hypotension

Laboratory Investigations in a case of Portal Hypertension- LFT, PT, Albumin / globulin, Viral hepatitis serologies, Platelet count decreased, ANA, AMA, ASM, IRM indices, Alpha antitrypsin deficiency, Cerulopl-asmin, 24 hour urinary copper (in hepatic + neurologic + psychiatric patients only)

Imaging Studies

- **USG with Duplex Doppler - Nodular liver-**Presence of collateral + splenomegaly; Doppler demonstrate portal flow helps in diagnosing cavernous transformation, thrombosis of portal or splenic vein.Limitations of USG-Reproducibility of data is a problem. Many variables circadian, meals, medications and portal hemodynamics, interobserver and intraob-server variations.
- **CT Scan and Spiral CT-** Three dimensional angiogra-phy visualize more accura-tely. Collaterals arising from portal system and dilatation of IVS are suggestive of portal hypertension. Limit-ation of CT-Cannot demonstrate venous and arterial flow profile. IV

contrast material cannot be used with renal failure. MRI / MRI angiography same as above.

- **Liver-Spleen Scan -**Using sulfur colloid technetium – not used now a days.Selective angiography of superior mesenteric artery or splenic artery.Hemodynamic measurement of portal pressure.

Endoscopy UGI for Oesoph-ageal Varices

Liver Biopsy - Risk of oesophageal variceal bleeding; Portal pressure > 12 mm Hg varices formed; Intrathoracic pressure gradient – coughing, sneezing or straining ;Damage to variceal wall by acid reflux.

Variceal size- Small varices does not bleed. Big varices bleed. Red and blue colour varices bleed.

Localized thinned walled blebs or Sacs look like red spots, streaks and they bleed. Diffuse pronounced blue colour indicate a large varix with stretched mucosa.

Non-Variceal Sources of Bleeding (30-50% of bleeding)-Portal hypertensive gastropathy; Increased incidence of acid peptic disease

Common sites of portal systemic collateral formation

Location	Portal Circulation	Systemic Circulation	Consequence
Proximal stomach and distal oesophagus	Coronary vein of stomach	Azygous vein	Submucosal G-E varices
Anterior abdominal wall	Umbilical vein in falciform ligament	Epigastric abdominal vein	Caput medusae
Retroperitoneal	Splenic vein branch of Sappey's veins (around liver and diaphragm)	Left renal vein	Usually none
		Retzius vein	Usually none
Anorectal	Middle and superior hemorroidal	Inferior hemorroidal vein	Hemorrhoids

Comparison of portal hypertensive gastropathy and inflammatory gastritis

	Portal hypertensive gastropathy	Inflammatory gastritis
Endoscopic appearance site	Mosaic pattern speckled red spots	Disease erosive lesion
Site	Fundus	Antrum
Histology	Scan inflammatory artery cells. Vasdilatation mucosal and submucosal lesion	Heavy inflammatory cell mucosal lesions
Treatment	Surgery? Beta blockers? Cytoprotective agents	Acid suppression Cytoprotective agents

Management - Oesophageal bleeding is due to varices. It stops spontaneously in 40%. But can re-bleed in 40% of patients. The associated mortality is 30%.

Initial Resuscitation-Replace blood volume, replace blood, modest target of HCT 25-30%.Avoid intravascular volume overload and variceal expansion.Diagnose source of bleeding.Prevent complications (hepatic encephalopathy, bronchial aspiration / SBP).

Prophylactic antibiotic use decrease - Rate of complications. Norflox, ciprofloxacin, specific treatment for bleeding lesions

Pharmacological- Somatostatin – splanchnic vasoconstrictor; Oestreotide is a synthetic analogue of somatostatin 50 mcg/h reduce bleeding; Vasopressin potent splanchnic constrictors. Should be accompanied; Adding nitrates to vasopressin significantly improve; Terlipressin is a synthetic analogue fewer side effects, compared to vasopressin.

Endoscopy Therapy -Hemostasis is good. Inj. Sclerosing therapy obliterating the lumen by thrombosis. The scleroscent available are- 5% sodium tetradecyl sulphate, 5% ethanolamine oleate; the volume of sclerosing agent used is about 10-15 ml. The complications of sclerosing therapy: Dysphagia, perforation, chest pain and mediastinum and ulceration.

- Endoscopic variceal ligation (EVL) is achieved by a banding device 1-3 bands are used. EVL is less prone to complications than sclerotherapy.

- Balloon tube tamponade should be used only in massive bleeding, definitive treatment can be instituted. An endotracheal tube should be placed before attempting to place the balloon tube. Complications are aspiration pneumonia, oesophageal perforation. Continued bleeding indicates an incorrectly positioned tube or bleeding from another site.

- The Minnesota tube has 4 lumens for gastric aspiration, 2 to inflate balloon and 1 above oesophageal balloon to suck secretion, inserted through the mouth. The gastric balloon is inflated, the gastric balloon is pulled up against the oesophageal gastric junction compressing the submucosal varices.

Primary Prophylaxis - Propranolol and nadolol.25% reduction in bleeding rate occurs. Dose of 20 mg every 12 hourly until 25% reduction in the resting heart rate occurs. Average dose is 40 mg BD. Nadolol dosing is half the dose of propranolol. Propranolol is contraindicated in asthma, COPD, AV block, intermittent claudication and beta blockers are continued life long.Prophylactic sclerotherapy has no advantage than controls. But prophylactic EVL is more effective than no treatment. Combination of EVL and pharmacotherapy is better in preventing variceal bleed.

TIPS (Transjugular Intrahepatic Portal Systemic Shunt) – It is a viable option and is less invasive for those recurrent bleeds. Staple transaction of the oesophagus is an option when pharmacotherapy fails. The contraindication for TIPS is the patient who had hepatic encephalopathy in the past. It is indicated in **-**Continued bleed despite medical therapy in child class C disease; Selected cases with class B; In portal hypertension of hepatic origin; Metal stent (1 cm diameter) is introduced through the tract created in the liver parenchyma via the interjugular vein cannula under fluoroscopy and USG. Portal vein à sinusoidal bed à hepatic veins à a shunt is created. The causes of recurrent portal hypertension and bleeding after TIPS are **-**Stent dysfunction; Hemobhilia. The complications of TIPS (Transjugular Intrahepatic Portal Systemic Shunt)-Perihepatic hematoma, portobiliary fistula, arterial portal fistula, increased bacteremia.

Surgery in portal hypertension

- **Total portal systemic shunts-**Shunt of 10 mm between portal vein tributaries and IVC; ECK fistula, end-to-side portacaval shunt; Side-to-side portacaval shunt

- **Partial Portal Systemic Shunts-**Selective shunt-Splenorenal shunt for refractory variceal bleed; Devascularization procedures – Sugiora procedure – gastrooe-sophageal devascularization,

splenectomy, gastro oesophageal transaction.

- **Splenectomy**

- **Liver transplantation –** orthotopic liver transplantation

Ascites in Portal Hypertension-It is seen with cirrhosis. The causes of ascites are- Malignant disease – hepatic, peritoneal; CCF; Cirrhosis of liver; Hyperproteinemia-Nephrotic syndrome,Protein loosing nephropathy, Enter-opathy, Malnutrition; Infectious tuberculosis, spontaneous bacterial peritonitis; Hepatic venous obstruction. Budd Chiari syndrome, veno-occlusive disease; Pancreatitis; Lymphatic obstruction; Rare Causes-Meig's syndrome, Vasculitis, Hypothyroidism, Renal dialysis

Pathogenesis of Ascites- Sodium and water retention is due to high venous pressure. Splanchnic vasodilatation – reduction in circulating volume – leading to activation of rennin angiotensin, increased sympathetic nervous activity, alternation in atrial natriuretic hormone secretion, altered activity of Kalikreinkinin system. Ascites fluid is transudate in portal hypertension, but it can be exudates with superaided TB infection / spontaneous bacterial peritonitis and if hepatic cellular carcinoma develops.

Leeven Shunt for Ascites- Peritoneovenous shunt. The complications are - Septicemia / CCF / DIC and thrombosis of shunt.

Management of Ascites- Remove precipitating factors- Excess of salt intake, mediation non-compliance, superimposed infection, worsening of liver disease, portal vein thrombosis or development of hepatocellular carcinoma.Paracentesis to see nature of fluid and also to relieve respiratory discomfort. Goal is the loss of no more than 1.0 kg/ day if both ascites and oedema present and 0.5 kg if only ascites is present.Salt restriction, diet containing sodium 800 mg (2 gm NaCl).Fluid restriction 1000 ml/ day correct hyponatremia.Spironolactone, traimferene, amilioride, azotemia / hyperkalemia reduce above diuretics. If response poor add proximal acting diuretics – furosemide, bumetamide, torsemide, spironolactone 100 mg/day. Total maximum dose 400 mg/day. Furosemide 40 mg/ day – 160 mg/day.

For refractory ascites- Portacaval shunt side-to-side; Peritoneal venous shunt

Hepatic encephalopathy- This may be due to **–**Uremia, Drugs: sedative, antidepressive and hypnotics; GI bleeding; Excess dietary / notes; Constipation; Paracentesis (Vol. 3-5 litres); Hypokalemia; Infection; Trauma, portosystemic shunts. The pathogenesis is -Ammonia; False neurochemicals transmitters – Octopamine; GABA level increased – Flumazenil, benzodiazepine antagonist; Hypokalemic alkalosis

Clinical Grading of Hepatic Encephalopathy

Grade-I- Poor concentration, slurred speech, slow mutation, sleep disorders

Grade-II- Drowsy but rousable, occasional aggressive behaviour, lethargic

Grade-III- Marked confusion, drowsy, sleepy but responds to pain and voice

Grade-IV- Deep coma, unresponsiveness

Management of Hepatic Encephalopathy- Remove precipitating causes;Reduce protein intake – less than 20 gm per day;Suppress production of neurotoxin by bacteria in the bowel; Glucose 300 gm/day orally or IV; Lactulose 15-30 ml reaches colon metabolized by colonic bacteria. Leads to diarrhea produces osmotic laxative effect reduces colonic pH and limit colonic ammonia absorption; Neomycin 1-4 gm 6 hourly, reduces bacterial contents poorly absorbed; Liver transplantation.

Conclusion-Commonest cause of portal hypertension is cirrhosis of liver. Complications of portal hypertension and diagnosis and their medical and surgical management are discussed.

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Cardio-Pulmonary Resuscitation - Advanced Cardiac Life Support

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The basic principle behind Advanced Cardiac Life Support (ACLS) is good Basic Life Support (BLS), beginning with Cardiopulmonary Resuscitation (CPR) and, for Ventricular Fibrillation (VF) / pulseless Ventricular Tachycardia (VT), early defibrillation within minutes of collapse. VF, rapid VT, Pulseless Electrical Activity (PEA), and asystole are rhythms that produce pulseless cardiac arrest. Survival from these arrest rhythms requires timely action including BLS and ACLS. Basic and early CPR takes precedence over administration of drugs though it remains an important aspect of the final survival of the collapsed victim. After beginning CPR and attempting defibrillation, secure intravenous access and consider drug therapy. Insert an advanced airway whenever feasible but is not to be given priority over CPR including uninterrupted cardiac massage. Central line access is not needed for resuscitation attempt. If peripheral intravenous (iv) access could not be secured then intraosseous (io) cannulation is to be attempted. When drug is given via peripheral line, it should be flushed with 20 ml bolus of iv fluid and elevate the extremity for 10-20 sec to facilitate drug delivery to the central circulation. If iv or io access can't be secured,

some drugs (lidocaine, epinephrine, atropine, naloxone, and vasopressin) may also be given via endotracheal (ET) route. The optimal ET dose of most drugs is still controversial, but typically 2 – 2½ times the recommended dose diluted in 5-10 ml of water or normal saline are given intratracheally.

Ventricular Fibrillation / Pulseless Electrical Activity- The most important interventions during the first minute of VF / pulseless VT are immediate CPR with minimal interruption in chest compressions and defibrillation as soon as possible. In cases of witnessed arrest with a defibrillator on site, after delivery of 2 rescue breaths, pulse is to be checked. If definite pulse is not felt within 10 sec, defibrillator pads should be applied and rhythm is to be checked. If the arrest is not witnessed, 5 cycles of CPR is to be given first and than attempt defibrillation. If VF / pulseless VT is present, 1 shock is to be delivered followed by CPR. If biphasic defibrillator is available, the dose at which that defibrillator has been shown to be effective for terminating VF (usually 120 –200 J) is to be given. If in doubt, use 200 J (biphasic), 360 J (monophasic) of energy for defibrillation. One shock is to be given rather than 3 stacked

shocks as recommended earlier. When a rhythm check reveals VF/VT, CPR is to be continued when defibrillator is being charged. Immediately after shock delivery, resume CPR without delay and continue for 5 cycles (or about 2 min if an advanced airway is in place), and then check rhythm. Once an advanced airway (e.g., ETT, Combitube, LMA) is placed, 2 rescuers no longer deliver cycles of compressions interrupted with pause for ventilation. Instead, the compressing rescuer should deliver 100 compressions per minute continuously; without pause for ventilation. The rescuer delivering ventilations should give 8-10 breaths per minute and should be careful to avoid delivering an excessive number of ventilations. While continuing with CPR, the cause of arrest – H's and T's (Hypovolemia, Hypoxia, Hydrogen ion disturbances, Hypo / Hyperkalamia, Hypoglycemia, Hypothermia, Toxins, Tamponade-cardiac, Tension pneumothorax, Thrombosis – coronary / pulmonary, Trauma) are to be looked for and managed accordingly. If VT/VF persists after delivery of 1 or 2 shocks and CPR as well, give vasopressor (epinephrine 1 mg every 3-5 min during cardiac arrest; one dose of vasopressin may replace first or second dose

of epinephrine). Do not interrupt CPR to give medications. The drug should be administered during CPR and as soon as possible after the rhythm is checked. After 5 cycles of CPR, analyze the rhythm again and be prepared to deliver another shock immediately if indicated. When VF/pulseless VT persists after 2 to 3 shocks plus CPR and administration of a vasopressor, consider administering an antiarrhythmic such as amiodarone (300 mg, bolus, once, then consider an additional 150 mg once again). If amiodarone is not available, lidocaine (1-1.5 mg/kg first dose, then 0.5-0.75 mg/kg, maximum 3 doses or 3 mg/kg) is the other alternative. Consider magnesium (1-2 gm iv) for Torsades de Poients associated with a long QT interval. If a nonshockable rhythm is present and the rhythm is organized (complexes appear regular or narrow), palpate for pulse. Rhythm check should be brief, and pulse checks should generally be performed only if an organized rhythm is observed. If a perfusing rhythm is transiently restored but not successfully maintained between repeated shocks (recurrent VF/VT), the patient may be a candidate for early treatment with antiarrhythmic medications

Asystole and Pulseless Electrical Activity - PEA encompasses a heterogeneous group of pulseless rhythms that includes pseudoelectromechanical dissociation, idioventricular rhythms, ventricular escape rhythms, post-defibrillation idioventricular rhythms,

and bradyasystolic rhythms. Research with cardiac ultrasonography and indwelling pressure catheters has confirmed that pulseless patients with electrical activity have associated mechanical contractions, but these contractions are too weak to produce a blood pressure detectable by palpation or noninvasive blood pressure monitoring. PEA is often caused by reversible conditions (H's and T's) and can be treated if those conditions are identified and corrected. The survival rate from cardiac arrest with asystole is dismal. As with PEA, the hope for resuscitation is to identify and treat a reversible cause. Patients who have either asystole or PEA do not require defibrillation attempts. The focus of resuscitation in such victims is to perform high-quality CPR with minimal interruptions in chest compressions and to identify and manage reversible causes or complicating factors. Minimize interruptions in chest compressions while inserting the airway or while establishing IV or IO access. If the rhythm check confirms asystole or PEA, resume CPR immediately. A vasopressor (epinephrine or vasopressin) may be administered at this time. Epinephrine is to be administered approximately every 3 to 5 minutes during cardiac arrest; one dose of vasopressin may be substituted for either the first or second epinephrine dose. For a patient in asystole or slow PEA, atropine (1mg iv/io to be repeated with a maximum total dose of 3 mg) is to be given. Do not interrupt CPR to deliver any

medication. Give the drug as soon as possible after the rhythm check. After drug delivery and approximately 5 cycles (or about 2 minutes) of CPR, recheck the rhythm. If a shockable rhythm is present, deliver a shock. If no rhythm is present or if there is no change in the rhythm, immediately resume CPR. Check pulse if an organized rhythm is present. If no pulse is present (or if there is any doubt about the presence of a pulse), continue CPR. If a pulse is present, identify the rhythm and treat appropriately. If the patient appears to have an organized rhythm with a good pulse, begin post-resuscitative care.

Management of Symptomatic Bradycardia and Tachycardia- The ECG and rhythm should be interpreted within the context of total patient assessment. Evaluate the patient's clinical symptoms and signs, including ventilation, oxygenation, heart rate, blood pressure, and level of consciousness, and look for signs of inadequate organ perfusion. The principles of arrhythmia recognition and management in adults are as follows:

- Bradycardia producing signs and symptoms (e.g. acute altered mental status, ongoing severe ischemic chest pain, congestive heart failure, hypotension, or other signs of shock) that persist despite adequate airway and breathing, pacing is to be considered. For symptomatic high-degree (second-degree or third-degree) atrioventricular (AV) block, provide transcutaneous pacing without delay.

- Tachycardic patient, who is unstable with severe signs and symptoms related to tachycardia, prepare for immediate cardioversion.
- Stable Tachycardic patient, determine if the patient has a narrow-complex or wide-complex tachycardia and then treat accordingly.

Bradycardia-Bradycardia is generally defined as a heart rate of <60 beats per minute. A slow heart rate may be physiologically normal for some patients and heart rates >60 beats per minute may be inadequate for others. So, clinically significant bradycardia (i.e. bradycardia that is inadequate for clinical condition) is to be managed with appropriate measures. Initial treatment of any patient with bradycardia should focus on support of airway and breathing. Give supplementary oxygen, monitor the patient, check blood pressure and oxyhemoglobin saturation, and establish IV access. Obtain an ECG to evaluate the rhythm. While initiating treatment, evaluate the clinical status of the patient and identify potential reversible causes and take steps to manage them. Look for signs and symptoms of poor perfusion (hypotension, acute altered mental status, chest pain, congestive heart failure, seizures, syncope, or other signs of shock related to the bradycardia) and relate if these signs are likely to be caused by the bradycardia. Signs and symptoms of bradycardia may be mild, and asymptomatic patients do not require treatment but should be monitored for any signs of deterioration.

Initiate transcutaneous pacing in patients who do not respond to atropine (or second-line drugs if these do not delay definitive management). Pacing is also required for severely symptomatic patients, especially when the block is at or below the His-Purkinje level (ie. type II second-degree or third-degree AV block). In the absence of reversible causes, atropine remains the first-line drug for acute symptomatic bradycardia transcutaneous pacing is usually indicated if the patient fails to respond to atropine, although second-line drug therapy with drugs such as dopamine or epinephrine may be successful. The recommended atropine dose for bradycardia is 0.5 mg IV every 3 to 5 minutes to a maximum total dose of 3 mg. Doses of atropine sulfate of <0.5 mg may paradoxically result in further slowing of the heart rate. Atropine administration should not delay implementation of external pacing for patients with poor perfusion.

Tachycardia-On the basis of QRS morphology, tachycardias can be classified as:

- Narrow–QRS-complex (SVT) tachycardias (QRS <0.12 second) (e.g. Sinus tachycardia, Atrial fibrillation, Atrial flutter, AV nodal reentry, Accessory pathway–mediated tachycardia, Atrial tachycardia, multifocal atrial tachycardia- MAT, Junctional tachycardia).
- Wide–QRS-complex tachycardias (QRS>0.12 second) (e.g. VT, SVT with aberrancy, Pre-excited tachycardias).

Patient is to be assessed while supporting the airway and breathing, administering oxygen, obtaining an ECG to identify the rhythm, and monitoring blood pressure and oxyhemoglobin saturation. Establish an IV access. Identify and treat reversible causes of the tachycardia. If signs and symptoms persist despite provision of supplementary oxygen and support of airway and ventilation, check if the patient is haemodynamically unstable and if signs of cardiovascular compromise are related to the tachycardia. If the patient demonstrates rate-related cardiovascular compromise, with signs and symptoms such as altered mental status, ongoing chest pain, hypotension, or other signs of shock, provide immediate synchronized cardioversion. Serious signs and symptoms are uncommon if the ventricular rate is <150 beats per minute in patients with a healthy heart. Patients with impaired cardiac function or significant comorbid conditions may become symptomatic at lower heart rates. If the patient is unstable with narrow-complex reentry SVT, adenosine is to be given, while preparations are made for synchronized cardioversion, but do not delay cardioversion to administer the drug or to establish IV access. Synchronized cardioversion is shock delivery that is timed (synchronized) with the QRS complex. This synchronization avoids shock delivery during the relative refractory period of the cardiac cycle ("vulnerable period"), when a shock could produce VF. Synchronized

cardioversion is recommended to treat (1) unstable SVT due to reentry, (2) unstable atrial fibrillation, and (3) unstable atrial flutter.

Regular Narrow-Complex Tachycardia

- **Sinus Tachycardia-**Sinus tachycardia usually results from a physiologic stimulus (fever, anaemia, or shock). Sinus tachycardia occurs when the sinus node discharge rate is >100 times per minute in response to a variety of stimuli or sympathomimetic agents. No specific drug treatment is required. Therapy is directed toward identification and treatment of the underlying cause. When cardiac function is poor, cardiac output can be dependent on a rapid heart rate. In such compensatory tachycardias, stroke volume is limited, so "normalizing" the heart rate can be detrimental.

- **Supraventricular Tachycardia -** Reentry SVT is a regular tachycardia that is caused by reentry, an abnormal rhythm circuit that allows a wave of depolarization to travel in a circle. The rate of reentry SVT exceeds the typical upper limits of sinus tachycardia at rest (>120 beats per minute) with or without discernible P waves. The rhythm is considered to be of supraventricular origin if the QRS complex is narrow (<120 milliseconds or <0.12 second) or if the QRS complex is wide (broad) and bundle branch aberrancy is known to be present. Reentry SVT may include AV nodal reentrant tachycardia or AV reentry tachycardia. Abrupt onset and termination of this tachyarrhythmia led to its original name, paroxysmal supraventricular tachycardia (PSVT).Vagal maneuvers and adenosine are the preferred initial therapeutic choices for the termination of stable reentry SVT. If reentry SVT does not respond to vagal maneuvers, give 6 mg of IV adenosine as a rapid IV push. Give adenosine rapidly over 1 to 3 seconds through a large vein followed by a 20-mL saline flush and elevation of the arm. If the rate does not convert within 1 to 2 minutes, give a 12-mg bolus. Give a second 12-mg bolus if the rate fails to convert within 1 to 2 minutes after the first 12-mg bolus. Adenosine is safe and effective in pregnancy. Adenosine, however, does have several important drug interactions. Larger doses may be required for patients with a significant blood level of theophylline, caffeine, or theobromine. The initial dose should be reduced to 3 mg in patients taking dipyridamole or carbamazepine, those with transplanted hearts, or if given by central venous access. Side effects with adenosine are common but transient; flushing, dyspnea, and chest pain are the most frequently observed. If the rhythm does convert, it was probably reentry SVT. Monitor the patient for recurrence and treat any recurrence with adenosine or control the rate with a longer-acting AV nodal blocking agent (e.g. diltiazem or ß-blocker). If adenosine fails to convert reentry SVT, attempt rate control with a nondihydropyridine calcium channel blocker (i.e., verapamil or diltiazem) or ß-blocker as a second-line agent. These drugs act primarily on nodal tissue either to slow the ventricular response to atrial arrhythmias by blocking conduction through the AV node or to terminate the reentry SVT that depends on conduction through the AV node.

- Verapamil and, to a lesser extent, diltiazem may decrease myocardial contractility and critically reduce cardiac output in patients with severe left ventricular dysfunction. Calcium channel blockers that affect the AV node (including verapamil and diltiazem) are considered harmful when given to patients with atrial fibrillation or atrial flutter associated with known pre-excitation (Wolff-Parkinson-White) syndrome. ß-Blockers should be used with caution in patients with pulmonary disease or congestive heart failure. Verapamil (2.5 to 5 mg IV

bolus over 2 minutes, if no therapeutic response and no drug-induced adverse event, repeat doses of 5 to 10 mg administered every 15 to 30 minutes to a total dose of 20 mg or an alternative dosing regimen is to give a 5-mg bolus every 15 minutes to a total dose of 30 mg) should be given only to patients with narrow-complex reentry SVT or arrhythmias known with certainty to be of supraventricular origin. It should not be given to patients with impaired ventricular function or heart failure. Diltiazem is to be given in a dose of 15 to 20 mg (0.25 mg/kg) IV over 2 minutes; if needed, in 15 minutes give an IV dose of 20 to 25 mg (0.35 mg/kg). The maintenance infusion dose is 5 to 15 mg/hr, titrated to heart rate.

- **Wide Complex Tachycardia-**Determine if the patient's condition is stable or unstable. An unstable patient with wide-complex tachycardia is presumed to have VT, and immediate cardioversion is performed. If the patient is stable, get a 12-lead ECG to evaluate the QRS duration (i.e., narrow or wide). If the patient becomes unstable at any time, proceed with synchronized cardioversion. If the patient develops pulseless arrest or is unstable with polymorphic VT, treat as VF and deliver high-energy unsynchronized shocks (i.e., defibrillation

doses). Check if the rhythm is regular or irregular. A regular wide-complex tachycardia is likely to be VT or SVT with aberrancy. An irregular wide-complex tachycardia may be atrial fibrillation with aberrancy, pre-excited atrial fibrillation, or polymorphic VT. Polymorphic VT may represent torsades de pointes. Adenosine (6 mg rapid IV push followed by a 12-mg bolus and a second 12-mg bolus if the rate fails to convert) is recommended if the wide-complex regular tachycardia is thought to be SVT. Treatment of mono-morphic (regular) wide-complex tachycardia, particularly if the patient is symptomatic (e.g. signs of altered level of consciousness) is synchronized cardioversion. If the rhythm is VT in a stable patient, IV antiarrhythmic drugs (amiodarone 150mg IV over 10 minutes; to be repeated as needed to a maximum dose of 2.2 g IV per 24 hours) may be effective. Alternative drugs for wide-complex regular tachycardias are procainamide and sotalol.

Irregular Tachycardias

- **Atrial Fibrillation and Flutter-**Atrial fibrillation with an uncontrolled ventricular response mani-fests as an irregular narrow-complex or wide-complex tachycardia. Other diagnostic possibilities include MAT. Management involves

the control of the rapid ventricular rate (rate control) and conversion of hemodynamically unstable atrial fibrillation to sinus rhythm (rhythm control). Patients with atrial fibrillation for >48 hours are at increased risk for cardioembolic events and must first undergo anticoagulation before rhythm control. Electric or pharmacologic cardioversion (conversion to normal sinus rhythm) should not be attempted in these patients unless the patient is unstable or the absence of a left atrial thrombus is documented by transeso-phageal echoca-rdiography.

- Magnesium diltiazem and ß-blocker are effective for rate control in the treatment of atrial fibrillation with a rapid ventricular response. Ibutilide and amiodarone have been shown to be effective for rhythm control in the treatment of atrial fibrillation. AV nodal blocking agents such as adenosine, calcium channel blockers, digoxin, and possibly ß-blockers are avoided in patients with pre-excitation atrial fibrillation or atrial flutter because these drugs can cause a paradoxical increase in the ventricular response to the rapid atrial impulses of atrial fibrillation.

- **Polymorphic VT-**Polym-orphic (irregular) VT may deteriorate to pulseless arrest thus necessitating early

intervention. Pharmacologic treatment of recurrent polymorphic VT is determined by the presence or absence of a long QT during the sinus rhythm. If a long QT interval is observed during the sinus rhythm (i.e. the VT is torsades de pointes), the first step is to stop medications known to prolong the QT interval. Correct electrolyte imbalance and other acute precipitants. Magnesium is commonly used to treat torsades de pointes VT (polymorphic VT associated with long QT interval). If the patient with polymorphic VT is or becomes unstable (i.e., demonstrates altered level of consciousness, hypotension, or other signs of shock, such as severe pulmonary edema), provide high-energy (i.e., defibrillation dose) unsynchronized shocks. If there is any doubt whether monomorphic or polym-orphic VT is present in the unstable patient, do not delay shock delivery for detailed rhythm analysis—provide high-energy unsynchronized shocks (i.e., defibrillation doses). After shock delivery start CPR immediately (beginning with chest compressions).

To summarize, early and timely CPR with BLS, accompanied with defibrillation and appropriate drug therapy gives good outcome in collapsed victims.

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Claude Beck, defibrillation and CPR

Defibrillator, 1947. Prototype model, for use in cases of open-chest defibrillation encountered inthe operating room at Lakeside Hospital, University Hospitals of Cleveland. Heart attack, or cardiac arrest, became a leading cause of death after the turn of the century. People had always suffered from cardiac problems, but they usually died from other causes, especially infectious diseases, long before reaching the age when heart problems threatened their well being. As medicine advanced and people lived longer, heart disease became a serious health issue. Claude Beck (1894-1971) pioneered heart surgery, especially operations to improve circulation in damaged heart muscles. He also devised ways to revive heart attack victims, including the defibrillator and CPR (cardio-pulmonary resusci-tation). Beck had trained as a neurosurgeon at Harvard and Johns Hopkins before coming to University Hospitals of Cleveland in 1924 as resident and Crile Research Fellow in Surgery. He soon turned to cardiovascular research and surgery, and devoted the remainder of his career to that field.

Preterm Labor-Prediction & Prevention

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Onset of labor in patients with intact membranes before 37 weeks. The lower limit of this definition is not clearly defined. It is 20wk in the US, 22 wk by FIGO & 24wk in UK. 5-10% of all deliveries in the world 10-15% in India. Inspite of considerable advances in obseterical care, PTL continues to be a major cause of perinatal mortality & morbidity. 50%-70% of perinatal deaths occur in preterm infants. Even in the developed countries, incidence of PTL has not decreased much over the years. As the other causes of PMR are decreasing with improved care, focus of research is now on the prevention of PTL in the last 2 decades.With improved neonatal care, even if the preterm baby survives, the physical & mental handicap can be considerable.

Despite all advances in neonatology, the most efficient system for decreasing perinatal mortality & morbidity is to keep the fetus inside the uterus until it reaches at least 1500g wt or 32week of gestation.

Complications- Increased Perinatal mortality; Perinatal morbidity – Necrotising enterocolitis, Intravetricular Hemorrhage; Long term sequelae- developmental delay, leukemoid reaction, bronchopulmonary dysplasia, cerebral palsy.

Etiology- Idiopathic: In 60-70% cases, there is no identifiable cause. It is called spontaneous PTL. It is associated with premature effacement of cervix with hyperirritable uterus & early engagement of head. Indicated PTD: 20-30%cases. In various medical & surgical complications in mother or fetus that create an unfavorable intrauterine enviroment for the fetus or is dangerous to the mother- severe preeclampsia, chronic HTN, DM, placenta previa, abruption. PPROM. In majority of the cases, multificatorial origin in found.

Prediction & Prevention- Requires identification of pregnancies at risk & effective interventions to prevent or reduce the morbidity of PTB

Prediction of PTL

Warning Signals- Menstrual like cramps;Low dull backache; Abdominal cramps; Feeling of pelvic pressure or heaviness in vagina; Increase/change in vaginal discharge; Leaking PV; Uterine contractions < 10min apart, even if painless.

Risk storing system- Papiernlik (1974) evolved an elaborate scoring system incorporating history, SES & lifestyle for detection of patients at high risk for spontaneous PTL. It was modified by Creasy et al. It was not found useful & has been abandoned as insensitive.

Prior PTDs- Strongly correlates with subsequent PTL. Risk of recurrent PTD is increased 3 fold. Although women with h/o PTD are clearly at risk of recurrence, they contribute to only 10% of total PTDs.

Table-1, Approximate neonatal survival of preterm infants born in the tertiary care centre

Gestation period	Weight	Survival
24-26week	500-750g	50%
26-28 week	750-1000g	75%
28-30 week	1000-1400g	90%
30-32 week	1400-1800g	96%
32-34 week	1800-2200g	99%
>34 week	2200g	100%

Table-2, Birth Outcome

Birth Outcome	II birth < 34week
I birth >35week	5%
I birth<34week	16%
I & II birth <34week	41%

Uterine contraction assessment-Antepartum uterine contractility-Approximately 2 weeks before the onset of active labour, contraction frequency & intensity both increase referred to as "prelabour synchronization" of uterine activity.

Routine PV Examination-Assess cervix at each AN visit has been advocated by some. Position, length, consistency of cervix & formation of lower US are checked. These may be advised in high risk patients only. All patients may not prefer it & it itself may be a risk factor for PTL. Although women with dilation & effacement in the III trimester are at risk for PTL, it has not been established that detection improves pregnancy outcome.Manual assessment of cervix is also nonspecific, subjective & nonreproducible & is also not accurate for evaluating the internal Os.

Transvaginal Ultrasongraphy (TVU)-Superior to manual assessment of cervix & prediction of PTB in women with PTL; Detects shortening of cervical canal before it becomes evident, with manual examination; More reproducible; Safety & acceptability confirmed by studies;TVU has been studied extensively as a predictor of PTB in patients with symptoms of PTL;TVU CL is measured at a mean gestational between 22-24 weeks;Not only do women and shortest cervical length have the greatest risk of PTB, those with CL at 50th percentiles have more risk than do women those CL is >75th percentile;Cut off: 20m taken(has the best PPV).TVU CL of 18mm is an optimal positive predictor value while 30mm is a negative predictor value;Although TVU of CL has been useful in redefining the contribution of cervical factors to the pathogenesis of early PTB, there is no evidence that interventions such as maternal rest or cerclage based on a finding of a short cervix can be translated into a reduced rate of PTB; It might help in avoiding hospitalization and more intensive therapies in women at a low risk of PTL.

T.V.U. Processes-Before the onset of dilatation and effacement, the fetal membranes in the endocervical have a perpendicular T-shaped relationship. As the cervix begins to efface, the internal os becomes disrupted, and the fetal membranes prolapse into the endocervical canal producing a funnel or Y shaped cervix on TVU. There is no change noted at the level of external os with direct vaginal speculum examination or with digital examination.As the membranes continue to descend into the endocervical canal and the closed CL shortens, V shape will be noted.The characteristic U shaped membrane prolapsed into the endocervical canal is associated with a markedly reduced CL & is associated with findings at the level of external os including visible membranes on examination & significant dilatation or effacement noted on digital examination.

Genital Colonization with Microorganisms - The association of maternal colonization with various genital microorganisms and risk of PTB,LBW, Leukemoid reaction, Bronchopulmonary dysplasia has led to investigations of screening tests to identify and treat women at risk.Infection could be intrauterine (overt or subclinical) or lower genital tract colonization. Studies demonstrated that either histologic chorioamniomits or the recovery of bacteria by culture of placenta and fetal membranes was associated with PTB but the combination was more common.

ESTRIOL-An exponential rise in estriol levels has been found after 34 weeks.This surge has been observed to occur 2-4 weeks before the onset of term labour.Measurements of estriol obtained from maternal saliva samples represented biologically active maternal serum values and have been used as a practical means for collecting samples. Administration of corticosteroids suppress estriol levels thereby potentially limiting the clinical use of this assay. Optimal cutoff – 2.3mg/ml. Sensitivity – 71% PPV – 23%.Specificity – 77%.After the initial test, 23%

with +ve test delivered with in week; 54% - in 2 week;85% - 3 week. After a II test, 70% in 1 week;90% in 2 weeks;100% in 3 weeks.Thus it is a better predicator of late (>34weeks) PTL

Other biochemical markers- Corticotropin – releasing hormones; BHCG & AFP; Activin;Inhibin Relaxin; IL-6,8;TNF,CRP;Collagenase, Granulocyte elastase; Matrix metalloprotienases; G-CSF. The Preterm Prediction study demonstrated that if all markers (previous SPTB, CL< 25mm & FFN) are +ve, there is 50% risk of delivery before 32 weeks.- Multiple marker test.

implantation & maintaining the integrity of the choriodecidual interface.The Cervicovaginal secretions contain FF early in gestation and then again just before term labour.The concentrations are normally low in the II & early III trimester. Hence these characteristics make FF a logical marker for threatened or actual PTL.Preclinical onset of PTL is associated with disruption of choriodecidual junction – release of FF which is detected in the cervicovaginal mucous. Quantization of FF can be performed & its presence or absence can then aid in the diagnosis and therapy of patient.50ng/ml is the cut off

with only cervical change): the likelihood ratio for +ve result is 4.01 before 34 wk & -ve result is 0.78.Thus it helps in decision making regarding admission, in utero transfer & administration of steroids. Though FFN is predictive, alterations in the subsequent management of a positive test need to be demonstrated to improve outcome.

Future Trends-Prediction- Development of multiple marker tests for SPTB & the use of molecular biology techniques (genomics & proteomics) may be the predictive methods of the future.Future research should focus on less invasive markers for

Table-3, Prediction at 22-24 weeks of SPTB before 35 Weeks

Tests	Sensitivity	Specificity	Positive predictive value	Negative predictive value
Multiple PTL symptoms	50%	63.5%	21.4%	86.4%
Uterine contractions>4hr	6.7%	92.3%	25%	84.7%
Bishop score>4	32%	91%	42.1%	87.4%
Cervical length <25mm	40.8%	89.5%	42.6%	88.8%
Fibronectin >50ng/ml	18%	95.3%	42.9%	85.6%

Biochemical markers for the Prediction of PTL -As of early 2005, only 2 tests have received approval by the FDA as markers for PTL. Fetal fibronectin –FF (Adeza Biomedical, Sunnyvale, California); Salivary estriol (Salest, Biex, Dublin, California)

Fetal Fibronectin(FF)- Stable glycoprotein. It is found in the choriodecidual junction and has role in establishing blastocyst

for a positive result.Specimen has to be collected before examining the cervix and after 24hr since last PV or intercourse.The greatest clinical value in excluding women with false labour from the costs & morbidity of aggressive treatment of PTL.A negative test reassures that delivery is unlikely in the next 7-14 days.Risk determined in asymptomatic women (h/o PTD or presenting

high risk population screening & predicting PTB.

Prevention of PTB-Based on identifying and correcting a potential "cause" of PTB with the expectation that the rate of PTB would decline.Intervention trials have addressed early identification of PTL through-Patient education;Pharmacologic suppression of uterine contractions; Antimicrobial therapy of

vaginal microorg-anisms; Cerclage sutures to bolster the cervix; reduction of maternal stress; improved access to PNC; reduced physical activity.

Education of at risk women about the signs & symptoms of PTL, life style changes & behavioral modifications. Initial studies described reductions in SPTB, but subsequent reports showed inconsistent results.

Antibiotics for prevention of PTB-Treatment of abnormal vaginal flora.Mixed literature describing success & failure. Results of trials indicate that infection doesn't operate independently as cause of PTB.

Recommendations for clinical practice-

- Bacteriuria-Screen & treat at first AN visit. Reduces PTB by 50%
- Chlamydia trachomatis-screen all patients using DNA amplification techniques and cervical/urine specimens;Doesn't seem to prevent PTB; Reduces vertical transmission & spread of STDs
- Neisseria gonorrhoea-Screen at risk women at first prenatal visit & treat to reduce vertical transmission
- Trichomoniasis- No need to screen asymptomatic women; symptomatic patients may be treated safely with oral metro.
- BV- Screen and PTB oral antibiotics x 7days in early pregnancy.
- GBS-Do not treat positive rectovaginal cultures in the antepartum periods. Follow the CDC guidelines

regarding intrapartum antibiotics.

*The Cochrane review concluded that treatment of Bacterial vaginosis is useful in decreasing the risk of PPROM & LBW in women with H/o previous PTB & not in asymptomatic women with BV. But 2 independent RCTs support the identification & treatment of bacterial vaginosis in low risk women at an earlier gestational age.

*The choice of antibiotic is controversial. Recent evidence suggests that Metronidazole may increase PTD in women selected by history & + FFN. Thus, treatment with Clindamycin seems prudent.

*Insertion of a vaginal pessary may be a cost effective preventive method in at risk patients but prospective controlled trials are needed.

Social support & Improved access to ANC-ANC in correlated with the rate of SPTB but the reason is not clear.Prospective intervention designed to reduce PTB through better access to care, reduced stress & the like have been unsuccessful.

Prophylactic cerclage for women with h/o PTB -Urgent cerclage is usually placed between 16-24 weeks gestation with most patients asymptomatic.The diagnosis is made by TVU with dilatation of the internal os, prolapse of the fetal membranes, a short CL, or by digital examination with demonstrable cervical dilatation & effacement.Low success rates but still employed due to the otherwise

poor prognosis due to visible membranes.McDonald cerclage is undertaken usually.4 RCTs have examined the efficacy of urgent cerclage .Concluded that no benefit is achieved by the procedure in terms of PTD, perinatal mortality & morbidity. The group of patients who benefit from urgent cerclage are yet to be defined.It is still considered as a procedure under investigation.An RCOG trial found decreased incidence of PTB in the cerclage group but didn't result in obvious benefit for neonates. It was found that 25 women were treated to prevent one PTB. Found beneficial in women with 3 or more II trimester losses.Thus, routine determination of CL in low risk women followed by cerclage in those with a shortened cervix cannot be advocated. The procedure might help individuals at risk based on their history.Studies show that measuring IL-8 concentration in cervical mucous may allow clinicians to better identify women who would most benefit from cerclage to prevent PTB.

Prophylactic Medication-Tocolytics have been widely used as prophylaxis but this has not been associated with either a reduction in PTB or LBW deliveries.

Parenteral PGN (17a hydroxy progesterone caproate) - Advocated for the last 50yrs for prophylaxis against PTL because of its role in maintaining pregnancy. A recent Cochrane meta-analysis assessing the benefits & harms of PGN administration showed that IM

PGN decreases the risk of SPTB before 37 weeks & infant birth weight < 2500g.No improvement in perinatal outcome.But limited evidence regarding benefits & harm (long term outcome). Hence to be used with caution.Existing evidence(double blind RCT) supports its use in high risk asymptomatic women from early in II trimester weekly until 36 weeks but not as tocolytic in symptomatic women. Based on this data , 5-6 women with a previous SPTB would need to be treated to prevent 1 PTB before 37wk & 12 women to prevent PTB before 32 weeks. Also infants of women treated had lower rates of secondary outcomes like NEC,IVH & need for supplemental oxygenation. No reported increase in congenital anomalies or adverse effects.Flaws of this studywere-Unusually high incidence of PTD in the control group; Not known if results can be applied to other high risk groups(multiple gestation); 17-a hydroxy-progesterone caproate not readily available; High risk of PTD in the treated group; Requires weekly IM regimen. A smaller study demonstrated that administration of **vaginal PGN** was also associated with reduced risk of PTD .Results encouraging because no need of systemic administration.Studies still needed to determine the best mode of administration, optimal dose, side effects, long term safety, gestation at which to commence therapy & duration of treatment.

CO_2 Inhibitors - (Rofecoxib) Didn't reduce the incidence of early PTB. Associated with adverse effects on fetal renal function & ductus arteriosis. Hence, not advocated.

Bed rest/Activity modific-ation-There is no evidence from Moberly conducted trials that bed rest or decreased activity offers any benefit for pregnant women. (Obstt. & Gynaecol. 1994).

Conclusion-Symptoms of early PTL may be subtle & nonspecific. It is important to be "liberal in looking for PTL, but conservative in the diagnosis". The goal of first contact with the patient should be sensitivity , while the goal of evaluation should be specificity.Prediction of early birth has become more precise with the advent of markers such as FFN & techniques such as TVU of CL. However, interventions to prevent delivery & more importantly to improve outcome, remain poor. An USG machine has been present in the labor room & delivery suites for decades for the assessment of the fetus. It is time now to add a dedicated vaginal probe to this machine, for assessment of patients at risk for PTL.Large trials are needed to test promising agents , but must be appropriately designed with meaningful endpoints such as perinatal mortality & morbidity rather than gestational age.

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National Board of Examinations offers following gold medal to the meritorious DNB candidates

Anaesthesia for Laparoscopic and other Endoscopic Surgery

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Minimally Invasive Surgery or Minimal Access Surgery is becoming more popular in modern era of surgical practice. Extensive endoscopic procedures are now being performed in all patients of all ages and even in those patients with various comorbid conditions. The development of minimally invasive surgery has revolutionized surgery and this process has influenced the practice of anaesthesiology also. Though endoscopic procedures facilitate faster recovery but it also poses various intraoperative challenges to both surgeons as well as anaesthesiologists. In addition these procedures are associated with potential life threatening complications that are usually not encountered with the traditional open procedure. The duration of some procedures, the risk of vascular or visceral injury and difficulty in evaluating blood loss add to the anaesthetic challenge. Thus, there is a need to modify the anesthetic technique to allow these novel surgical procedures to be performed safely. Laparoscopic techniques have been applied not only in gastrointestinal (gastric, colonic, splenic, hepatic), but also in gynaecologic (e.g. hysterectomy), urologic (e.g. nephrectomy), thoracic (VATS) surgery.

Anaesthetic concerns in various laparoscopic surgeries can be enumerated as-

- Physiological changes occurring during laparoscopy (Table -1)
- Changes affected by patient's positioning
- Effects produced by organ handling and complications occurring during surgery
- Effect produced by associated co-morbid conditions

The cardiopulmonary changes occurring during laparoscopy are complex and depend on the interaction of the patient's preexisting cardiopulmonary status, the anaesthetic technique used (ventilatory technique and anaesthetic agents used), and several surgical factors including intra-abdominal pressure (IAP), carbon dioxide (CO_2) absorption, patient position and duration of the surgical procedure along with neurohumoral responses. Laparoscopic surgery requires the creation of a pneumoperitoneum. CO_2 is used for gas insufflation via a Veress needle, at a rate of 1-6 liters/min, to a pressure of 14-20 mmHg. Constant CO_2 flow of about 200-400 ml/min maintains pneumoperitoneum perioperatively, thus creating the "tension pneumoperitoneum".

Table- 1, Physiological changes during laparoscopy Cardiovascular changes

Variables	Hemodynamic effect due to Pneumoperitoneum
Cardiac output	Variable (depends on gas used / position of patient)
Cardiac Filling	Venous return – reducedLeft ventricular end diastolic volume – reducedIntrathoracic pressure – increasedRight atrial and pulmonary artery occlusion pressure -increased
Heart Rate	Unchanged / minimally increased
Arterial Pressure	Increased
Systemic and Pulmonary Vascular Resistance	Increased

Pneumoperitoneum causes an increase in systemic vascular resistance (SVR), mean arterial pressure, and cardiac filling pressures. The increase in SVR is due to increased sympathetic output from CO_2 absorption and a neuroendocrine response to pneumoperitoneum. The increase in SVR increases the myocardial wall tension and thus, increases the myocardial oxygen demand. Compression of the abdominal organs (e.g., liver and spleen) caused by increased IAP and increased sympathetic output, may be responsible as one of the causes of increased cardiac filling pressures. In addition, the cardiac filling pressures may reflect the increased intrathoracic pressures caused by pneumoperitoneum. After an initial increase, there is a decrease in venous return due to creation of pneumoperitoneum. These changes become more marked as the intraabdominal pressure increases. Higher insufflation pressures (>25cm H_2O or 18 mm Hg), however tend to collapse the major abdominal veins (particularly, the inferior vena cava), which decreases venous return and leads to a drop in preload and cardiac output in some patients. Gas insufflation may be associated with various arrhythmias including AV dissociation, nodal rhythm, sinus bradycardia and asystyole. Responses are more pronounced with rapid stretching of peritoneum at the beginning of insufflation probably because of vagus mediated reflex. Incidences of these complications are more pronounced when associated with hypercarbia and use of halothane. Hypercarbia, if allowed to develop, will stimulate the sympathetic nervous system and thus increases blood pressure, heart rate, and the risk of arrhythmias. Hypercapnia may cause a decrease in myocardial contractility and lower arrhythmia threshold. The anticipated direct vascular effect of hypercapnia, producing arteriolar dilation and decreased SVR, is modulated by mechanical and neurohumoral responses including catecholamine release. Attempting to compensate by increasing the tidal volume or respiratory rate will increase the mean intrathoracic pressure further hindering venous return and increasing pulmonary artery pressures. These effects can prove particularly challenging in patients with restrictive lung disease, impaired cardiac function, or intravascular volume depletion.

Pulmonary changes- The respiratory system can be affected by the following mechanisms:

- Effects of pneumoperitonium i.e. insufflation of the peritoneum by CO_2.
- Effects of CO_2 absorption.
- Diaphragmatic movement impairment.
- Effects of Trendelenberg or reverse - Trendelenberg position needed during the procedure.

The increased IAP leads to cephalad displacement of diaphragm and decreases functional residual capacity and total lung compliance, resulting in basal atelectasis and increased airway pressures. The increase in minute ventilation required to maintain normocarbia further increases peak airway pressures. These pulmonary changes are compounded with those caused by changes in position (decreased pulmonary compliance (by 30 – 50%), increased airway resistance and increase in pulmonary resistance, resulting in increase in airway pressure for any given tidal volume). Furthermore, these changes may be exaggerated in obese patients and in those with respiratory dysfunction and have increased chances of barotraumas during IPPV. Partial pressure of CO_2 reaches plateau in 15 – 30 minutes after beginning of CO_2 insufflation in patients with controlled mechanical ventilation. Any significant increase in $PaCO_2$ after this period of plateau requires a search for a cause independent of or related to CO_2 insufflation such CO_2 emphysema. During CO_2 pneumoperitoneum, the increase of $PaCO_2$ may be multifactorial: absorption of CO_2 from the peritoneal cavity, impairment of pulmonary ventilation and perfusion by mechanical factors such as abdominal distension, patient position and volume controlled mechanical ventilation, and depression of ventilation by premedicant and anaesthetic agents in the case of spontaneous breathing. The observation of an increase in $PaCO_2$, but not nitrous oxide or helium, was used as the insufflating gas suggests that the main mechanism of the increased $PaCO_2$ during CO_2 pneumoperitoneum is absorption of

CO_2 rather than the mechanical ventilation reperc-ussions of increased IAP. The absorption of a gas from the peritoneal cavity depends on its diffusibilty, the absorption area, and the vascularity of the walls of that cavity. CO_2 absorption is greater during extraperitoneal (pelvic) insufflation than during intraperitoneal insufflation. CO_2 reaches a plateau after initiation of intraperitonael insufflation but continues to increase slowly throughout extraperitoneal insulation. A reduction in FRC relative to closing volume may be associated with the development of intraoperative atelectasis and intrapulmonary shunting. These changes may occur during general anesthesia because of a variety of factors-

- Cephalad shift of the diaphragm associated with supine position

- Loss of inspiratory muscle tone

- Appearance of end-expiratory muscle tone in the abdominal expiratory muscles

- Changes in intrathoracic blood volume associated with induction of anesthesia, and

- Influence of muscle relaxa-nts on diaphragmatic excursion.

The reduction in FRC associated with general anesthesia may be compounded by the CO_2-induced pneumoperitoneum during laparoscopic cholecys-tectomy. A reduced cardiac output secondary to reduction in venous return or drug-induced myocardial depression may reduce mixed-venous O_2 tension. Hypoxemia may be present when these changes are accompanied with reduction in the cardiac output. Restriction in diaphra-gmatic mobility promotes uneven distribution of ventilation to the non-dependent part of the lung. This results in ventilation perfusion mismatch with hypoxaemia and hypercarbia (CO_2 absorption form pneum-operitoneum may also cause hypercarbia).

Gastrointestinal effects - Trochar insertion can damage viscera, particularly distended stomach, probably caused by manual ventilation during intubation. Therefore nasogastric aspiration should always be done prior to trochar insertion. Increased incidence of nausea and vomiting has been associated with the laparoscopic surgery, so regular antiemetic drugs may be considered. Though increased IAP may be considered to increase the chances of regurgitation but it also increases the barrier pressure thus preventing chances of regurg-itation.

Effects on other systems- Pneumoperitoneum, changes in patient position, reductions in cardiac output, and systemic CO_2 absorption influence splanchnic, renal, and cerebral blood flow during minimal access procedures. Numerous regional circulatory changes also occur during laparoscopy including increased cerebral blood flow and intracranial pressure, decreased total hepatic blood flow, reduced bowel circulation resulting in decreased gastric intramucosal pH (suggesting reduced gut perfusion), reduction in renal blood flow and urine output (because of increase in renal vascular resistance, reduction in glomerular filtration gradient and decrease in cardiac output), and decreased femoral vein blood flow which may increase the risk of deep vein thrombosis. Massive elevation in IAP produces lactic acidosis, probably by severely lowering cardiac output and by impairing hepatic clearance of blood lactate. Of importance, the changes in regional circulation may have clinical implications in critically ill patients undergoing extensive laparoscopic procedures requ-iring prolonged pneumop-eritoneum. As with all surgery, there is an associated stress response with elevated cortisol and circulating catecholamines.

Neurohumoral response- Potential mediators of the increased SVR observed during pneumoperitoneum include vasopressin and catecholamines. Hypercapnia and pneumop-eritoneum are likely to cause stimulation of the sympathetic nervous system and catecho-lamine release.

Alternatives to CO_2 pneum-operitoneum - The ideal insufflating gas for pneumope-ritoneum should be nontoxic, colourless, readily soluble in blood, easily ventilated through lungs, nonflammable and inexpensive. Various gases that have been used in laparoscopic procedures are air, O_2, N_2O, CO_2, argon, and helium. CO_2 is the insufflation gas of choice for

laparoscopic surgery for various reasons, unlike nitrous oxide; it does not support combustion and therefore can be used safely with diathermy. Compared to helium, the high blood solubility of CO_2 and its capability for pulmonary excretion reduces the risk of adverse outcome in the event of gas embolism. CO_2 insufflation into the peritoneal cavity increases arterial CO_2 tension which can be managed by increasing minute ventilation. Insufflation of inert gases like helium, argon avoids the increase in $PaCO_2$. Consequently, hyperventilation is not required, though ventilatory consequences of increased IAP persist. The hemodynamic responses with pneumoperitoneum using inert gases are almost similar to those of CO_2. The low blood gas solubility of the inert gases raise the issue of safety in the event of gas embolism.

Gasless Laparoscopy-The peritoneal cavity is expanded using abdominal wall lift obtained with fan retractors. This technique avoids the haemodynamic and respiratory repercussions of increased IAP and the consequences of the use of CO_2. However, it compromises surgical exposure and increases technical difficulties. Combining abdo-minal wall lifting with low pressure CO_2 pneumop-eritoneum (5mmHg) may improve surgical conditions without having untoward consequences of increased IAP.

Technique of Pneumope-ritoneum - Technique for pneumoperitoneum creation include insufflation of gas after insertion of the Veress needle infra-umbilicus or open laparo-scopy involving dissection through the linea alba and opening of the peritoneum under direct vision. The Veress needle consists of a blunt tipped, spring-loaded inner stylet and sharp outer needle that penetrates the tissue layers of the abdominal wall. The Veress needle is inserted at an angle of 45 – 90 degrees facing downwards depending upon the operator's preference, patient's habitus, and previous abdominal surgery. The aortic bifurcation lies directly beneath the umbilicus, especially if Trendelenberg position has already been established. Open insertion of the trocar technique has been described, thereby guaranteeing pneumoperitoneum and avoiding dangers of blind trocar insertion. Modern laparoscopic insufflators autom-atically terminate gas flow when a preset intra-abdominal pressure (10-15 mm Hg) is reached. An access port is then inserted in place of the needle to maintain insufflation during surgery.

Patient positioning-Patient positioning depends on the site of the surgery and the type of surgical approach. Various positions according to the type of approach are:

Extraperitoneal - Lateral appr-oach – complete flank position (Kidney position); Posterior approach – prone position; Pelvic surgery – supine with Trende-lenberg position.

Transperitoneal - Anterior approach - supine with Trendel-enberg position; Lateral approach – complete flank position (Kidney position); Pelvic surgery – supine with Trendelenberg position (head down)

Head down tilt is used for pelvic and inframesocolic surgery, the head up position is preferred for supramesocolic surgery. Patients are often placed in the Trendelenberg position for laparoscopic gynaecologic procedures while laparoscopic cholecystectomy usually change to steep reverse Trendelenberg, with left lateral tilt to facilitate retraction of the gallbladder fundus and to minimize the diaphragmatic dysfunction associated with the induced pneumoperitoneum . The patient is often placed in Lithotomy position in various urologic and gynaecologic procedures. Head down position (Trendelenberg) is commonly requested during insertion of Verres needle and cannula. Patient tilt should be reduced as much as possible and should not exceed 15 – 20 degrees. Tilting must be slow and progressive to avoid sudden haemodynamic and respiratory changes. With Trendelenberg position and pneumoperitoneum, cardiac output fall by 60% and there are no changes in heart rate. Though preload is increased mean arterial pressure remains unchanged or decreases. Moderate fall in stroke volume occurs. Stroke index and cardiac index fall by 42%. Total peripheral resistance increased by 100% in balanced anaesthesia from 1620 to 2491 dynes. cm$^{-5.}$M^{-2}. These seemingly paradoxical responses may be explained by carotid and aortic baroreceptor –

mediated reflexes. The reverse Trendelenberg position decreases preload, cardiac output, and mean arterial pressures. Head down, or Lithotomy, for lower abdominal procedures causes a cephalad shift in abdominal viscera and the diaphragm. FRC, total lung volume, and pulmonary compliance will be decreased. Although these changes are usually well tolerated by healthy patients, but in patients with preexisting lung disease or obesity increases the likelihood of hypoxemia. Cephalad movement of diaphragm due to pneumoperotoneum, combined with Trendelenberg position may cause displacement of endotracheal tip and endobro-nchial intubation. A head down position also tends to shift the trachea upward, so that the tracheal tube anchored at the mouth may migrate into the right main stem bronchus. This tracheobronchial shift may be exacerbated during insufflation of the abdomen. Venous congestion of head and neck may compromise cerebral perfusion and produce intracerebral and intraocular hypertension. There is a decrease in mean arterial pressure and cardiac index, as well as increase in peripheral and pulmonary vascular resistance when patient is placed in reverse Trendelenberg, lateral tilt position with p n e u m o p - e r i t o n e u m . Anaesthetic agents may blunt these effects. There is also an increase in left ventricular end-systolic wall stress and decreased left ventricular end-diastolic area but left ventricular ejection fraction was maintained during a

study by trans-esophageal echocardiography. In head up position for upper abdominal surgery, there is improved pulmonary function at expense of decreased cardiac function. Nerve compression is a potential complication during the head down position. Overextension of the arm must be avoided. Shoulder braces should be used with great caution and must not impinge on the brachial plexus. Lower limb neuropathies (e.g. peroneal neuropathy, meralgia paresthetica, femoral neuropathy) have been reported after laparoscopy. Prolonged lithotomy position, as required for some operative procedures, can result in lower extremity compartment syndrome.

Anaesthetic management -An optimal anesthetic technique should provide excellent intraoperative conditions while ensuring rapid recovery, low incidence of adverse effects, and early return to daily activities. Anaesthetic approaches to laparoscopic surgery include infiltration of local anaesthetic with an intravenous sedative, epidural or spinal anaesthesia, or general anaesthesia. General anesthesia with muscle paralysis and tracheal intubation with positive pressure ventilation remains the preferred technique for most laparoscopic procedures for many reasons: the risk of regurgitation from increased intraabdominal pressure during insufflation; the necessity for controlled ventilation to prevent hypercapnia; the relatively high peak inspiratory pressures required because of the

pneumoperitoneum; the need for neuromuscular blockade during surgery to allow lower insufflation pressures, provide better visualization, and prevent unexpected patient movement; and the placement of nasogastric tube and gastric decompression to minimize the risk of visceral perforation during trocar introduction and optimize visualization. Muscle paralysis reduces the IAP needed for the same degree of abdominal distention. Ventilatory settings have to be adjusted according to respiratory and haemodynamic response of the patient. Large tidal volumes (12-15 ml/kg) prevent progressive atelactasis and hypoxemia and allows for more effective alveolar ventilation and CO_2 elimination. However this may cause excessive increase in intrathoracic pressure and thus deleterious cardiovascular effects that will result in an increased alveolar dead space. Isoflurane is the volatile anaesthetic agent of choice because it is less arrhythmogenic and causes less myocardial depression.

Nitrous oxide (N_2O) - N_2O is widely used in anaesthesia because of its amnesic and analgesic properties, as well as its ability to reduce the requirements of expensive inhaled and intravenous anesthetic drugs. However, the use of N_2O during laparoscopic procedures remains controversial because of concerns regarding its ability to diffuse into the bowel lumen, causing distention and impaired surgical access. As N_2O is more soluble than nitrogen, a closed air

containing space may accumulate N_2O more rapidly than it can eliminate. Use of N_2O has also been reported to increase the incidence of postoperative nausea and vomiting (PONV). A recent large study, reported that omission of N_2O from a propofol -based anaesthetic does not increase postoperative adverse events or the time to home readiness. Thus, there is no convincing reason to avoid N_2O during laparoscopic procedures. However, omitting N_2O from the anesthesia regimen may be an option in patients at risk or when there are surgical difficulties.

Local and regional anesthesia-Experience with local anaesthesia has been largely limited to brief gynaecologic procedures (laparoscopic tubal ligation, intrafallopian transfers) in young, healthy, and motivated patients and adjuncted with careful titrated sedation. Although postoperative recovery is rapid, patient discomfort and suboptimal visualization of intra-abdominal organs preclude the use of this local anaesthesia technique for laparoscopic cholecystectomy. Epidural or spinal anaesthesia represents another alternative approach for laparoscopic surgery in patients with certain associated comorbid condition like cystic fibrosis in which general anaesthesia carries more risk. Regional anaesthesia has numerous advantages such as early recovery, reduced PONV, lower postoperative pain, and shorter hospital stay. It is necessary to use lower IAP and reduce the degree of head-down tilt. A high level is required for

complete muscle relaxation and to prevent diaphragmatic irritation caused by gas insufflation and surgical manipulations of upper GI structures. But this causes myocardial depression and reduced venous return which aggravates the effects of tension pneumoperitoneum. Vagal mediated arrhythmias are also exacerbated. Of note, N_2O supports combustion and may increase the risk of fire and explosion when used with electrocautery. An obese individual with lung disease may not be able to increase spontaneous ventilation to maintain normocarbia in the face of a high (T2 level) regional block during insufflation and a 20° Trendelenberg position. Another disadvantage of a regional technique is occasional occurrence of referred shoulder pain from diaphragmatic irritation.

Monitoring during laparoscopy- Routine intraoperative monitors include ECG, pulse oximetry, blood pressure, pulse rate, and $EtCO_2$ are essential. However, ventilator performance, anaesthetic gases concentration and patient's temperature can be monitored depending upon the availability. As for the question of blood gases, $EtCO_2$ is most commonly used as a non-invasive substitute for $PaCO_2$ in evaluating the adequacy of ventilation during laparoscopic surgery. However, $EtCO_2$ may differ considerably from $PaCO_2$ because of ventilation-perfusion (V/Q) mismatching, and erroneous

clinical decisions may be reached if the two values are assumed to be equal, to change proportionally, or even to change in the same direction. $EtCO_2$ monitor is also useful for early detection of gas embolus. For haemodynamically unstable or compromised patient and patients with cardio-respiratory chronic diseases, careful monitoring of cardiovascular and blood gases are indicated. This also applies in case of obese patients. Radial artery cannulation for continuous blood pressure recording and frequent arterial blood gas analysis should be considered in patients with preoperative cardiorespiratory disease and in situations where intra-operative hypoxemia, high airway pressures, or elevated $EtCO_2$ are encountered. There is a need for a urinary bladder catheter and naso-gastric tubes to decompress the viscera and thus avoid injury to intra-abdominal contents during trocar insertion.

Post operative nausea and d vomiting (PONV) -PONV remains an important concern for surgical patients, as it is extremely unpleasant. PONV is a common complication for both inpatients and outpatients undergoing laparoscopic surgery, regardless of the anaesthetic technique used. However, the risks of PONV associated with Total Intravenous anesthesia (i.e., propofol-based anaesthetic) appear to lower than that associated with inhalation anesthesia recent data suggest that combinations of antiemetics administered prophylatically are more effective than either

antiemetic administered alone, particularly in high-risk patients. A multimodal approach to prevention of PONV includes use of combinations of droperidol 0.625–1mg, 5-HT$_3$ antagonists (ondansetron 4 mg or dolasetron 12.5–5mg), and dexamethasone 4–8 mg, as well as aggressive hydration, use of minimal doses of opioids, and good pain control.

Pain Management-While less intense and less prolonged compared to open operations, pain after laparoscopic surgical procedures may be quite severe, particularly in the early postoperative period. There is more visceral pain after laparoscopic procedures compared with parietal (i.e., abdominal wall) pain after open abdominal procedures. In addition, shoulder pain secondary to diaphragmatic irritation is a frequent occurrence after laparoscopy and can persist for as long as four days. There is a strong correlation between the severity of shoulder pain and the volume of residual subdiaphragmatic gas. Therefore, every attempt should be made to remove as much CO_2 as possible at the end of the procedure. The severity of postoperative pain has also been shown to be associated with the duration of surgery. In addition, stretching of the intra-abdominal cavity from higher insufflations pressures significantly increases the severity of pain.

There is increasing evidence suggesting multimodal analgesia techniques [i.e., combination of opioids, nonsteroidal anti-inflammatory drugs (NSAIDs),

and local anaesthetics (nerve block, infiltration)] provide more effective pain relief with fewer side effects and earlier recovery. Infiltration of the laparoscopy portals with a local anesthetic provides excellent analgesia that may outlast the duration of action of the local anaesthetic. However, it is important that the local anaesthetic is administered not only subcutaneously but also into the subfascial layers. Another simple and effective method of reducing the intensity of postlaparoscopic pain is intraperitoneal instillation of 20 mL of 0.25% bupivacaine between the liver and diaphragm (Table 2).

Opioids remain an important component of a balanced general anaesthic technique for minimal access procedures. Concern has been raised regarding narcotic induced spasms of the sphincter

of Oddi, leading to misinterpretation of intraoperative cholangiographic findings during laparoscopic cholecystectomy. Many opioids, including fentanyl, have been implicated and there are conflicting reports regarding the relative effects of individual opioids. Several drugs including glucagon, naloxone, and atropine can antagonize opioids induced spasm of the sphincter of Oddi. NSAIDs are now widely used in the perioperative period because of their opioid-sparing effects. Ketorolac administered intravenously in the early intraoperative period reduces intraoperative opioid require-ments, postoperative pain scores and analgesic require-ments. NSAIDs also appear to be effective when administered orally prior to surgery.

Table-2, Pain management

NSAIDs	Diclofenac Sodium buprofe COX2 inhibitors(Etorocoxib) Tenoxicam
Local anaesthetics	Intraperitoneal infiltration (Bupivacaine)Incisional infiltrationEpidural LA
Opioids	Systemic (Fenatanyl, Morphine, Sufentanyl)Intraperitoneal (Tramodol)Intrathecal morphine Epidural morphine, fentanyl
Others	Rectus sheath block Warm gas for insufflations

Complications of Laparoscopy procedure-Awareness of the potential complications associated with laparoscopic procedures should allow early detection and treatment, and improve patient care and safety. The complications associated with laparoscopy include those related to surgical instrumentation, creation of the pneumoperitoneum, and patient's positioning (Table 3).

Complications from surgical instrumentation-The initial access into the peritoneal cavity can be achieved using the Verres needle that is placed through a subumbilical incision. Because the Verres needle is placed blindly, there is a potential for its misplacement in the subcutaneous space, vascular space, viscus, omentum, mesentery, or retroperitoneum. Uncontrollable hemorrhage from injury to major vessels caused by surgical instrum-entation can cause significant hypotension. On the other hand, concealed bleeding from vessel injury can present in the postoperative period as a fall in hematocrit values. Stomach injuries can be reduced by gastric decompression using NG tube prior to placement of the Verres needle. Similarly, decompression of the urinary bladder by placement of a urinary catheter or asking patients to void prior to surgery should decrease the possibility of bladder trauma. The complications associated with the Verres needle may be avoided by placement of the first trocar through a minilaparotomy incision. Fulgaration has been associated with bowel burns and bowel gas explosions.

Cardiovascular complications-Although rare, acute cardiovascular collapse during laparoscopy has been reported and may be caused by profound vasovagal reaction, cardiac dysrhythmias, acute blood loss, myocardial

Table- 3, Complications during laparoscopy

Complications During Laparoscopy				
Intraoperative	Surgery related	Due to pneumoperitoneum	Cardiovasular	Arrhythmias Blood pressure changes
			Respiratory	Gas Embolism Subcutaneous- emphysema Pneumothorax Pneumomediastinum Pneumopericardium
		Trocar insertion	Vascular injur Organ injury	
	Positioning	Nerve injury Endobronchial intubation Cardiovascular and respiratory compromise		
	Thermal injuries			
Postoperative	PainNausea and vomiting Deep Vein Thrombosis Infections Spread of malignancy			

dysfunction, tension pneumothorax, venous gas embolism, severe respiratory acidosis, cardiac tamponade, excessive IAP, and anesthetic drugs. In addition to routine evaluation (i.e., depth of anaesthesia and intravascular volume status), it must be confirmed that the IAP have not exceeded 14 mmHg. If these measures are inadequate, deflation of the abdomen may be needed. In patients with a significant fall in cardiac output and high systemic vascular resistance, pharmacological interventions with nitroglycerin and/or ß-adrenergic agonists may be necessary to maintain haemodynamic status. Rarely, it may be necessary to convert to an "open" procedure if the significant impairment persists.

Pulmonary complications-The pulmonary changes during laparoscopy can cause severe hypoxemia and hypercarbia. The differential diagnosis of significant pulmonary dysfunction includes CO_2 absorption, hypoventilation (airway obstruction, ventilator or breathing circuit leaks), increased dead space (associated with abdominal distention, position of the patient, mechanical ventilation), endobronchial intubation, reduced cardiac output, CO_2 embolism, pneumothorax, pneumomediastinum, pneumopericardium, and subcutaneous emphysema.

Subcutaneous emphysema- Subcutaneous emphysema can occur from inadvertent extraperitoneal insufflations or from extension of certain laparoscopic surgical procedures that require intentional extra-peritoneal insufflations, such as inguinal hernia repair, renal surgery, and pelvic lymphadenectomy. Subcutaneous emphysema increases the area for CO_2 diffusion, which can result in significant hypercarbia and respiratory acidosis. It can be diagnosed by the development of crepitus and increase in end-tidal CO_2 concentrations. In most cases, subcutaneous emphysema resolves soon after abdominal deflation. If the emphysema extends to the chest wall and the neck, the CO_2 can track to the thorax and mediastinum, thereby resulting in pneumothorax or pneumomediastinum. Therefore, a chest *x-ray* should be obtained if a patient develops cervical emphysema.

Pneumothorax- Pneumothorax can occur from insufflated CO_2 tracking into the thorax through a tear in the visceral peritoneum, breach of the parietal pleura during dissection around the esophagus, or a congenital defect in the diaphragm. Early diagnosis and treatment of pneumothorax can be achieved by maintaining a high index of suspicion. It can be diagnosed by the development of increased peak airway pressures, reduced oxygen saturation, and in rare cases significant hypotension and cardiac arrest. Treatment of pneumothorax includes deflation of the abdomen and supportive treatment. If there is minimal physiologic compromise conservative treatment with close observation may be adequate because CO_2 is rapidly absorbed. In patients with severe compromise, placement of an intercostal cannula may be necessary, followed by a chest drain if re-accumulation occurs. Conversion to an open procedure might be necessary after stabilization.

Gas Embolism-Gas embolism is a potentially fatal condition if it is not managed timely. Gas may enter circulation if Verres needle or trochar directly punctures blood vessel. However, in some cases of gas embolus, no evidence of vascular injury is found. The effects of gas embolism depend on rate, amount and nature of gas. Magnitude of physiological disturbances caused by CO_2 is 6.5 times less than that of air because of its higher blood solubility. The diagnosis of gas embolism depends on the detection of gas emboli in the right side of the heart or on recognition of the physiologic changes from embolization. Early events, occurring with 0.5 ml/kg of air or less, include changes in Doppler sounds and increased mean pulmonary artery pressure when these monitors are used. Tachycardia, cardiac arrhythmias, hypotension, increased central venous pressure, alteration in heart tones (i.e. millwheel murmur), cyanosis, and ECG changes of right heart strain can develop, when the size of embolus increases to 2ml/kg of air. Slow infusion of gas results in absorption across pulmonary capillary-alveolar membrane without causing any clinical effect. At higher infusion rates, gas bubbles lodge in peripheral pulmonary circulation.

Pulmonary gas embolism causes increase in right ventricular afterload and may lead to right ventricular failure. Large bolus of gas may lead to complete mechanical obstruction of right atrium and ventricle. Paradoxical emboli can also occur. This provokes neutrophil clumping, activation of coagulation cascade and platelet aggregation can occur. Release of chemical mediators produces pulmonary vasoconstriction, bronchospasm, and pulmonary edema and occasionally delayed pulmonary hemorrhage. Gas bubbles attached to fibrin deposits and platelet aggregates also mechanically obstruct the pulmonary vasculature, further increasing pulmonary vascular resistance. Portal venous gas embolism causes an initial trapping of gas in hepatic portal circulation. This can subsequently lead to delayed manifestations in the post-operative period. Treatment of CO_2 embolism consists of immediate cessation of insufflations and release of pneumoperitoneum. The patient is placed in steep head down and left lateral (Durant) position. Discontinuing N_2O allows ventilation with 100% O_2 to correct hypoxemia and reduces the size of the gas embolus and its consequences. If these simple measures are not effective, a central venous catheter or pulmonary artery catheter may be introduced for aspiration of the gas. Hyperbaric oxygen is strongly considered if cerebral gas embolism is suspected.

Hypothermia-It is generally expected that the degree of hypothermia during a closed procedure such as laparoscopy would be less than that during an open procedure because the abdominal contents are not exposed to the atmosphere. However, the incidence of hypothermia during laparoscopic procedures is similar to that of open abdominal operations. Although built-in heating elements to warm the insufflating gas have become available commercially, warming (without humidification) of insufflations gas does not prevent hypothermia and is thus unnecessary during laparoscopy. Some authors have reported that the use of heated humidified insufflating gas may reduce postoperative morbidity, including postoperative pain. However, larger studies are necessary to show the advantages of humidifying insufflating gas. Prevention of surgical hypothermia should address other, more significant, sources of heat loss. It is important to take an aggressive approach in maintaining patients' temperature including humidification of anaesthetic gases, infusion of heated intravenous fluids, pre-warming irrigating fluids, and forced-air warming to improve postoperative outcome.

Postoperative complications-After deflation of the abdomen, it may require approximately 45 min for the arterial CO_2 concentrations to return to pre-insufflations values. Of note, normalization of CO_2 concentration may take longer after extra-peritoneal insufflations than that after intra-peritoneal insufflations. Impaired postoperative ventilation from residual anaesthetics and/or neuromuscular blockade may result in significant hypercapnia. In patients with significant respiratory dysfunction and restricted CO_2 clearance, positive pressure ventilation may be required in the postoperative period until the patient can eliminate the CO_2 load with resumption of spontaneous respiration. Increased IAP during pneumoperitoneum has been reported to cause venous stasis that can increase the potential for deep vein thrombosis and pulmonary embolism. Measures to reduce venous stasis such as graduated elastic compression stockings are indicated in the perioperative period. Minimal tissue trauma with laparoscopic techniques, facilitating early postoperative ambulation, also reduces risk.

Laparoscopy in pregnancy-The most common non-obstetric surgical procedures during pregnancy are adnexal surgery, appendectomy, and cholecystectomy, and they are amenable to laparoscopic surgery. Various concerns in laparoscopic procedures during pregnancy include issues of increase risk of miscarriage, premature labour, and damage to gravid uterus. The increased IAP and hypercarbia leads to acidosis, increased heart rate, arterial pressures in human fetus but these changes are minimal. Provided maternal $PaCO_2$ is maintained at normal levels, fetal placental perfusion

pressure and blood flow, pH, and blood gas tensions are unaffected by insufflations and deflation. The operation should be done in the 2nd trimester to minimize the risk of premature labour and to maintain adequate intrabdominal space. Tocolytics, though controversial, may be used to arrest preterm labour. Open laparoscopy should be used for abdominal access to avoid damaging the uterus. Fetus should be monitored using transvaginal ultrasonography. Mechanical ventilation must be adjusted to maintain a physiologic maternal alkalosis. Gasless laparoscopy is an alternative to avoid the potential side effects of CO_2 pneumoperitoneum.

Laparoscopy in Paediatrics- Laparoscopy is also being done in infants and children. General anaesthesia with controlled ventilation is recommended in all paediatric patients undergoing laparoscopy. CO_2 pneumoperitoneum induces similar changes in respiratory mechanics to those reported in adults. CO_2 absorption may be more intense and faster in infants than in adults because the peritoneal surface area referred to body weight is greater in infants. Haemodynamic changes observed in children are similar to those reported in adults but may be offset to certain extent by preloading with 20ml/kg of fluid before creating carboperitoneum. IAP should be restricted to 10-12 mm Hg. Intraoperative oxygenation can be improved by addition of PEEP.

Laparoscopy for Gynaecologic surgeries-Gynecologic surgeries

may be diagnostic (dye studies, biopsy), or therapeutic (e.g. tubal ligation, Laparoscopic Assisted Vaginal Hysterectomy). Premedication includes aspiration prophylaxis, anxiolysis (alprazolam/diazepam) and other medications (for associated comorbid diseases). General anaesthesia with inhalational or intravenous drugs with a secured airway is the preferred technique. Neuromuscular blockade with an optimal anesthetic depth is required for laparoscopic procedures, as inadequate muscle relaxation may resist abdominal distension and visualization and uncontrolled diaphragmatic movement may preclude finer maneuvers during laparoscopy. Usually patient is positioned in Lithotomy with Trendelenberg tilt. Instrumentation of the vagina is needed to manipulate the uterus or to inject the dye. Manipulation of the pelvic organs often cause significant bradycardia. Postoperative nausea and vomiting is more after gynaecological surgeries.

Hysteroscopy – In this procedure direct examination of the uterine cavity using a fiberoptic endoscope for diagnostic or surgical intervention is done. Uterine distension is necessary for visualization. The use of irrigation fluid also permits removal of blood and detritus, and dissipation of heat. Distension is usually achieved with fluid like saline, glycine, and dextran. Fluid absorption may occur excessively leading to circulatory overload along with dilutional hyponatremia, hypoproteinemia. Symptoms of TUR

syndrome may follow. Measurement of fluid instilled and retrieved can be difficult but should be attempted because of the possible effects of fluid absorption. CO_2 may occasionally be used. This may leak through fallopian tube in the abdomen leading to features as of pneumoperitoneum in long surgical procedures. Sometimes LASER is used for surgical corrections and universal safety precautions of laser must be meticulously followed. Regional (epidural or spinal) anaesthesia is suitable along with light sedation.

Laparoscopic Surgical Procedure - Among various abdominal procedures done by laparoscopic approach, some of them are cholecystectomy, Nissen fundoplication for gastroesophageal reflux disease, antero-posterior resection (APR), splenectomy, and adrenelectomy.

Laparoscopic Cholecy -stectomy - Now standard treatment for symptomatic gallstones is laparoscopic cholecystectomy. Patients presenting with acute cholecy-stetis may be jaundiced and/or dehydrated. Special attention should be paid to renal function. If coagulation is deranged, 10 mg of vitamin K, fresh frozen plasma should be considered. Standard intravenous induction, nondepolarising muscle relaxation and tracheal intubation are the anaesthetic technique.

Gastrointestinal Endoscopy- Various diagnostic and therapeutic procedures (upper endoscopy, foreign body

removal, sigmoidoscopy, colonoscopy, endoscopic retrograde cholangiopancreatography (ERCP), esophageal dilatation, stenting, transjugular intrahepatic portosystemic shunt) are being performed. Usually the procedures can be accomplished under sedation using benzodiazepine with or without opioid. Sedation dose of propofol as continuous infusion remains to be an alternative technique. Upper GI Endoscopy is performed for diagnostic procedures, such as biopsy, and for therapeutic procedures, such as retrieval of foreign body, esophageal varices sclerotherapy or band ligation, dilatation of esophageal strictures, and placement of percutaneous endoscopic gastrostomy. In most of the patients procedures can be done with conscious sedation. But in uncooperative patients and children, general anaesthesia may be required. With GA, patients usually require tracheal intubation to protect the airway and facilitate passage of the endoscope. Local anaesthesia may be sprayed into the oropharynx to facilitate passage of the endoscope; this can abolish the gag reflex, and increasing the risk of aspiration. Bite block is inserted to prevent the patient from biting the endoscope and damaging both their teeth and the endoscope. Care and attention should be paid to pressure areas; particularly the eyes, lips and teeth as prone or semi prone position may be required. Extreme rotation of the neck should be avoided. Colonoscopy is painful secondary to the insertion and

manipulation of the endoscope, and may be associated with cardiovascular effects, including dysrhythmias, bradycardia, hypotension, myocardial infarction, and death. The mechanism is not exactly known but probably mediated by the autonomic nervous system when stimulated by anxiety or discomfort. Opioid and midazolam provides adequate patient comfort.

Endoscopic Retrograde Cholangiopancreatography (ERCP) - ERCP is important in the diagnosis and treatment of both biliary and pancreatic disease. Conscious or deep sedation technique is recommended. Opiods should be avoided as they may cause spasm of sphincter of Oddi. Patient presenting for emergency ERCP may have significant co morbidity like acute cholangitis with septicemia, jaundice with liver dysfunction, coagulopathy, bleeding from varices and hence require adequate optimization.

Anesthesia for Endo-Urologic Surgery-Incisional surgery in urology is being replaced by endoscopic procedures in which endoscopes are passed along natural pathways (e.g. urethra, ureter) or through small incisions. As with anaesthesia for any other group of elderly patients, intercurrent diseases are of the utmost importance to the anaesthetist. Where obstruction of the lower urinary tract has been long standing, it is of great importance to ensure that the kidney function is normal and that the levels of urea and creatinine are not rising.

Cystourethroscopy and ureteral procedures - Cystourethroscopy is commonly performed for lower urinary tract disease. Earlier in the days of rigid endoscopes, general or regional anaesthesia was required for patient comfort. After the introduction of flexible endoscopes, these procedures can be accomplished using local anaesthesia like lignocaine jelly. For longer and extensive procedures, conscious sedation may be given for patient comfort. If general anaesthesia is required for cystoscopy or urethral procedures, a laryngeal mask airway is a good alternative for securing airway. A spontaneously breathing technique with volatile anaesthtics is well accepted. TIVA with propofol is another suitable alternative. Uretroscopy is an extension of cystoscopic techniques providing access to the upper urinary tract and kidney for diagnostic endoscopy and biopsy, removal of ureteral and renal calculi, passage of a ureteral stent, dilatation and incision of strictures, fulguration of tumors and laser treatments. Ureteroscopy usually requires dilatation of ureteral orifice and intramural ureter, often necessitating regional or general anaesthesia.

Transurethral Resection of the Prostate (TURP) - TURP is the primary treatment for symptomatic benign prostatic hyperplasia. After performing the cystoscopy, a specialized instrument with an electrode capable of both coagulating and cutting tissue, known as resectoscope is then introduced through a

modified cystoscope into the bladder. The tissue protruding into the prostatic urethra is resected with resectoscope. Continuous irrigation of the bladder is required to maintain visibility, distend the operative site, and remove dissected tissue and blood. Patient is required to be in lithotomy position. Usually the patient is an elderly one and may have associated various comorbid conditions and needs to be evaluated and optimized preoperatively. The prostate gland contains a rich plexus of veins that can be opened during surgical resection. If the pressure of the irrigating fluid during TURP procedure exceeds venous pressure, intravascular absorption of the fluid may occur via these open venous sinuses. The ideal irrigating fluid, for use during TURP should be isotonic and non-hemolytic if absorbed. In addition, it should be non-electrolytic (prevents dispersal of electric current), transparent (to allow clear visibility for the surgeon), non-metabolized, nontoxic, rapidly excreted, and inexpensive. Among variety of irrigating fluids, some of them are distilled water, glycine (1.2%, 1.5%), sorbitol (3.3%), mannitol (5%), glucose (2.5%), urea (1%), cytal (2.7% sorbitol and 0.54% mannitol). Distilled water was used because of its non-conductance and good surgical visibility. Because of its low tonicity, it gets absorbed and cause massive intravascular haemolysis, haemoglobinemia, renal failure and dilutional hyponatremia. Different solute has been added to water to make its osmolality closer to that of plasma. It is essential that the anaesthesiologists be aware of the type of irrigating solution being used during an endoscopic procedure, because of this fluid can be associated with numerous perioperative complications. Glycine, the most preferred fluid is metabolized in liver to ammonia and glyoxilic acid. Depressed mental function and coma has been reported secondary to hyperammonemia. Visual disturbances, including blurred vision and transient blindness, have also been reported following TURP procedures with glycine containing irrigation fluids. Transurethral resection (TUR) syndrome (water intoxication syndrome) may manifest with various neurologic and cardiopulmonary symptoms. Its principal components are respiratory distress secondary to volume expansion from rapid intravascular absorption of the irrigating fluid, dilution of electrolytes and proteins by electrolyte free irrigating fluid, and symptoms related to type of irrigating fluid. The amount of irrigating fluid absorbed during TURP is 10 – 30 ml /min of resection time. The amount of irrigation fluid absorbed is directly related to the number and size of venous sinuses opened, duration of resection, hydrostatic pressure of the irrigating fluid, and venous pressure at the irrigant-fluid interface. In an attempt to prevent excessive fluid absorption, it is recommended that the resection time be limited to less than 60 minutes and height of the fluid to be no more than 60 cm so as to maintain an intravesical pressure of no more than 15 cm of water. Clinical manifestations of TUR syndrome range from mild (restlessness, nausea, shortness of breath, dizziness) to severe (seizures, coma, hypertension, bradycardia, cardiovascular collapse). In the awake patient with a regional anaesthesia, a classic triad of symptoms of an increase in both systolic and diastolic pressures associated with an increase in pulse pressure, bradycardia and mental status changes. During general anaesthesia, many of the more subtle signs of TUR syndrome are obscured, making early diagnosis more difficult. The acute dilutional hyponatremia is responsible for many signs and symptoms of TUR syndrome. Prompt intervention is necessary when neurological or cardiovascular complications of TUR procedures are recognized including adequate oxygenation, and circulatory support. Mild symptoms may be treated with fluid restriction and loop diuretics. Severe symptoms require administration of hypertonic saline. The rate at which serum sodium is increased should not exceed 12mEq/L in a day.

Serum Na+(mEq/L)	CNS changes	ECG changes
120	Confusion, restlessness	Possible widening of QRS complex
115	Somnolence, nausea	Widened QRS complex, Elevated ST segment
110	Seizures, coma	VT/VF
100	Cardiac arrest	Asystole

Regional anaesthesia is considered as an anaesthesia of choice. It allows the patient to remain awake during the TUR procedure and facilitate early diagnosis of TUR syndrome or extravasation of irrigation fluid. Also patient may have intercurrent chest disease, and may benefit after surgery from not having a general anaesthetic. Regional anaesthesia also reduces the incidence of postoperative thromboembolic disease in these elderly high risk patients. Spinal anaesthesia is usually preferred over epidural anaesthesia as sacral segments are sometimes inadequately blocked with lumbar epidural anaesthesia. A T10 dermatome level is required to block the pain from bladder distension by the irrigating fluid. Warm irrigating fluid should be used in order to maintain core temperature.

Laparoscopic Radical Prostatectomy-Laparoscopic Radical Prostatectomy allows optimal operative procedure with reduced morbidity as compared to open procedure. The quality of surgery is improved by a better visualization of the operative site owing to the optical magnification and the maneuverability of the laparoscope. In addition to the improved postoperative course, laparoscopic radical prostactemy allows better preservation of periprostatic vascular, muscular and neurovascular structures. General anaesthesia with patient positioned in supine with head down tilt with legs flexed and abducted is the requirement for the procedure. The arms are kept by the side of the patient to avoid prolonged abduction of the shoulder and minimize chances of brachial plexus injury. Perioperatively, an antithrombotic regimen should be considered in view of risk factors of cancer sugery, pelvic surgery and laparoscopy. Complications associated with the procedure include bladder or ureteric injury leading to urinary leak, vascular injury, bowel injury.

Transurethral Resection of Bladder Tumors-Endoscopic transurethral resection is requires for diagnosing and treating bladder cancers. The choice of anaesthesia is as same as for TURP. If the bladder tumor lies near the obturator nerve, the electrocautery may cause the thigh muscles to contract violently, resulting in inadvertent bladder perforation, regardless of type of anaesthesia. If the tumour is vascular, patient may require blood transfusion.

Percutaneous Renal Procedures-Percuataneous nephrostomy (PCN) is done for various urologic problems like relief of renal obstruction, stone removal, biopsy of tumors, ureteral stent removal. Percutenous nephrolithotomy (PCNL), a procedure to remove renal calculi too large to be removed by lithotripsy, is one of the most common endourologic procedures, requiring dilatation of nephrostomy tract. Preoperative renal function should be reviewed. As patient is usually placed in prone position and movement should be avoided during renal puncture, general anesthesia with controlled ventilation is the preferred technique. Precautions pertaining to prone position should be meticulously taken. Eyes protected, shoulders should not be over abducted, pillow to be placed under chest and pelvis to free the abdomen for ventilation. Neck should not be excessively turned. Warm irrigation fluid will maintain patient's temperature. One should be vigilant of complications like trauma to spleen, liver, kidney, colon, pleura, and may have excessive bleeding. Intravenous absorption of irrigation fluid during percutaenous renal procedures

can have similar symptommatology as of TUR syndrome.

Urologic Laparoscopy-Laparoscopic procedures required in urology include diagnostic procedures for evaluating undescended testes, orchipexy, varicocelectomy, bladder suspension, pelvic lymphadenectomy, nephrectomy, partial nephrectomy, nephrou-reterectomy, adren-aectomy, prostectomy, and cystectomy. Laparoscopic urologic procedures differ from conve-ntional laparoscopy in several respects. Many structures in the genitourinary system are extraperitoneal (pelvic lymph nodes, bladder, ureters, adrenal glands, kidney) and urologists often prefer extraperitoneal insufflation. CO_2 absorption is greater with extraperitoneal compared with intraperitoneal insufflations and also do not achieve a plateau $EtCO_2$ thus mandating monitoring and adjusting ventilatory parameters. Subcutaneous emphysema is frequent in these patients and may extend all the way to the head and neck. Because of significant increases in intraabdominal and intrathoracic pressures as a result of insufflated carbon dioxide, a steep Trendelenberg position, and lengthy procedures, a general anaesthesia with controlled ventilation is the method of choice. Despite adequate hydration, intraoperative oliguria may occur and be followed by diuresis in the immediate postoperative period.

Laparoscopic adrenalectomy-Laparoscopic surgery is appropriate for benign (functional / nonfunctional) adrenal tumors. Surgery is performed in modified flank (kidney) position. Table flexion for optimal positioning may lead to neuromuscular injury.

Laparoscopic Donor Nephrectomy-Laparoscopic donor nephrectomies are picking up the pace as its equivalent safety compared to open technique and also it reduces postoperative pain, shorten hospital stay, allow faster return to work, and improve cosmesis. General anaesthesia, securing of airway with endotracheal tube is the preferred technique. Nasogastric tube and Foley's catheter are placed. Patient is placed in modified lateral decubitus position at 45 degree and torso is allowed to rotate posteriorly to allow exposure to lower midline. After kidney dissection , the kidney is removed via paraumblical incision.

Laparoscopy for Bariatric Surgery - Among various procedures available for obesity, usual procedure done includes laparoscopic banding, endoscopic placement of bioenteric intragastric balloon. Concerns for an anaesthesiologist includes preoperative evaluation and optimization, intraoperative monitoring and management, and postoperative care. During the preoperative evaluation, attention should focus on issues unique to the obese patient, particularly cardiorespiratory status and airway (folds of tissues in mouth and pharynx, short thick neck, suprasternal, presternal and posterior cervical neck points towards difficult airway). Appetite suppressants like fenfluramine have catecholamine depleting effect so that indirect acting vasopressors will be ineffective necessitating the need of direct acting vasopressors. Pulmonary hypertension, which is reversible after stoppage of fenfluramine, is another concern. The anaesthetic technique of choice for laparoscopic bariatric surgery is general anaesthesia with controlled ventilation. Difficult venous access, extra width OT table, large size BP cuff, difficult intubation are the other concerns. For pain management opioid, PCA should be used very cautiously. NSAIDS and local infiltration of trocar sites may be preferred.

Bronchoscopy- Bronchoscopy may be required for various diagnostic (biopsy, lavage) and therapeutic (foreign body removal) reasons. Due precautions must be taken as these patients may be respiratory compromised. Bronchoscopy may be done using a rigid or a flexible bronchoscope. Patient profile and the procedure to be done is the decisive factor for the type of anaesthetic management. For foreign body retrieval in children inhalational induction is preferred. After an adequate depth of anaesthesia has been attained, larynx may be sprayed with local anaesthestic. Muscle relaxants and thus IPPV are often avoided during bronchoscopy, as spontaneous ventilation is desirable, providing greater flexibility and additional time for endoscopist. Furthermore, IPPV

could distal migration of foreign body. In addition, if foreign body produces a ball valve phenomenon, the use of IPPV of the lungs could contribute to hyperinflation and possibly pneumothorax. Spontaneous ventilation technique may require higher concentration of inhalational anaesthetic to avoid any movement during anaesthesia, which is sometimes difficult to attain in these children considering the condition of lungs. However, the choice of anaesthesia depends upon the site of the foreign body, the clinical condition of the patient, the experience of surgeon and the experience of the anaesthetist. During bronchoscopy, anaesthesia is maintained using volatile anaesthetics and oxygen and muscle relaxant if patient's ventilation is being controlled.

Thoracoscopic procedures- Video assisted thoracoscopic surgery (VATS) has largely superseded thoracoscopy because of superior quality of visualization of the operative field that can be obtained, and because it frees the surgeon's hands to manipulate operating instruments rather than holding the scope to maintain a view. The surgical and anaesthetic considerations are essentially the same for VATS and thoracoscopy. The VATS is indicated for pleural, pulmonary, mediastinal, and esophageal pathology. VATS permits drainage of pleural effusions, visualization and biopsy of pleura and lung parenchyma and reexpansion of the lung under direct vision. VATS is the early

intervention of choice in the management of empyema, as drainage, adhesiolysis, irrigation and decortication can all be performed thoracoscopically. Resection of emphysematous bullae, division of pleural adhesions, decortication and pleurodesis may be carried out via VATS, and lung reexpansion directly visualized. Peripheral solitary lung lesions and pulmonary metastases may be biopsied and resected. Lung volume reduction surgery in patients with extensive emphysematous lung disease and poor respiratory reserve can be done via VATS. Discrete lesions and lymph nodes of the mediastinum may be biopsied under direct vision. Thymectomy in patients with myasthenia gravis has been carried out using VATS. VATS allows direct assess to the pericardium for biopsy, and a pericardial window may be created for drainage of a pericardial effusion. Enucleation of benign tumors, treatment of benign motility disorder, and preoperative staging of malignant esophageal disease is possible via VATS. VATS has been reported in the management of multiple disease of the thoracic spine , including disc herniation, disc space abscesses, and spinal deformities. VATS has several advantages over formal thoracotomy: decreased incidence of lobar atelectasis, less diaphragmatic disturbances, and decreased analgesic requirement postoperatively.

The patient is generally placed in the lateral decubitus position. The table is maximally flexed, or

a support is placed underneath the thorax, in an attempt to maximize the exposure of the thorax and widen the intercostal spaces. One lung ventilation is required, and the nondependent lung collapsed before insertion of the trocars to prevent iatrogenic trauma to the lungs. For thymectomy the patient may be kept supine. Anaesthetic considerations: Patients presenting for VATS fall within a broad spectrum of the disease. Careful patient selection after thorough evaluation and optimization of preoperative condition is as essential as for thoracotomy. Adequate collapse of lung may not always be possible because of adhesions and may lead to lung injury during surgical manipulation. The anaesthetic technique should allow for a variable length of procedure and possibility of conversion to formal thoracotomy. While extubation is usual at the end of VATS procedures, high dependency or intensive care facilities may be required to accommodate patients with poor preoperative respiratory function.

In addition to routine preoperative tests, blood should be grouped and saved or cross-matched since the procedure is associated with the potential for major hemorrhage. Pulmonary function tests, and arterial blood gases in patients with severe lung disease, are helpful in assessing the patient's suitability for one lung ventilation and need for postoperative support. Chest X-ray and computerized axial tomograms should be reviewed,

as the nature and extent of adhesions or tumor invasion may preclude collapse of the lung or passage of the endobronchial tube.

Premedication is tailored to individual requirements; the use of benzodiazepine for anxiolysis and sedation is satisfactory. Opioids may be associated with excessive respiratory depression in patients with poor respiratory reserve. Antisialogogues may lead to insipissated secretions, predisposing to increased postoperative complications. Apart from routine noninvasive monitoring, invasive arterial monitoring (for blood pressure and blood gas analysis) is also required. Thoracoscopy may be performed under local, regional or general anaesthesia depending upon the procedure to be performed. Local anaesthesia is by infiltration of the incisions and parietal pleura. Regional techniques used are intercostals nerve blocks paravertebral blocks or thoracic epidural block. The choice of anaesthetic technique is aimed at optimizing surgical conditions while ensuring maximal patient comfort and safety. Local and regional anaesthesia are rarely used in isolation except in critically ill patient, but are frequently used as an adjunct to general anaesthesia. Most VATS are performed under general anaesthesia with one lung ventilation provided through double lumen endobronchial tube placed in the left main stem bronchus and verified bronchoscopically. Muscle relaxation facilitates intubation and ventilation, and application of IPPV prevents mediastinal shift and paradoxical respiration during one lung ventilation. One lung ventilation is attained preferably using a double lumen tube, but single lumen endobronchial tube or bronchial blocker may also be used. Large bore peripheral venous access should be established in view of the potential for significant haemorrhage. Central venous access may be required depending on the patient's condition and the nature of the procedure.

The majority of the patients undergoing VATS may be extubated in operating theatre, or early in recovery. Supplemental oxygen is administered and the patient nursed sitting up to optimize lung mechanics. Patients with severe preexisting lung disease may require prolonged ventilation, and in these cases endobronchial tube should be replaced by single lumen endotracheal tube at the end of the procedure. Postoperative chest X-ray is taken in recovery to exclude pneumothorax, persistent atel-ectasis or haemothorax, and to check the correct positioning of the chest drains.

Early mobilization is required. A multimodal approach to pain management is generally used.

Intraoperative complications like hypoxia on one lung ventilation, heamorrhage, air embolism, surgical emphysema, cardiac arrhythmias, trauma to liver or paraesophageal hernia, and creation of aorto-pleuro-cutaneous fistula should be detected early and managed in time.

Additional problems during thoracoscopic procedures:

- Complete isolation of one lung mandatory to provide surgical access
- Application of CPAP to the isolated lung not practicable as it causes partial expansion of lung
- Pneumothorax best created by opening the taps of the cannulae to atmosphere and allowing lung to collapse under own elastic recoil
- Pressure in pneumothorax must be carefully regulated to prevent excessive mediastinal displacement and cardiovascular collapse
- Suctioning within enclosed thoracic cavity can lead to negative pressure within the hemithorax and mediastinal displacement. Air entry vent should be inserted
- Damage to heart is potential complication

Mediastinoscopy- Mediastinoscopy is carried out to assess mediastinal lymph nodes. In mediastinoscopy, a medistinoscope is passed into the pretracheal area via a small incision above the suprasternal approach. Anaesthesia is maintained with a single lumen endotracheal tube. Patient is positioned supine with a sandbag under the shoulders, the head on a ring, and neck extended. Wide bore cannula must be secured, as vascular structures may be biopsied in error.

Conclusion -With improvement in technology and growing surgical expertise, more extensive

and prolonged laparoscopic procedures will be performed in a wide range of patients. A systemic approach and a good intraoperative monitoring can easily decrease the incidence of complications. An alert and experienced anaesthesiologist and laparoscopic surgeon are the main keys to avoid complications in laparoscopic surgery. Modification of the anesthetic technique and the prevention of common postoperative complic-ations including pain, nausea, and vomiting using the multimodal approach should allow early recovery. Although the laparoscopic approach provides significant benefits, a thorough understanding of the associated cardiopulmonary changes and the potential complications are necessary to maintain patient's safety.

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Obesity in Children

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Obesity is defined as body weight which is more than 120% of the expected median weight for the child's sex and height age. Weight for height more than 95th centile is also suggestive of obesity. According to World health organization (WHO) definition, body mass index (BMI) more than 30 or more than 95th centile for age and gender is taken as Obesity. A BMI between the 85th and 95th centile is considered overweight.

Prevalence-There is a sharp rise in the incidence of obesity worldwide. According to the National health examination survey (NHANES) IV[1], the prevalence of obesity among children aged 6-11 years has increased three-fold from 4% in early 1960s to 13% in 1999. Children in the age group of 12-19 years too showed a similar increase from 5% in 1996 to 14% in 1999. In India according to a recent study in Pune[2], the prevalence of obesity in affluent school boys was 5.7% and that of overweight close to 20%.In another study among adolescent females in Delhi[3], the prevalence for obesity and overweight was found to be 5.3 and 15.2% respectively.

Theories of obesity-Obesity is a result of imbalance between energy intake and expenditure. The rapid increase in obesity rates in western countries is due to their high fat, high calorie diet and rapid change from an active to a sedentary lifestyle. This kind of behavior is soon catching up in the Indian subcontinent too and has accounted for the recent increased rate of obesity in affluent Indian children. According to thrifty gene theory[4], individuals exposed to harsh living conditions and famines develop genetic selection with highly efficient metabolism. When these populations migrate to affluent industrial societies, they are predisposed to obesity because of their low metabolic rates. Another hypothesis is that insulin producing cells of pancreas and insulin sensitive tissues in the body adapt in response to poor nutrition during fetal and infant life resulting in decreased growth in early life at the cost of increased risk of obesity and type 2 diabetes in later childhood and adulthood[5]. This hypothesis is referred to as the thrifty phenotype hypothesis.

Pathogenesis-Balance between energy intake and expenditure is controlled by interaction between peripheral signaling, effector system and neuroendocrine systems. Leptins play an integral part in such interactions. Decreased plasma insulin and leptin levels stimulate the production of neuropeptide Y and inhibition of sympathetic activity. This in turn results in modifications in the effector system in hypothalamus which results in synthesis of Orexin A and B leading on to increased feeding and decreased activity. On the other hand, increased plasma insulin and leptin levels result in increased secretion of melanocyte stimulating hormone (MSH), thyrotrophin, cortico-trophin and interleukin 1B from the hypothalamus which inhibit the Orexins and thereby lead to decreased intake and increased expenditure. Leptins released from peripheral adipose tissues acts on specific leptin receptors in the brain directly and results in increased secretion of POMC(Pro-Opio Melano Cortin) which is further broken down to various peptide hormones like prohormone convertase I (PC I) by proteases. PC I stimulate the production of alpha MSH which acts on receptors (MC4R/MC3R) in hypothalamus and inhibit feeding and increases expenditure. Growth hormone is known to influence feeding behavior and sense of well being in humans. The concentration of growth hormone and its receptors are present in sufficient quantities in regions of the brain that is known to control feeding like that of hypothalamus and amygdale. Ghrelin, a gastric hormone exerts a stimulatory effect on appetite and fat accumulation. Circulating ghrelin

levels are usually low in obesity and in states of positive energy balance. However experimental studies show that for the central action of ghrelins, an intact growth hormone receptor signaling is a must.Mutations in the genes controlling the satiety centres have been identified in idiopathic obesity. Mutations in ob/ob; lep ob; db/db; lep db genes have been identified. These mutations are inherited in a autosomal recessive fashion and produce severe hyperphagia, obesity, type 2 DM, defective thermogenesis and infertility. Mutations in the leptin receptor gene and prohormone convertase I genes have also been identified. This is manifested as obesity with impaired processing of insulin, DM, hyperinsulinemia and ACTH deficiency due to impaired processing of Proopiomelanocortin (POMC). Mutations in POMC gene cause severe early onset obesity with ACTH deficiency and red hair.

In addition to neuroendocrine regulation and genetic predispositions, family environmental factors too can play a role in pathogenesis of obesity. Parents who are overweight themselves or have difficulty in controlling their own eating behavior or those who are concerned about their children's risk of overweight may adopt controlling methods of child feeding and these control attempts may interact with genetic predispositions to promote the development of problematic eating styles and childhood overweight.

The various risk factors for obesity have been summarized as follows:

- Obese parents or obese siblings-If one child or parent is obese, 40% chance of sibling obesity; If both parents are obese, 80% chance of obesity in offspring

- Maternal age more than 35 years

- Single child or single parent

- Early rebound of BMI following dip in infancy

- Lack of breast feeding

- Formula feeding.

Types of Obesity-For the purpose of understanding the etiology of Obesity and to help in management decisions, Obesity can be broadly classified as endogenous obesity and exogenous variety. Endogenous obesity is a result of genetic, endocrine or neurological syndromes. They account for less than 1% of all obesity in childhood. Family history of obesity is uncommon. They have a retarded bone age and their height is usually below the 25th centile and they have other abnormalities on physical examination. On the other hand, no physical dysfunction is present in exogenous obesity other than an excessive calorie intake. Their height is usually above the fiftieth centile and they have an advanced bone age.

Aetiology of Obesity

- Nutritional- Familial; Nonfamilial

- Inactivity

- Endocrineobesity-Hypothyroidism; GH deficiency; Cushings syndrome; Hypogonadism; Polycystic ovary disease; Hyperinsulinism

- Hypothalamic syndrome- Post traumatic; Post inflammatory; Neoplastic

- Genetic- Laurence Moon Biedl syndrome; Praderwilli syndrome

- Iatrogenic drugs-Corticosteroids; Sodium valproate

- Miscellaneous- Severe mental retardation; Psychosocial factors; Ethnic factors; Familial.

Assessment of the obese child or Adolescent [6]-History regarding ethnicity, early medical history, details of pregnancy including maternal gestational diabetes, birth weight, developmental history, pubertal or menstrual history, the duration of weight gain, effect on daily activities, time spent on sedentary activities like watching television and playing computer games. Dietary history should focus on the normal meal pattern consumption of junk foods, binge eating and food fads. Family history should include relative weights of other family members, history of type 2 diabetes, cardiovascular diseases or obstructive sleep apnea in family members. History relating to complications of obesity likes psychological problems (victim of teasing and bullying) snoring, breathing difficulty; musculoskeletal problems etc have to be explored.

Examination includes dysmorphology examination, measurement of anthropometric parameters namely height, weight, head circumference, triceps skin fold thickness, waist circumference and description of

adipose tissue distribution .Tanner staging of pubertal development, measurement of blood pressure and looking for stigmata of endogenous obesity like hump, striae, moon face, hirsutism etc as well as presence of acanthosis nigricans which hypothyroidism. Plasma Leptin and fasting insulin levels should be estimated. Screening for genetic mutations, karyotyping or chromosomal studies for genetic causes of obesity like Praderwilli syndrome, Down syndrome and Laurence Moon- Biedl syndrome. complication of obesity although it occurs only in a minority of patients. Increased protein trafficking across the glomeruli can cause glomerulosclerosis. More than 40% of obese individuals have non alcoholic steatohepatitis and weight

Table- 1 Medical evaluation of overweight children [7]

BMI	Risk factors	Diagnostic tests
85 – 95th Centile	No	Fasting Lipid Profile
	Yes	Fasting Lipid, Fasting Insulin & Glucose, Hba1c,Blood Chemistries
>95th Centile	Yes or No	Fasting Lipid Profile, Fasting Insulin And Glucose, Hba1c, Blood Chemistries
Depending on History & Physical		Sleep Studies, ECG Holter, Extremity Radiographs, Thyroid Function Tests

may herald the onset of type 2 diabetes. Systemic examination should focus on presence of hepatomegaly, cardiac and respiratory system involvement intellectual impairment, visual impairment (field defects, night blindness), examination of musculoskeletal system, skeletal muscle power, gait etc.

Investigations- Baseline- blood sugar, triglycerides, cholesterol, blood urea, creatinine, electrolytes, bone age estimation should be done in all cases.Hormonal evaluation depending on clinical features if BMI more than 95th centile: 24 hour urinary free cortisol which is increased in patients with Cushing syndrome. In doubtful cases confirmation is done by dexamethasone suppression test. Growth hormone assay-may be suppressed or fails to increase after provocation tests. Free T4 and TSH to identify and treat

Other investigations are directed towards identification and treatment of complications.

Complications of obesity-The prevalence of type-2 diabetes is increasing throughout the world wherever childhood obesity is becoming more prevalent. Elevated blood pressure is seen in obese children with the greatest elevation seen in children with predominant abdominal adipose distribution. Respiratory diseases like asthma and obstructive sleep apnea are seen in children with obesity. Increased fat mass causes difficulty in respiration and also fat deposition occurs around airways causing obstructive symptoms. Obesity is prevalent in 50 to 80% of children with Blount's disease and slipped capital femoral epiphysis. Degenerative joint diseases are common in obese individuals. Proteinuria is a recognized

reduction is shown to decrease liver enzymes and normalize the size of liver. Cholelithiasis is common especially in adolescent females. The risk is fourfold compared to non obese adolescent females. Laxity of lower esophageal sphincter and hiatal hernias can contribute to the development of gastro esophageal reflux in obese individuals. Obese females can develop benign premature adrenarche which in turn leads to ovarian hyperandrogenism causing oligomenorrhea, ameno-rrhea and infertility. They also suffer from body image distortion, low self esteem, depression and anxiety and may face discrimination in social life and work place.

Management-Diet control and exercises-Goal is to decrease energy intake and increase energy expenditure. In older children reduction in body

weight up to 10% is achievable whereas in younger children preventing further weight gain would suffice. Dietary calories and fat content are reduced and dietary fiber is increased. If children are not losing 0.23 kilograms per week, the calorie intake are decreased further on the other hand, if they lose more than 0.45 kilograms per week, the calorie intake is increased. The current version of traffic light diet classifies food guide pyramid according to fat content and simple sugar content. In general, foods that contain between 0 and 1.90 grams of fat per serving are green (go) foods. Those that contain between 2 and 4.9 grams of fat per serving are called yellow (caution) foods and those with 5 gram or more fat per serving are known as red (stop) foods. The focus is on increasing the intake of green foods particularly those in the fruit, vegetable and dairy groups of the food guide pyramid. Regular aerobic exercises, fast walking, jogging, swimming, cycling are encouraged. Sedentary activities like television watching and computer games are restricted to not more than 2 hours a day. It is unclear what amounts and intensities of exercise may be used safely and effectively to treat childhood obesity. Thirty minutes of moderate intensity or greater exercise for six days in a week in a graded fashion can be attempted.

Medications-There are numerous classes of medications available to treat Obesity. However only a limited number of drugs are safe to use and recommended for children. Anorexians are not to be used in children. Serotonergic drugs and noradrenergic drugs produce lot of side effects and are not well tolerated by children. Neuropeptide agonists, antagonists and recombinant leptons are under trial. Amino acids like phenylalanine, tyrosine, and trytophan are known to reduce hunger. Rimonabant (SR141716) is a neurokinin-3 antagonist and selective cannabinoid (CB1) receptor antagonist currently being researched and developed. The chemical name is N-piperino-5-(4-chlorophenyl)-1-(2,4-dichlorophenyl)-4-methylpyrazole-3-carboxamide.[8] It acts by selectively blocking the cannabinoid-1 receptors with resultant central and metabolic peripheral effects, thereby decreasing food intake.

The National Health and Medical Research Council (NHMRC) guidelines[9] suggest that use of both sibutramine and orlistat in obese adolescents with complications should take place only in a specialist centre, and only when there is a reasonable expectation of benefit over risk. Metformin

Table- 2,Drugs used in obesity

Drug	Class	Adverse Effects
Sibutramine	Multiple amine synaptic reuptake inhibitor	Hypotension, dry mouth, headache, insomnia, nervousness, depression, edema, palpitations& mydriasis
Orlistat	Intestinal lipase inhibitor	Fecal incontinence, oily spotting, flatus with discharge & impaired vitamin absorption
Octreotide	Synthetic somatostatin analogue	Diarrhea, abdominal cramps, nausea & bloating
Metformin	Biguanide	Nausea, flatulence, diarrhea, vitamin B12 deficiency, metabolic acidosis.

has a potential role in therapy in these adolescents, and that metformin therapy should be considered in obese adolescents with significant hyperinsulinaemia and a family history of diabetes.

Surgical-Bariatric surgery includes jejunoileal bypass, biliopancreatic diversion with duodenal switch modification, endoscopically placed bariatric intragastric balloon, gastroplasty, gastric banding and roux-en-y gastric bypass. At present, surgical treatment is reserved for only those children who are in imminent life threatening danger of succumbing to their obesity due to deteriorating cardiac or pulmonary status.

Behavioral therapy-Parental consent and participation is an essential prerequisite in implementing behavioral therapy.Target children and their parents are motivated to participate and are seen separately in weekly sessions for 6 to 8 weeks with each session lasting for at least an hour. During these sessions, they are asked to record their daily eating and physical activities in their habit books. Families are taught to use two positive reinforcement techniques - praise and point system, to help their children learn new behavior and to maintain them. Opportunities for praise are standardized by having each parent and child meet in evenings to review daily eating and physical activities. Parents are told to praise specific behavior change and to be consistent in using praise. They can also use point based reward system for the same purpose. Families and children are provided information about the traffic light diet, food guide pyramid and healthy eating and are encouraged to choose healthy foods based on individual and familial preferences from lists of commonly available food. Another important component in behavior therapy is stimulus control or modification of environment. Families should reduce access to high fat low nutrient dense foods, shop differently and cook healthier foods. Stimulus control for physical activity involves increasing access to physical activity and decreasing access to behaviors that prompt inactivity such as television watching or playing videogames. Finally methods to deal with peer pressure and to engage friends in making changes to their eating and activity behavior are discussed.

Conclusion-Obesity may be endogenous (genetic or endocrine) or exogenous and may be associated with complications in children. Primary goal of treatment is healthy eating and inculcating good habits of physical activity. Treatment of overweight or obesity should begin early and involve the family.

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Malaria

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Malaria was linked with poisonous vapours of swamps or stagnant water on the ground since time immemorial. This probable relationship was so firmly established that it gave the two most frequently used names to the disease *mal'aria*, later shortened to one word *malaria*, and *paludisme*. The term malaria (from the Italian mala "bad" and aria "air") was used by the Italians to describe the cause of intermittent fevers associated with exposure to marsh air or miasma. The word was introduced to English by Horace Walpole, who wrote in 1740 about a "horrid thing called mal'aria, that comes to Rome every summer and kills one." The term malaria, without the apostrophe, evolved into the name of the disease only in the 20th century.

Malaria in India-Malaria has been a problem in India for centuries. Details of this disease can be found even in the ancient Indian medical literature like the 'Charaka Samhita'. In the 30's there was no aspect of life in the country that was not affected by malaria. The economic loss due to the loss of man-days due to malaria was estimated to be at Rs. 10,000 million per year in 1935. To combat this menace, the Govt. of India launched the National Malaria Control

Programme in April 1953. The programme proved highly successful and within five years the incidence dropped to 2 million. Encouraged by this, the programme was changed to a more ambitious National Malaria Eradication Programme in 1958.By 1961 the incidence dropped to a mere 50,00 cases a year. But since then the programme suffered repeated set-backs due to technical, operational and administrative reasons and the cases started rising again. Malaria has now staged a dramatic comeback in India after its near eradication in the early and mid sixties.Later in the 1960s and 1970s malaria resurgence was the result of technical, financial and operational problems. In the late 1960s malaria cases in urban areas started to multiply, and upsurge of malaria was widespread. As a result in 1976, 6.45 million cases were recorded by the National Malaria Eradication Programme (NMEP), highest since resurgence. The implementation of urban malaria scheme (UMS) in 1971-72 and the modified plan of operation (MPO) in 1977 improved the malaria situation for 5-6 years. Malaria cases were reduced to about 2 million. The impact was mainly on vivax malaria. Easy availability of drugs under the MPO prevented deaths due to malaria and reduced morbidity, a peculiar feature of

malaria during the resurgence. The Plasmodium falciparum containment programme (PfCP) launched in 1977 to contain the spread of falciparum malaria reduced falciparum malaria in the areas where the containment programme was operated but its general spread could not be containedMalaria control has become a complex enterprise, and its management requires decentralization and approaches based on local transmission involving multi-sectoral action and community participation.

Causitive agent- Malaria is caused by the parasites called Plasmodia.Phylum: Protozoa Subphylum: Apicomplexa; Class: Sporozoa Subclass: Coccidia

Types of Plasmodium malaria - *P. malariae, P. vivax, P. falciparum* and *P. ovale* are the most important types affecting man. The other species are primarily parasites of monkeys or apes, affecting man only rarely and not seriously.While the benign malaria parasites (*P. vivax, P. malariae, P. ovale*) seem to have evolved from primate malaria, the malignant malaria (*P. falciparum*) appears to have evolved only 10000 years ago from avian malaria species.3 subtypes of *P. vivax* have been reported: Two variants of the circumsporozoite protein of *P. vivax* (VK210 and VK247) have been identified and recently, a

putative third CS variant of *P. vivax*, referred to as causing *P. vivax*-like malaria has been reported. *P. vivax* is the commonest cause of human malaria in Central America, North Africa, southern and western Asia. *P. falciparum* is the predominant species in Africa, New Guinea and Haiti. Both *P. vivax* and *P. falciparum* are found in South America, other parts of Asia and Oceania. *P. falciparum* is the cause for all mortality and most of the morbidity due to malaria. *P. ovale* is restricted to

When a mosquito bites an infected individual, it sucks the gametocytes, the sexual forms of the parasite, along with blood. These gametocytes continue the sexual phase of the cycle and the sporozoites fill the salivary glands of the infested mosquito. When this female mosquito bites the man for a blood meal, which it needs to nourish its eggs, it inoculates the sporozoites into human blood stream, thus spreading the infection. The female anopheles mosquito bites man between 5 PM and 7 AM,

- **Blood transfusion (Transfusion malaria)-** This is fairly common in endemic areas. Following an attack of malaria, the donor may remain infective for years (1-3 years in *P. falciparum*, 3-4 years in *P. vivax*, and 15-50 years in *P. malariae*.) Most infections occur in cases of transfusion of blood stored for less than 5 days and it is rare in transfusions of blood stored for more than 2 weeks. Frozen plasma is not known to transmit malaria.

Comparison of malarial parasites

	P. falciparum	P. vivax	**P. ovale**	**P. malariae**
Tissue schizogony	8 - 25 days	8 - 27 days	9 - 17 days	15 - 30 days
Erythrocytic phase	48 hours	48 hours	48 hours	72 hours
Red cells affected	All	Reticulocytes	Reticulocytes	Mature RBC's
Merozoites per schizont	8 - 32	12 - 24	4 - 16	6 - 12
Relapse from persistent liver forms	No	Yes	Yes	No, but blood forms can persist up to 30 years
Drug resistance	Yes	Yes	No	No

western Africa. *P. malariae* is probably more wide spread than as made out to be in only a few pockets here and there.

Principal mode of spread of malaria is by the bites of female anopheles mosquito- The female anopheles mosquito is the vector for human malaria. Some 60 species of this mosquito have been identified as vectors for malaria, and their distribution varies from country to country.

with maximum intensity at midnight.

Other modes of transmission- Rarely malaria can spread by the inoculation of blood from an infected person to a healthy person. In this type of malaria, asexual forms are directly inoculated into the blood and pre-erythrocytic development of the parasite in the liver does not occur. Therefore, this type of malaria has a shorter incubation period and relapses do not occur.
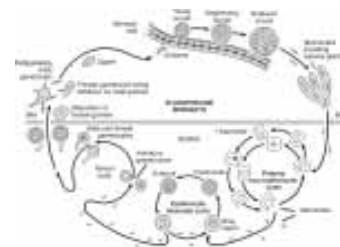

Diagram depicting the life cycle of human malaria

(Asexual phase in human body and sexual phase in the mosquito)

- **Mother to the growing fetus (Congenital malaria)** - Intrauterine transmission of infection from mother to child is well documented. Placenta becomes heavily infested with the parasites. Congenital malaria is more common in first pregnancy, among non - immune populations.

- **Needle stick injury-** Accidental transmission can occur among drug addicts who share syringes and needles. (Therapeutic inoculation of malarial parasites, so as to induce fever, was a mode of treatment for neurosyphilis!)

Clinical features - The characteristic, text-book picture of malarial illness is not commonly seen. It includes three stages viz. Cold stage, Hot stage and Sweating stage. The febrile episode starts with shaking chills, usually at mid-day between 11 a.m. to 12 noon, and this lasts from 15 minutes to 1 hour (the cold stage), followed by high grade fever, even reaching above 106^0 F, which lasts 2 to 6 hours (the hot stage). This is followed by profuse sweating and the fever gradually subsides over 2-4 hours. These typical features are seen after the infection gets established for about a week. The febrile paroxysms are usually accompanied by head aches, vomiting, delirium, anxiety and restlessness. These are as a rule transient and disappear with normalization of the temperature.
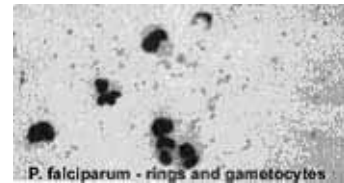
Diagnosis

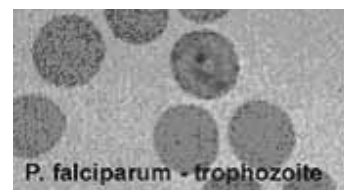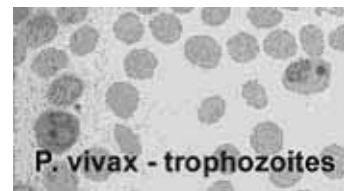**Peripheral smear study for malarial parasites -The MP test-**Peripheral smear study for malarial parasites is the gold standard in diagnosing malarial infection. It involves collection of a blood smear, its staining with Romanowsky stains and examination of the Red Blood Cells for intracellular malarial parasites. The smear can be prepared from blood collected by venipuncture, finger prick and ear lobe stab. In obstetric practice, cord blood and placental impression smears can be used. In fatal cases, post-mortem smears of cerebral grey matter obtained by needle necropsy through the foramen magnum, superior orbital fissure, ethmoid sinus via the nose or through fontanelle in young children can be used.Sometimes no parasites can be found in peripheral blood smears from patients with malaria, even in severe infections. This may be explained by partial antimalarial treatment or by sequestration of parasitised cells in deep vascular beds. In these cases, parasites, or malarial pigment may be found in the bone marrow aspirates. Presence of malarial pigment in circulating neutrophils and monocytes may also suggest the possibility of malaria.Thick and thin smears are usually prepared. Thick smears are used to identify the parasites and thin smears for identifying the species.
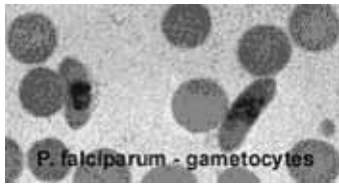
Staining methods- Giemsa, Lieshman's , Jaswanth Singh Battacharya. An experienced technician can detect as few as 5 parasites/µl in a thick film and 200/µl in a thin film.

Thick Film Examination

P. vivax - trophozoites, and gametocytes

P. falciparum - rings and gametocytes

Thin Film Examination

P. vivax - trophozoites

P. vivax - schizont

P. vivax - gametocyte

P. falciparum - trophozoite

P. falciparum - gametocytes

Other Tests

- Quantitative Buffy Coat (QBC) test
- RDT -Para Sight F test
- RDT -OptiMal Assay
- RDT-The immuno chromatographic test (ICT Malaria P. f. test)
- Polymerase Chain Reaction
- Detection of antibodies by Radio immuno assay, immunofluorescence or enzyme immuno assay

Differential diagnosis

- Malaria can be offered as a differential diagnosis for a big list of diseases.
- **General:** All other causes of fever, migraine, sinusitis, tension headache etc.
- **Respiratory system :** Pharyngitis, bronchitis, pneumonia, broncho-pneumonia, pleurisy.
- **Cardiovascular :** Acute myocardial infarction, cardiogenic shock, left ventricular failure, pericarditis
- **Abdominal:** Hepatitis, liver abscess, splenitis, splenic abscess, other causes of splenomegaly, subdiaphragmatic abscess, acute abdomen, cholecystitis, cholangitis, gastroenteritis, amebiasis, appendicitis, etc.

- **Central nervous system:** Acute encephalitis, meningitis, intra-cranial space occupying lesions, stroke, metabolic encephalopathy etc.
- **Psychiatry:** Acute confusional states, acute psychosis, mood disorders
- **Renal:** Acute nephritis, nephrotic syndrome, acute renal failure
- **Haematological:** All other causes of anemia; blood dyscrasias, hemoglobinopathies, hemolytic anemias, intra vascular hemolysis, bleeding diathesis, DIC etc.

Complications of malaria

Complications of *P. vivax, P. ovale* and Quartan malaria- *Plasmodium vivax* and *P. ovale* infections are generally benign and complications leading to significant morbidity and mortality are uncommon. The clinical symptoms of fever, headache, nausea and vomiting may be incapacitating, particularly for those who are non-immune and suffering the infection for the first time.

- **Rupture of spleen-** Malaria is an important cause for spontaneous rupture of spleen. It is more common in vivax malaria than falciparum malaria and tends to occur in up to 0.7% of the patients.Rupture occurs in acute, rapid, hyperplastic enlargement of spleen. It is rare in chronic malaria, despite massive enlargement. Rapid enlargement results in increased capsular tension and increased parenchymal

friability. Marked splenomegaly can occur even in low-grade parasitemia (50/ml) and it may persist for weeks or months after effective and complete treatment.Patients present with abdominal pain, fever, tachycardia, prostration and rapidly developing anemia and hypotension. Some of these manifestations are seen in malaria itself and therefore splenic rupture can be easily missed. A degree of suspicion is required to differentiate the two conditions. Leukocytosis, severe anemia and hypotension are more in favour of splenic rupture. Ultra sound evaluation of abdomen and paracentesis of the abdomen can confirm the diagnosis. Treatment includes replacement of fluid and blood, laparotomy and splenectomy. Splenic rupture carries a high mortality of about 80% and this is partly attributed to lack of awareness and missed diagnosis.

- **Hepatic dysfunction -** Hepatomegaly and non-specific hepatitis, with or without jaundice can occur in vivax malaria. Fever, jaundice, tender hepatomegaly, mild elevation in the levels of hepatic enzymes and bilirubin are observed. Liver biopsy in such cases has demonstrated brown malarial pigments in Kupffer's cells, small to moderate sized granulo-

matous lesions with mononuclear infiltration and hepatocyte necrosis. Liver function returns to normal shortly after antimalarial treatment.

- **Thrombocytopenia-** Decrease in platelet counts can occur in vivax malaria, however, it is usually mild and bleeding does not occur.

- **Severe anemia-** *P. vivax* can cause severe anemia, particularly when it is chronic and recurrent. Very rarely this can be life threatening or even fatal.

- **C.N.S. manifestations-** Changes in behaviour and level of sensorium can occur in *P. vivax* malaria. Frank cerebral malaria does not occur and if present, it should prompt a search for other causes, most commonly an associated *P. falciparum* infection. Some of the C.N.S. manifestations could be caused by chloroquine also.

- **Quartan malarial nephropathy -** In areas where *P. malariae* is prevalent, there is epidemiological evidence to link *P. malariae* infection to immune-complex mediated glomeru-lonephritis, leading to nephrotic syndrome. Because only a few of the infected develop nephrosis, it is possible that other factors are also involved in the pathogenesis of this entity. Histologically there is progressive focal and s e g m e n t a l glomerulosclerosis with fibrillary splitting or flaking of the capillary basement membrane, producing characteristic lacunae. Dense subendothelial deposits are seen on electron microscopy and immunofluorescence reveals deposits of compliments and immunoglobulins. In about 25% of patients *P. malariae* antigen may also be seen. Patients usually present by the age of 15 years with typical features of nephrotic syndrome. Treatment with antimalarial drugs, corticosteroids or cytotoxic agents may not

Severe manifestations and complications of *P. falciparum* malaria

In a patient with falciparum malaria in whom other diseases have been excluded, the presence of one or more of the following manifestations is sufficient for a diagnosis of severe falciparum malaria.

Cerebral malaria C.N.S. dysfunction in falciparum malaria could be multi factorial. Therefore, to differentiate from various causes of transient cerebral dysfunction, a strict definition of cerebral malaria has been developed.	**For a diagnosis of cerebral malaria, the following criteria should be met: Deep, unarousable coma:** Motor response to noxious stimuli is non-localising or absent. **Exclusion of other encephalopathies:** Coma should persist for more than 30 minutes after a generalized convulsion to exclude transient post-ictal coma. Hypoglycemia, meningoencephalitis, eclampsia, intoxications, head injuries, cerebrovascular accidents and metabolic disorders should be excluded as the cause of coma. **Confirmation of *P. falciparum* infection:** Asexual forms of *P. falciparum* must be demonstrated in peripheral blood or bone marrow smear during life, or in a brain smear after death.
Severe anemia	Hematocrit less than 15% (hemoglobin less than 3.1 mmol/l or 5g/dl).
Metabolic (Lactic) Acidosis	Metabolic acidosis is defined by an arterial blood pH of <7.35 with a plasma bicarbonate concentration of <22 mmol/L; hyperlactatemia is defined as a plasma lactate concentration of 2-5 mmol/L and lactic acidosis is characterized by a pH <7.25 and a plasma lactate >5 mmol/L.

Jaundice	Serum bilirubin of more than 50m mol/l (3 mg/dl).
Renal failure	Urine output of less than 400 ml in 24 hours or <12ml/kg per 24 hours in children and a serum creatinine of more than 265 m mol/l (> 3.0 mg/dl), failing to improve after rehydration.
Pulmonary edema or	Breathlessness, bilateral crackles, and other features of pulmonary oedema.
ARDS	
Hypoglycemia	Blood glucose concentration of less than 2.2 mmol/l (less than 40 mg/dl).
Hypotension and shock	Systolic blood pressure <50 mmHg in children 1-5 years or <80 mm Hg in adults; core-skin temperature difference >10^0C
Bleeding and clotting disturbances	Significant bleeding and haemorrhage from the gums, nose, gastrointestinal tract, retinal haemorrhages and/or evidence of disseminated intravascular coagulation.
Hyperpyrexia	Rectal temperature above 40^0C
Fluid, electrolyte or acid-base disturbances	Requiring intravenous fluid therapy; arterial pH <7.25 or plasma bicarbonate <15 mmol/L, venous lactate >6mmol/L
Haemoglobinuria	Macroscopic black, brown or red urine; not associated with effects of oxidant drugs or enzyme defects (like G6PD deficiency)
Hyperparasitemia	Density of asexual forms of *P. falciparum* in the peripheral smear exceeding 5% of erythrocytes (more than 250,000 parasites per m l at normal red cell counts)
Complicating or associated infections	Aspiration bronchopneumonia, septicemia, urinary tract infection etc.
Vomiting of oral drugs	Patients with persistent vomiting may have to be admitted for parenteral therapy.
Impaired Consciousness	Various levels of impairment may indicate severe infection although not falling into the definition of cerebral malaria. These patients are generally arousable.
Extreme weakness Convulsions	Prostration, dehydration, needs support More than two generalized seizures in 24 hours with regaining of consciousness.
Other indicators of poor prognosis	Leukocyte count >12,000/cumm; high C.S.F. lactate and low C.S.F. glucose; low antithrombin III levels; peripheral schizontemia

Treatment
Aims of Treatment

Aims	Causation	Therapy	Drugs
To alleviate symptoms	Symptoms are caused by blood forms of the parasite	Blood schizonticidal drugs	Chloroquine, quinine, pyrimethamine/sulphadoxine, artemisinin
To prevent relapses	Relapses are due to hypnozoites of *P. vivax/P. ovale*	Tissue schizonticidal drugs	Primaquine
To prevent spread	Spread is through the gametocytes	Gametocytocidal drugs	Primaquine for *P. falciparum*, Chloroquine for all other

Principles of Treatment- Treatment of malaria depends on the following factors:
- Type of infection.
- Severity of infection.
- Status of the host.
- Associated conditions/diseases.

Type of infection- Treatment obviously depends on the type of infection. Patients with *P. falciparum* malaria should be evaluated thoroughly in view of potential seriousness of the disease and possibility of resistance to anti malarial drugs. *P. vivax:* Only Chloroquine 25 mg/kg + Primaquine for 14 days. *P. falciparum:* Treat depending on severity & sensitivity. Primaquine as gametocytocidal is a must to prevent spread. **Mixed infections:** Blood schizonticides as for *P. falciparum* and Primaquine as for *P. vivax*. All cases of *P. vivax* malaria and uncomplicated cases of *P. falciparum* malaria are treated with oral drugs. Chloroquine is the ONLY drug used for *P. vivax* malaria, because resistance to chloroquine in *P. vivax* malaria is almost unknown (only sporadic reports). Most cases of *P. falciparum* malaria can also be treated with chloroquine alone, however, in areas with known resistance to chloroquine, it is safer to combine chloroquine with another oral antimalarial like pyrimethamine/ sulphadoxine. Primaquine should be used in both types of malaria for radical treatment.

Dose of commonly used antimalarial drugs

Age in years	Dose of Chloroquine (as base) (Each 250 mg tablet contains 150 mg base and each 5 ml of suspension contains 50 mg base)				Dose of Primaquine		Dose of Pyri/Sulpha (Of 25+500 mg tablet)
	1st dose*	2nd dose	3rd dose	4th dose	*P. vivax*/mixed (for 14 days)**	*P. falciparum* Single dose	
0-1	75 mg	37.5 mg	37.5 mg	37.5 mg	Nil	Nil	1/4 tablet
1-5	150 mg	75 mg	75 mg	75 mg	2.5 mg	7.5 mg	1/2 tablet
5-9	300 mg	150 mg	150 mg	150 mg	5 mg	15 mg	1 tablet
9-14	450 mg	225 mg	225 mg	225 mg	10 mg	30 mg	2 tablets
>14	600 mg	300 mg	300 mg	300 mg	15 mg	45 mg	3 tablets

*1st dose of chloroquine should always be larger to obtain sufficient blood levels, in view of large volume of distribution.

** The National Malaria Eradication Programme in India recommends a 5 day course of primaquine instead of 14 days.

Dose spacing for chloroquine	1st dose	2nd dose	3rd dose	4th dose
If the patient comes in the morning and treatment can be started by mid-day	Stat.	After 6 hours	After 24 hours	After 48 hours
If the patient comes in the afternoon and treatment is started by evening	Stat	After 12 hours	After 24 hours	After 36 hours
If the patient is coming from a far off place and /or if the MP test report is available only next day	Stat (as presumptive)	2nd and 3rd doses together after 24 hours		After 48 hours

Parenteral Chloroquine: Parenteral chloroquine may be needed in patients with complicated, yet drug sensitive, *P. falciparum* malaria and in case of persistent vomiting.

Intravenous infusion	10 mg / kg (max.600mg) in isotonic fluid, over 8 hours; followed by 15 mg / kg (max.900mg) over 24 hours.
Intramuscular or subcutaneous injections	3.5 mg of base/ kg (max.200 mg) every 6 hours or 2.5 mg of base/ kg (max.150mg) every 4 hours.(Intramuscular injection can cause fatal hypotension, especially in children)

Treatment of complicated/ chloroquine resistant *P. falciparum* malaria
It is safer to treat cases of severe *P. falciparum* malaria as chloroquine resistant, unless one is very certain about the sensitivity. It is better to use two drugs, one rapid acting and one slower acting. Severe malaria should always be treated with parenteral antimalarials to ensure adequate treatment.
Quinine

Intravenous	**In intensive care unit:** 7mg of salt/kg over 30 minutes., followed immediately by 10mg/kg diluted in 10ml/kg isotonic fluid over 4 hours; after 4 hour interval, 10mg/ kg over 4 hours, repeated every 8-12 hours until patient can swallow. **OR** 20mg of salt/kg diluted in 10 ml/kg isotonic fluid, infused over 4 hrs; then 10 mg of salt / kg over 4 hrs, every 8-12 hrs until patient can swallow.**Children:** 24 mg of salt/kg diluted in 10 ml/kg isotonic fluid, infused over 4 hrs; then 12mg of salt/kg over 4 hrs, every 8-12 hrs until patient can swallow.
Intramuscular	20mg of salt/kg diluted to 60 mg/ml by deep i.m. injection, (divided into two sites); then 10mg of salt/kg every 8 hours.
Oral	Adults: 600mg of salt 3 times a day for 7 days (max. of 1800mg/day) Children: Approximately 10mg/kg 3 times a day for 7 days.
In areas where resistance to quinine is known or suspected, add single dose of pyrimethamine/ sulphadoxine OR Tetracycline or Doxycycline for 7 days (for non-pregnant adults only)	

Artemisinin derivatives

Preparation	Dose and administration
Artemether:(Availability: 80 mg/ml Inj. and 40 mg cap.)	**IM:** 3.2mg/kg as loading dose, followed by 1.6mg/kg daily, until patient is able to swallow or for 5 days. (Maximum dose: 480 mg in adults and 9.6mg/kg in children.)**Oral:** 160mg in two doses on the first day, then 80 mg/day for total 5 days
Arteether:(Availability: 150mg/2 ml injection)	Adults: 150mg IM once daily for 3 consecutive days. Children: 3 mg/kg once daily for 3 consecutive days.
Artesunate:(Availability: 60mg powder with 1 ml of 5% sodium bicarbonate ampoule for injection and 50 mg tablet)	**Injection:** The powder should be reconstituted in 1 ml of 5% sodium bicarbonate and then further diluted with isotonic saline or 5% dextrose (to a total of 3 ml for im and 6 ml for iv use).**Dose:** 2.4mg/kg on the first day (additional 1.2 mg/kg after 4 hours in case of severe falciparum malaria), followed by 1.2 mg/kg daily until patient is able to swallow or for a maximum of 7 days.**Oral:** 100 mg on the first day, followed by 50 mg/ day for 7 days.

Other drugs

Drug	Dose
Mefloquine	15-25 mg/kg (max. of 1500 mg), given as two doses, 6-8 hrs apart
Tetracycline	250 mg 4 times a day for 7 days (for patients > 8 years and non-pregnant)
Doxycycline	100 mg twice a day for 7 days (for patients > 8 years and non-pregnant)

Combination therapy-Combination therapy with antimalarial drugs is the simultaneous use of two or more blood schizontocidal drugs with independent modes of action and different biochemical targets in the parasite. The concept of combination therapy is based on the synergistic or additive potential of two or more drugs, to improve therapeutic efficacy and also delay the development of resistance to the individual components of the combination.Artemisinin based combinations are known to improve cure rates, reduce the development of resistance and they might decrease transmission of drug-resistant parasites. The total effect of artemisinin combinations (which can be simultaneous or sequential) is to reduce the chance of parasite recrudescence, reduce the within-patient selection pressure, and prevent transmission.

Antimalarial Drug Combinations	
Artemisinin based combinations	
Artesunate + Chloroquine	
Efficacy	Very high chloroquine failure rates (>60%) and sub-optimal efficacy of the combination (?85% cure rate)
Status	Not approved; Not a viable option in areas with pre-existing moderate to high levels of *P. falciparum* resistance to Chloroquine
Artesunate + Amodiaquine	
Efficacy and advantages	Better efficacy than amodiaquine alone (cure rate >90%); Well tolerated
Disadvantages	?Neutropenia; Pharmacokinetic mismatch
Dose	Artesunate 4mg/kg and amodiaquine 10mg base/ kg once a day 3 days
Status	Approved
Artesunate + Mefloquine	
Efficacy and advantages	In use for many years and the first-line treatment in several parts of SE Asia
Disadvantages	Pharmacokinetic mismatch; Mefloquine induced neuropsychiatric effects, cardiotoxic effects, incidents of vomiting in children; but combination with artesunate results in less adverse reactions than the use of mefloquine alone
Dose	Artesunate (4mg/kg once daily) for 3 days + mefloquine (25mg base/kg) as a split dose of 15mg/kg on Day 2 and 10mg/kg on Day 3. (Alternatively 8mg/kg mefloquine daily for three days)
Status	Not approved; Not considered a viable option as first-line therapy in Africa
Artesunate + Sulfadoxine/Pyrimethamine (SP)	
Efficacy and advantages	Well tolerated; Efficacy dependent on the level of pre-existing resistance to SP
Disadvantages	Pharmacokinetic mismatch; adverse effects to SP
Dose	Artesunate 4mg/kg once daily for 3 days and SP single dose of 25mg/kg and 1.25mg/kg respectively
Status	Approved (in areas where SP efficacy is high); Resistance to SP limits the use
Artemether + Lumefantrine *(Coartem,[TM] Riamet[TM])*	
Efficacy and advantages	As effective, and better tolerated, as artesunate plus mefloquine; No serious adverse reactions documented
Disadvantages	?Irreversible hearing impairment
Dose	Artemether 1.5mg/kg and Lumifantrine 9mg/kg at 0, 8, 24, 36, 48 and 60 hours
Status	Approved; Not recommended for use in pregnancy and lactating women

Non-Artemisinin based Combinations	
Sulfadoxine-pyrimethamine based combinations	
Sulfadoxine-pyrimethamine (SP)	
Efficacy and advantages	Single dose; Cheap
Disadvantages	Drug resistance; Serious adverse effects
Dose	Sulfadoxine 25mg/kg and Pyrimethamine 1.25mg/kg as single dose
Status	Not approved; Considered as single drug
SP + Chloroquine	
Advantages	Cheap; Similar pharmacokinetic profiles, with varied modes of action on different biochemical targets in the parasite
Disadvantages	Drug resistance; Serious adverse effects to SP
Dose	Chloroquine 25mg/kg over 3 days; SP single dose as above
Status	Not approved; an be used where resistance to SP is not a problem
SP + Amodiaquine	
Advantages	Similar pharmacokinetic profiles
Disadvantages	Adverse effects of amodiaquine and SP
Dose	Amodiaquine 10mg/kg daily for 3 days; SP single dose as above
Status	Approved (In areas where efficacy of both amodiaquine and SP remain high - countries in West Africa)
SP + Quinine	
Advantages	Effective where resistance to SP is not a problem
Disadvantages	Drug resistance; Serious adverse effects
Dose	Quinine 15mg/kg 12 hourly for 3 days; SP single dose as above
Status	Not approved
SP + Mefloquin *(Fansimef™)*	
Advantages	Fixed dose pill, single dose
Disadvantages	Not an additive or synergistic combination; Each drug has a different pharmacokinetic profile; Expensive; Resistance known
Dose	Mefloquine 15mg/kg and SP as above single dose
Status	Not approved; Not recommended for general use since 1990
Atovaquone + Proguanil (Malarone™)	
Advantages	Synergistic activity; Good safety and tolerability in children and adults

Disadvantages	High cost; Restricted availability; Contra-indicated in case of hypersensitivity or renal insufficiency
Dose	Atovaquone 20mg/kg and Proguanil 8mg/kg once daily for 3 days
Status	Approved; Highly efficacious against *P. falciparum*, including strains that are resistant to chloroquine and mefloquine, with cure rates of 94-100%

Chlorproguanil + Dapsone (*LapDap^TM*)

Advantages	Well tolerated; Efficacious
Disadvantages	Dapsone induced methaemoglobinaemia and haemolysis in G6PD deficiency; Potential cross-resistance with SP
Dose	Chlorproguanil 2mg/kg and Dapsone 2·5mg/kg once daily for 3 days
Status	Approved

Quinine based Combinations

Quinine + Tetracycline

Advantages	Efficacious
Disadvantages	7-day course, multiple doses daily; Cinchonism; Tetracyclines contraindicated in children and pregnant women; Emergence of resistance
Dose	Quinine 10mg/kg 8 hourly and Tetracycline 4mg/kg 6 hourly for 7 days
Status	Not approved; Difficult to recommend as a first-line treatment for uncomplicated malaria

Quinine + Clindamycin

Advantages	Good efficacy; Safe in children and pregnant women; Lesser risk of resistance
Disadvantages	Cinchonism
Dose	Quinine 15mg/kg 12 hourly and Clindamycin 5mg/kg 12 hourly for 3 days
Status	Not approved

New Combinations

Piperaquine + Dihydroartemisinin + Trimethoprim *(Artecom^TM)* and Artecom^TM plus Primaquine *(CV8^TM)* (CV = China-Viet Nam)

Advantages	Efficacy consistently above 93%
Disadvantages	Animal toxicology studies indicate additive toxicity; No serious adverse events observed in human studies
Status	Trials; May prove to be more affordable

Current clinical practice guidelines for treatment of Malaria by WHO-For uncomplicated plasmodium falciparum malaria any one ACT of Table 1 is preferable. In South-east Asia artesunate + melfoquine (AS + MQ) or artemether + Lumefantrine (AL) should be preferred, wheress artesunate + sulfadoxine-pyremethamine (AS+SP) and artesunate + amodiaquine (AS + AQ) to be prefedded in Africa in view of wide spread resistance of SP in South east Asia and intolerance of MQ among African People. In the event of failure of one ACT regimen another ACT regimen should be used. The alternative second line regimens were be used. The alternative second line regimens were artesunate (2mg/lg/day) for 7 days + tetracyckine (4 mg/kg/hr) for 6 hourly or Doxycycline (3.5 mg/kg/day) or clindamycin (10 mg/kg) twice daily for 7 days or quinine (10 mg/kg) 8 hourly for 7 days + tetracycline/ doxycycline or clindamycin in the previous doses for 7 days to be used in order of preference.In severe P. falciparum malaria artesunate (2.4 mg/kg) in 0 hr, 12 hrs and then daily for 7 days with tetracycline / doxycycline / clindamycin should be preferred over quinine + tetracycline / doxycycline / clindamycin.In vivex malaria, chloroquine with primaquine is still effective but ACT can be used except AS + SP. Multidrug resistant malaria parasite has forced the use of combinations antimalarial regimen. Artemisinin based combinations are preferred until better combination regimens are found. There is no place monotherapy in malaria.

Christianity's share in the development of medical science

As long as the cruel persecution the Church lasted throughout the Roman Empire, it was impossible for Christians to take direct part in the development of medical science. But provision had been made for medical aid within the community, because the priest, like the rabbi of small Jewish communities in the late Middle Ages, was also a physician. This is clear from the story of the two brothers, Sts. Cosmas and Damian, who studied medicine in Syria and were martyred under Diocletian. The exercise of practical charity under the direction of deacons of the churches gave rise to systematic nursing and hospitals. In recent times it has, indeed, been alleged that the existence of hospitals among the Buddhists, even in the third century before Christ, and their existence in ancient Mexico at the time of its discovery is demonstrable, and that hospitals had their origin in general philanthropy; but nobody denies that the nursing of the sick, especially during epidemics, had never before been so widespread, so well organized, so self-sacrificing as in the early Christian communities. Christianity tended the sick and devised and executed extensive schemes for the care of deserted children (foundling, orphans), of the feeble and infirm, of those out of work and of pilgrims. The era of persecution ended, we find large alms-houses and hospitals like that of St. Basilius in Caesarea (370), those of the Roman Lady Fabiola in Rome and Ostia (400), that of St. Samson adjoining the church of St. Sofia in Constantinople in the sixth century, the foundling asylum of Archbishop Datheus of Milan in 787, and many others. In 1198 Pope Innocent III rebuilt the pilgrims' shelter, which had been founded in 726 by a British king, but had been repeatedly destroyed by fire. He turned it into a refuge for travellers and a hospital, and entrusted it to the Brothers of the Holy Ghost established by Guy de Montpellier. Mention must also be made here of the religious orders of knights and the houses for lepers of later times. The great hospitals of the Arabs in Dschondisapor and Bagdad were built after Christian models. The celebrated ecclesiastical writer Tertullian (born A.D. 160) possessed a wide knowledge of medicine, which, following the custom of his time, he calls a "sister of philosophy". Clement of Alexandria, about the middle of the century, lays down valuable hygienic laws in his "Paedagogus". Lactantius in the fourth century speaks in his work "De Opificio Dei" about the structure of the human body. One of the most learned priests of his time, St. Isidore of Seville (d. 636), treats of medicine in the fourth book of his "Origines S. Etymologiae". St. Benedict of Nursia (480) made it a duty for the sciences, and among them medicine, as aids to the exercise of hospitality.

Multislice CT (MSCT) – a New Tool for Non Invasive Evaluation of Coronary Artery Disease

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Invasive Coronary Angiography (ICA) is the Gold Standard for evaluation of Coronary artery disease (CAD). It has got unprecedented temporal and spatial resolution facilitating accurate evaluation of Coronary tree for evidence of significant coronary artery lesions (>50% stenosis). It has got added advantage, as the therapeutic interventions can be performed at the same sitting.But invasive Coronary Angiography, though relatively free from serious complications, is invasive and requires hospitalization. It is not suitable for patients in whom probability of CAD is low / intermediate. It is also not suitable and can not be used as a screening modality for evaluation of CAD.Though many non invasive alternatives are available like Tread Mill Test (TMT), they lack sensitivity and specificity. Sensitivity of TMT varies from as low as 25% in single vessel disease to 65% to 80% in multi vessel disease[1]. This results in large number of false positive results leading to unnecessary ICA or false negative results leading to false sense of security.All these factors led to search for Non invasive alternatives for evaluation of CAD. Currently available MSCT scanner appears to be ideally suited for evaluation of CAD

Non Invasively. CT scanning technology has improved by leaps and bounds from axial CT scanner, which were capable of taking approximately 1 slice/min in early 80's to latest MS CT scanners which are capable of taking 64 slices per rotation. Rapid progression in soft ware and hardware capabilities of CT scanner has resulted in very fast Gantry rotation –(speed of 0.33 sec/rotation). Acquisition of 64 slices of 0.4 mm isotropic voxels/rotation. i.e. unprecedented 194 slices of 0.4mm isotropic voxels/sec. Technique of performing CT Coronary Angiography by using MS CT is straight forward and is more or less standardized in previous 2-3 years by different studies conducted through out the world. Relatively Junior Radiologists and Technologists with experience in spiral CT can be trained with very little learning curve.

Indications for CT coronary angiogram[2]

- Patients with suspected congenital or acquired coronary anomalies (figure 7).
- Asymptomatic High risk patients or patients with atypical or stable angio who -have Inconclusive TMT; cannot undergo TMT; need

to undergo major Non cardiac surgery; have undergone Coronary artery By-pass Graft and need Evaluation for patency of Grafts.

There is no role of MSCT coronary angiography for evaluation of patients with Myocardial infarction or in whom TMT is strongly positive and probability of CAD is very high. Such patients should directly under go ICA.

Patient Preparation

- Patient should be on empty stomach. Atleast 4 hrs fasting is necessary. It is advisable to avoid Coffee which will increase the Heart beat.
- Patient should be given Beta blocker to reduce the pulse rate to about 60/ min.
- 100 mgs of Atenolol to given if the pulse rate is > 65 /min.
- 50 mgs Atenolol to be given if the pulse rate is >50 - < 65/min
- BP, Heart rate and ECG should be monitored before the procedure.
- If necessary IV Metaprolol (5 to 30 mgs) can be given just before the start of the procedure to achieve a target Heart rate of <65/min.

- Some centers use sublingual Nitroglycerine 0.4 mgs, 1 min before the procedure[3].

- Steady regular pulse rate of < 65 /min is pre requisite for good quality diagnostic images free from motion artifacts. Pulse rate-is less of a problem in 64 slice scanners and usually no patient with regular pulse rate is excluded from the study, even if the pulse rate is on higher side. (upto 80 / min)

- Breath Hold-Patient should be adequately trained for breath hold during the scan. This should not be a problem is 64 slice scanner which is capable of imaging the entire coronary tree in less than 12 seconds

It is evident from above discussion that patients with irregular Heart beats like Multiple extra systole, Atrial fibrillation and Patient who are un co-operative and unable to hold the breath for about 15 seconds can not be adequately evaluated by MSCT.

Protocol-Initial unenhanced ECG gated scans should be performed for Calcium scoring from the level of the Carina inferiorly upto the level of Diaphragm. This un enhanced scans serves three purposes:

- To evaluate Calcium in the coronary tree

- One can ascertain if the patient is adequately trained to hold breath during the data acquisition.

- To monitor the pulse rate during the data acquisition.

Calcium scoring can be rated as under:

0 - No calcium is seen.
1 - Calcium present – no image impairment.
2 – Calcium covering < 50% of the lumen
3 – Calcium covering >50% of the lumen

In practice total Calcium score can be calculated by the soft ware provided by the Vendor. Using this soft ware total calcium score in Agaston units can be calculated both for individual vessels and total calcium in all vessels[4]

Calcium score is calcified as –

Mild 0 – 100 Agaston units.

Moderate101 – 400 Agaston units.

Severe > 400 Agaston units.

Total Calcium score, Heart rate and body Mass Index (Kg /meter square) are very important patient characteristics which have direct bearing on the ultimate quality of the images obtained and are summarized in the Table 1 to 3. Sensitivity, Specificity, Positive Predictive value (PPV) and Negative predictive values (NPV) are calculated for MSCT Coronary angiography, taking ICA as Gold Standard[3]

It is evident that patients with Calcium score of over 400 units, obese patients (body mass >30 kg/msq.) and patients with heart rate over 70/min remain a challenge to diagnostic capabilities of MSCT. Of the three factors only pulse rate can be modified by Beta blockers.

CT Coronary Angiogram-After plain study for Calcium score. Contrast study is done from the level of Carina upto the level of Diaphragm. 100 ml of Non ionic contrast media is injected intravenously at the rate of 5 ml /sec. followed by 40 cc of saline chase by Dual Head Pressure Injector. Bolus tracking is done with Trigger at the ascending aorta. Scan start is automatically initiated after reaching the threshold level of density in aorta. (140 HU)[5]. Ideally at the start of the scan, contrast should have washed out from right ventricle, and most of the contrast should be in left ventricle. Typically following scan protocols are used for scanning. X-ray tube potential - 120 KV, Tube current - 700 MAS, slice collimation - 64 + 0.6 mm. Table feed 9.2 mm /rotation and pitch of 0.24. Images are acquired with retrospective ECG gating [5].Image are reconstructed in different phases of cardiac cycle at 10% interval . Cardiac cycle in which the images are sharpest are used for reconstruction. Preview series is done with Right Coronary artery in AV groove. As the RCA is the most moving part in the entire coronary tree. Phase of cardiac cycle when RCA is sharper is selected for reconstruction. Images are reconstructed with 0.75 mm thickness and an increment of 0.5 mm (thin overlapping slices) Medium soft tissue reconst-ruction kernel is used for reconstruction (B30 f) [5]. Once thin axial data set is available, post processing is done to get MPR

(multiplanner reconstruction) with MIP (Maximum intensity projection) and VRT (Volume rendered Technique) in dedicated work station.

Coronary artery is analysed segment wise according to the guidelines of American Heart Association [6]. The coronary artery is segmented as follows: (Fig- 1) [7]

- Right Coronary artery subdivided in Proximal, Middle and Distal segments
- Left Main
- Left anterior descending (LAD), proximal, mid and distal.
- Left circumflex arteries (LCX) – proximal and distal
- Diagonal branches of LAD
- OM Branches of LCX
- Posterior Descending Artery (PDA) and Posterior Left Ventricular (PLV) branch are taken as independent segments. Coronary Arteries are analysed for luminal narrowing in all segments upto 1.5 mm diameter.

Each segment is analysed in atleast two planes - one perpendicular and another parallel to the course of the vessel for evidence of narrowing. Analysis is done by both Quantitative and Qualitative methods [3]. Quantitative analysis is done by Digital caliper method [8.] Narrowing of the luminal diameter is calculated by dedicated soft ware provided by the vendor. Qualitatively lesions are classified as under [3]

- 0 –No stenosis
- Grade I –1-25%
- Grade II –26-50%
- Grade III –51 to 75%
- Grade IV –76 to 99 %
- Grade V –Total occlusion.

Practically lesion can be classified as

- Mild - Less than 50% narrowing
- Moderate – 50 to 70%
- Severe – More than 70% narrowing.

Significant coronary artery disease is said to be present if there is stenosis of more than 50% on any artery. If the initial calcium scoring study shows high calcium score, (more than 1,000 units). It is not advisable to proceed with the MSCT coronary Angiographin. Such patients will be benefited by ICA. (Fig- 2). VRT images show normal Coronary anatomy in relation to Cardiac chambers. (Fig. 3 & 4). Significant narrowing of proximal LAD , LCX and RCA are shown in fig. 5 and 6.

For evaluation of patients who have undergone CABG-120 ml of Contrast is used. Field of view should include origin of Left Internal Mammary Artery (LIMA) from subclavian artery. Tube current has to be adjusted to the maximum permissible limit as dictated by the Tube cooling to allow for increased duration of scanning.

MSCT Coronary Angiography is ideally suited for evaluation of graft patients, in patients who have undergone CABG. Graft vessels are adequately visualised as these vessels are less prone to motion artifacts from moving heart. ICA is not ideal for evaluation of such patients, as selective catheterization of individual graft vessels may be technically difficult and time consuming. Most of the patients are reluctant to undergo ICA, as MSCT offers no invasive alternate to such patients. Graft occlusion, narrowing and anastomatic site are adequately visualised. ICA is indicated for therapeutic interventions of any graft vessel which is suitable for Angioplasty. Calcific plaques is not a problem in visualization of graft vessels. Patency of both venous and arterial grafts and degree of narrowing can be assessed (Fig. 8 to 10).Though flow across the stent may not be adequately seen due to artifacts, patency of stent flow proximal and distal to the stent can be adequately evaluated (Fig. 11).

To be clinically acceptable as screening modality, main requirement of CTA are:

- CTA should be able to visualize all therapeutically relevant Coronary artery segments
- No segment should be excluded from analysis
- Sensitivity and specificity of CTA has significantly increased over the period of time.

With currently available 64 slice CT all therapeutically relevant coronary segments can be analyzed.

- With 4 slice CT – Sensitive of 58% to 80% was achieved. However upto 32 % of the vessels were excluded from analysis, due to decrease in image Quality[5].

- In 16 slice CT – 73 to 95% of sensitivity is achieved. (All segments included.)

- In 64 slice CT – Sensitivity 94% specificity 97%. NPV 99% is achieved and no segment need to be excluded from analysis[5].

Calcium plaques are responsible for majority of false positive and most of false negative results. All false positive lesions appear as wall irregularities in ICA[5].

Radiation dose-Radiation dose to the patient seems to be the real problem. Typical radiation dose per study is 13 mSV to 18 mSV[3]. This Radiation dose is in excess of the radiation dose received by the patient in ICA, excluding left ventricular Angiogram(3 to 5mSv)[8.] Most effective way to reduce radiation exposure is prospective ECG tube current modulation (ECG pulse)5 This technique may result in impairment of image quality during early diastolic phase which is very crucial for evaluation of RCA. Anatomy based dose regulation soft ware, provided by most vendors significantly reduces effective radiation dose[5].One more option is to skip non contrast scan for Calcium scoring[5].

64 slice CT Allows a Non Invasive assessment of haemodynamically significant CAD.

Extensive arterial wall calcification is still a problem.

MSCT current status - Is still a screening modality. If MSCT is normal ICA not required.If MSCT is abnormal ICA is required for revascularization procedures.

MSCT – Future-With rapid increase in hard ware and soft ware capabilities, in not too distant future, MSCT may be accepted as a diagnostic tool. Revascularization could be done based on MSCT findings alone.Role of ICA may be limited to therapeutic interventions, avoiding ICA for diagnostic purpose.

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Table – 1[3]

Sr. No.	Calcium scoring	Sensitivity	Specificity	PPV	NPV
1.	0 – 100 units	94%	95%	94%	95%
2	101 – 400 units	100%	88%	90%	100%
3	> 400 units	93%	67%	93%	67%

Table : 2[3]

Sr. No.	Body Mass Index	Sensitivity	Specificity	PPV	NPV
1	Normal <25kg.Msq	100%	100%	100%	100%
2	Over weight(25-29.9)	100%	91%	93%	100%
3	Obese (>36 kg/Msq)	90%	86%	91%	86%

Table - 3[3]

Sr. No.	Pulse rate	Sensitivity	Specificity	PPV	NPV
1	Under 70 beats/min	97%	95%	97%	95%
2	71 to 85 beats/min	88%	71%	78%	83%
3	Over 85 beats/min	0 %	100%	0 %	100%
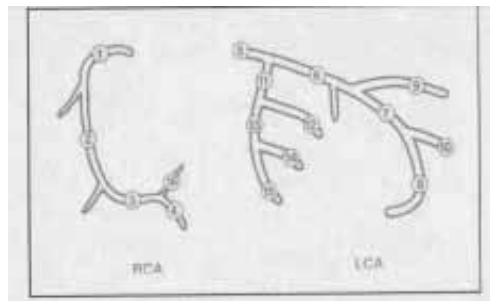
Figure-1, Segmental anatomy of the coronary arteries after a modified AHA classification. 1, RCA proximal; 2, RCA mid; 3, RCA distal; 4, right posterior descendens; 5, main stem; 6, LAD proximal; 7, LAD mid; 8, LAD distal; 9, first diagonal; 10, second diagonal; 11, LXC proximal; 12, obtuse marginal; 13, LCX distal; 14, LCX posterolateral branch; 15, LCX posterodescendens branch; 16, RCA posterolateral branch. RCA, right coronary artery; LCX, left circumflex artery; LAD, left anterior descending coronary artery.

Fig-2, Calcium scoring scan shows heavily calcified LAD and LCX. ICA (Left) shows total occlusion of LCX and severe narrowing of LAD
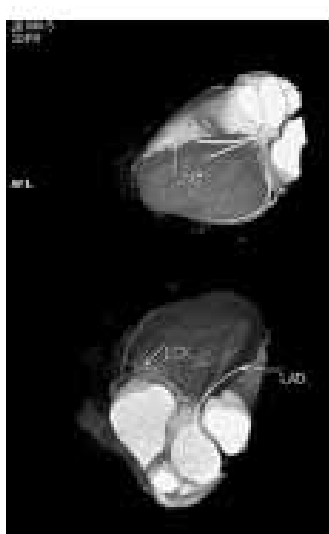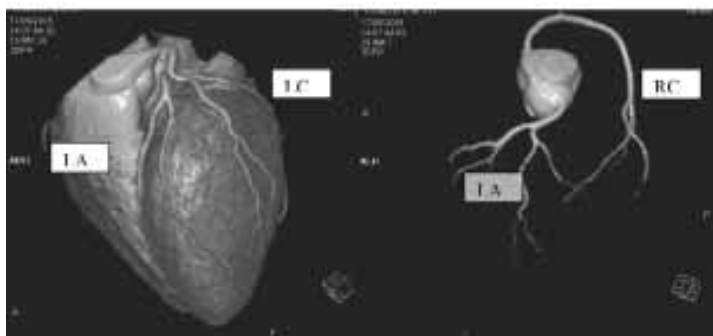
Fig-3, Normal Coronary Tree (VRT)

Fig-4, Normal Coronary Tree (VRT)

Fig-5, Shows multi vessel disease – with severe narrowing of LAD, LCX, OM1 and D1

Fig-6, Shows severe narrowing of Proximal LAD and LCX compared to corresponding images of ICA (left)

Fig-7, Single Coronary Artery From Rt.Cusp
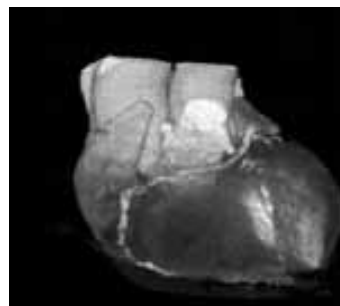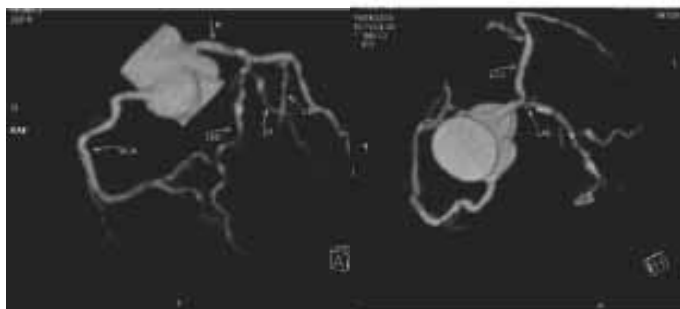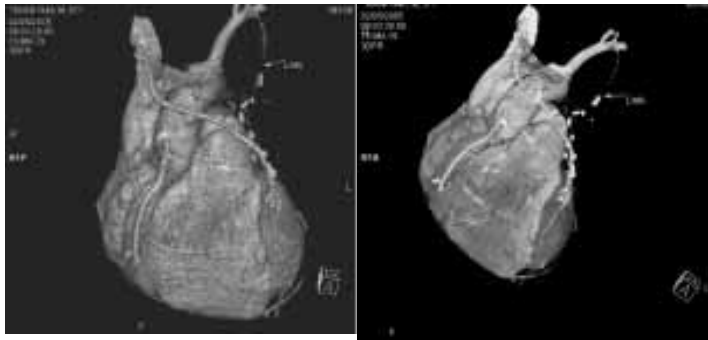
Fig-8, LIMA to LAD Block – SVG to Om1 Block; RIMA to LAD Patent

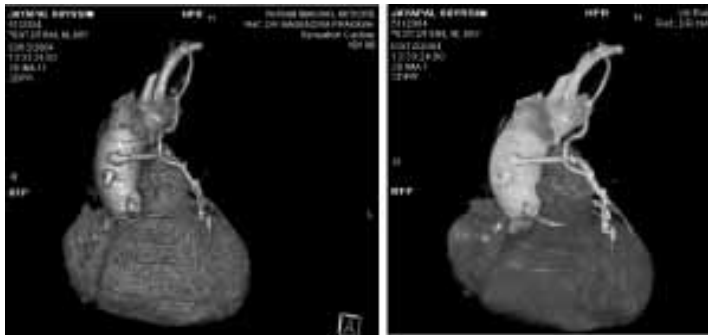Fig-11, Patent stent in Distal RCA corresponding ICA image on left

Fig-9, Total occlusion of Venous Graft to RCA. Patent venous graft to OM1 and LIMA to LAD
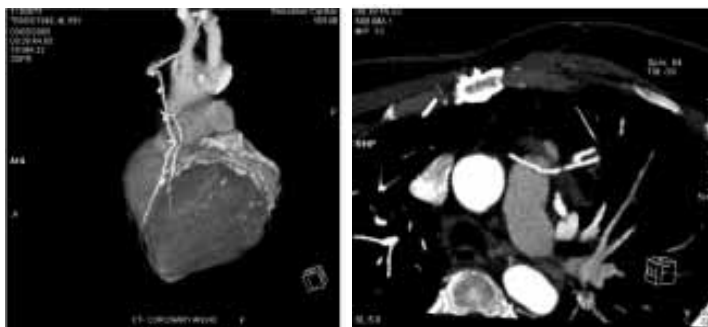
Fig-10, VRT and MIP (left) – shows discrete mid segment stenosis of Venous Graft to OM1

Gastrointestinal Tract-Management of Sigmoid Volvulus

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Volvulus is derived from the Latin word *volvere*, which means "to twist upon". In the colon, it refers to a condition in which the colon is twisted on its mesentery causing acute, subacute, or chronic colonic obstruction. For a volvulus to occur, the colon must be mobile and have sufficient length to rotate around a relatively narrow and fixed mesenteric base. As a result, the most commonly involved sites are the sigmoid colon and the caecum.

Incidence-It varies widely in different parts of the world. It is very common in India, Pakistan, Africa, Russia, Eastern Europe, and Scandinavia (volvulus belt), where as its incidence is very low in Britain, most European countries, and America. Volvulus of the colon accounts for approximately 5% of intestinal obstructions and 10 – 15% of colonic obstructions in patients in the United States.[1,2] Its exact incidence in India is not available but is said to vary between 12 – 30% of colonic obstructions.[3] Sigmoid volvulus is the most common form of volvulus of the gastrointestinal tract and is responsible for 8% of all intestinal obstructions. It accounts for 75% of all colonic volvulus, and 10% of all colonic obstructions.

Mortality/morbidity-Mortality rates are 20-25%, depending on the interval between diagnosis and treatment. Therefore, radiographic recognition of sigmoid volvulus is important.[5]

Risk factors-The risk factors that can make a person more likely to have sigmoid volvulus are Hirschsprung's disease, intestinal pseudo-obstructions, and megacolon. Adults, children, and infants can all have sigmoid volvulus. It is more common in men than in women, possibly because men have longer sigmoid colon. It is also more common in people over age 60, in African Americans, and in instit-ionalized individuals who are on medications for psychiatric disorders. The common factor is chronic constipation. In addition, children with malrot-ation are more likely to get sigmoid volvulus.

Anatomy-Examination of the base of the sigmoid at the time of surgery for volvulus may show that the two limbs of colon are bound closed, usually by fibrous adhesions within the peritoneum. Two anatomic differences can increase the risk of sigmoid volvulus. One is an elongated or movable sigmoid colon that is unattached to the left sidewall of the abdomen. Another is a narrow mesentery that allows twisting at its base. Sigmoid

volvulus, however, can occur even without an anatomic abnormality. This, plus the dependent position of a redundant sigmoid loop, predisposes patients to the volvulus. The most common and clinically significant twist of the sigmoid occurs in the mesenteric axis, although a less frequent and more benign form of the twist may occur around the longitudinal axis of the sigmoid loop. This longitudinal twist has been variably termed as the kink, axial torsion, or physiologic incomplete torsion. Patients with this twist are usually not symptomatic, and it may be an incidental finding on routine barium enema examination.[5] In acute sigmoid volvulus, the degree of torsion varies from 180° (35% of cases) to 540° (10% of cases). 360 ° torsion is seen in 50% of patients. The torsion is usually counterclockwise. The common form of volvulus around the mesenteric axis usually is sited 15-25 cm from the anus and is therefore accessible with sigmoidoscopic exami-nation.

Pathophysiology-An unusually narrow attachment of the root of the sigmoid mesentery to the posterior abdominal wall permits close approximation of the two limbs of the sigmoid colon. This in turn may predispose patients

to a twisting in the sigmoid colon around its mesenteric axis. The anatomic defect may be complicated by predisposing factors, including a high-roughage diet, chronic constipation, and lead poisoning. Bowel gas may be able to enter the closed sigmoid loop through the twist, but it cannot escape. This condition results in massive dilatation of the sigmoid loop and further tightening of the obstructive twist leading to complete obstruction. The part of the digestive system above the volvulus continues to function and may swell as it fills with digested food, fluid, and gas. Failure in providing prompt diagnosis and treatment ultimately leads to colonic ischemia with perforation and peritonitis. The extent of sigmoid colon ischemic changes must be determined prior to resection to prevent anastomosis of the ischemic colon and subsequent stenosis or anastomotic leak.

Clinical details- The clinical presentation of volvulus of the colon is similar regardless of the site of the twist. Although a sigmoid volvulus may present insidiously with chronic abdominal distension, constipation, vague lower abdominal discomfort, and vomiting, it is seen more often as an abdominal emergency where a crampy abdominal pain, distension, diminished stool output, and nausea and vomiting consistent with obstruction are the hallmark complaints. Progress to constant abdominal pain implies the

development of serositis of the involved segment, which may act as a closed- loop obstruction increasing intraluminal pressure that leads to ischaemia. Furthermore, the mesenteric vasculature may be compromised by mechanical torsion of the volvulus around the mesenteric pedicle. Acute presentations such as this represent more than half of the total episodes of volvulus. Vomiting occurs late, and the distension may be gross enough to compromise respiratory and cardiac function. A subgroup of patients with colonic volvulus describes similar episodes in the past that resolved spontaneously, often with an associated explosive bowel movements or passage of gas. Patients with recurrent volvulus need a careful assessment to rule out the diagnosis of colonic inertia and megacolon, which may mandate a more extensive colonic resection.

Predisposing factors common to all sites of volvulus include previous abdominal surgery and a history of chronic constipation. A detailed history should include potential comorbidities that must be incorporated into the overall treatment plan as many of these patients are elderly, debilitated, and have multiple coexisting medical conditions. Physical examination reveals a distended abdomen which is tympanitic, varying degrees of tenderness over the obstructed segment and a palpable mass may be present. Shock and an elevation of temperature may be present in

instances of vascular compromise or colonic perforation. Rectal examination shows only an empty rectal ampulla.

Sigmoid Volvulus-Varieties

Acute Fulminating Type

- Mortality 37-80%
- Younger patient, sudden onset, rapid course
- Early vomiting, severe pain, peritonitis, and gangrene
- Minimal distension often, hard to diagnose

Subacute Progressive Type

- Generally older pt., more gradual onset
- History of prior attacks, chronic constipation
- Abdominal distension often extreme
- Late vomiting, pain is minimal, no peritonitis

Investigation-The diagnosis of sigmoid volvulus is usually made on plain abdominal radiographs.

Radiographic Findings-The key radiological features are those of a double-loop obstruction, which has been reported in approximately 50% of patients. The key finding consists of a dilated loop of pelvic colon, associated with features of small bowel obstruction and retention of feces in an undistended proximal colon. The dilated loop usually lies in the right side of the abdomen, and the limbs taper inferiorly into the right lower quadrant. Medial deviation of the distal descending colon is a rare but highly specific finding.

Plain radiographs show a markedly distended sigmoid loop, which assumes a bent inner tube or inverted *U*-shaped appearance, with the limbs of the sigmoid loop directed towards the pelvis. The colonic haustra are lost, and progressive distension elevates the sigmoid loop under one of the diaphragms. An upright radiograph shows a greatly distended sigmoid loop with air-fluid levels mainly on the left side of the abdomen extending toward the right hemidiaphragm. The involved bowel walls are edematous, and the contiguous walls form a dense white line on radiographs. This line is surrounded by the curved and dilated gas-filled lumen, resulting in a coffee bean–shaped structure; this is the coffee bean sign.[6] A dilated sigmoid colon that ascends to the transverse colon (northern exposure sign) is said to be a reliable sign of a sigmoid volvulus on a supine abdominal radiograph.[7] If more fluid than air is in the obstructed loop of the sigmoid, the volvulus may be demonstrable by a soft-tissue mass or a pseudotumor sign. Single-contrast barium enema examination is adequate because the barium readily enters the empty rectum and usually encounters a complete stenosis, which is likened to a beak, the so-called bird's beak or bird-of-prey sign. Barium enema examination can also demonstrate obstruction at the rectosigmoid junction. If barium can enter the obstructed segment, spiraling of the mucosal

folds may be seen. Signs of bowel ischemia, such as thumbprinting, transverse ridging, and mucosal ulceration, may be observed.

Limitations of Radiography-Diagnostic difficulties may occur with plain abdominal radiographs if the degree of proximal dilatation is so marked that the sigmoid loop may not be recognized as such. Similar difficulties may be encountered when a large amount of fluid is associated with a small amount of air. This situation causes poor definition of the sigmoid colon on a supine radiograph, and the high air-fluid level demonstrated on erect images may be inadequate to define the sigmoid loop accurately. However, in 60-70% of patients, diagnosis of sigmoid volvulus can be made by using plain abdominal radiographic findings. In 20-30% of patients, the two limbs of the twisted sigmoid colon may overlap or deviate to the right or left, obscuring the remainder of the colon. In these instances, the findings are those of a nonspecific large-bowel obstruction, and barium enema examination is required for confirmation of the diagnosis. Barium enema examination is contraindicated in patients in whom a gangrenous bowel is suggested or when a pneumoperitoneum is noted on a plain abdominal radiograph or erect chest radiograph. The examination is also contraindicated in patients with clinical signs of peritonitis. Sigmoidoscopy, rather than barium enema

examination, is the procedure of choice if an ileosigmoid knot is suspected.[8]

CT Findings-CT is the least invasive imaging technique that allows assessment of mural ischemia and helps in identifying the cause of an acute large bowel obstruction in most of cases.

CT findings of sigmoid volvulus include the whirl sign, which represents tension on the tightly twisted mesocolon by the afferent and efferent limbs of the dilated colon. It may also be useful in identifying the etiology and site of obstruction resulting from other pathologies and in demonstrating ischemia resulting from strangulation. CT signs of ischemia include a serrated beak at the site of obstruction, mesenteric edema or engrossment, and moderate-to-severe thickening of the bowel wall.[9]Intramural gas or portal venous gas may be seen (grave prognostic signs), and in patients in whom a perforation has occurred, a large amount of free intraperitoneal gas or fluid may be noted.

MRI Findings-MRI has been used successfully in the assessment of large-bowel obstruction (not specifically in sigmoid volvulus). These examinations are performed with the retrograde insufflations of 1000-1200 mL of air through a Foley catheter placed in the rectum and with scopolamine to inhibit peristalsis to demonstrate the site of bowel obstruction. In addition, MRI has been used in the diagnosis of mural necrosis

in infants, and theoretically, it can be used in adults.[10] However, with the limited experience at the present stage, routine use of MRI in cases of intestinal obstruction is not recommended.

USG Findings-Sonography might occasionally be useful in assessing large-bowel obstruction. But the experience in diagnosing sigmoid volvulus by using ultrasonography is limited as the images fail to depict the cause in most patients.[11, 12]

Differential Diagnosis-Other problems to be considered are:

- Other forms of large-bowel obstruction, especially carcinoma of the sigmoid colon
- Pseudo-obstruction
- Giant sigmoid diverticulum
- Ileosigmoid knot
- Constipation

Management - The initial treatment in the patient with no evidence of bowel necrosis based on history and physical examination should involve an urgent non operative endoscopic attempt at reduction of volvulus. Failure to successfully reduce the volvulus endoscopically or clinical evidence of vascularly compromised bowel mandates emergent exploration.[13]

Non operative intervention-Since its introduction by Bruusgaard in 1947, non operative decompression has become the treatment of choice for patients without any signs of peritonitis. With the patient in the left lateral position, decom-pression and untwisting of the sigmoid loop may be achieved by the passage of a long soft tube through the obstruction, per rectum under fluoroscopic or endoscopic control. This procedure allows for rapid decompression of the distended colon, with the immediate relief of symptoms. The tube may be left in situ for 48 hours to allow for complete emptying of the loop and for the resolution of mural edema.Most patients are elderly persons, and they may be treated conservatively with tube decompression per rectum. If rectal decompression is instituted, the patient should be observed for persistent abdominal pain and bloodstained stools, signs that may herald ischemia and indicate the need for surgical intervention.

Surgical management-
Surgery is reserved for patients in who tube decompression fails or for those in whom signs of ischemia are suggested. After conservative treatment, further episodes of volvulus occur in approximately 60% of patients. Such a high recurrence rate justifies an elective prophylactic sigmoid resection during the same hospitalization after the first episode of volvulus in all patients except in high – risk surgical candidates.[13]

Elective Surgery-Following successful decompression patient is planned for an elective resection of sigmoid colon. If associated with megacolon, total colectomy or subtotal colectomy is advised. One problem frequently encountered at laparotomy, especially when the volvulus is recurrent or chronic is the discrepancy between the proximal and distal bowel lumen. Moreover, the wall of the proximal bowel may be much thicker making it difficult for stapling. If a primary anasto-mosis has been decided upon, this can be undertaken by a hand sutured end - to - end anastomosis by taking wider bites of the proximal bowel. Alternatively, a stapled end - to - side anastomosis using a circular stapler is also recommended. This is fashioned by placing the anvil into the rectal stump, using a purse string and passing the gun into the open end of proximal bowel. The spike is advanced to pass through the anti – mesenteric aspect of the bowel leaving enough length beyond the staple line for subsequent closure once the gun is fired. The open end is closed using a linear stapler. Laparoscopic resection of the sigmoid colon for decompressed sigmoid volvulus may be a useful alternative in high risk patients or in the elderly who may not tolerate conventional colonic surgery.

Non Resection Surgery

- **Colopexy-** is said to have advantage of not requiring resection of the sigmoid colon and not requiring bowel preparation. Percu-taneous endoscopic colop-exy using PEG Kit is also in vogue.

- **Mesosigmoidoplasty-** first described by Tiwary and

Prasad in 1976 which constitutes broadening the base of the mesosigmoid and reduction of its length. It is a simple operation with low rate of operative morbidity and mortality. Also has advantage of no likelihood of anastomotic leakage and sepsis. Undue post-operative constipation is not a problem. In Subrahmanyam's series of 126 pts with an average follow up of 8.2 years showed a recurrence rate of 1.6% and no mortality. However, the lack of verification of Subrahmanyam's results in other surgeons' hands and a high recurrence rate counts against its routine use.[14, 15, 16]

- **Extraperitonealization for Sigmoid Volvulus** - Bhatnagar *et al* introduced the technique of extraperitonealizing the whole segment of the sigmoid colon with favorable results.[17]

But recent literature show that fixation procedures for the management of sigmoid volvulus are associated with high recurrence rates and are not recommended.[15]

Emergency surgery-The indications for emergency laparotomy are:

- The presence of peritonitis.
- The failure to decompress endoscopically.
- When ischaemia or strangulation is suspected.

The exact procedure will depend upon the viability of the colon. If the colon is gangrenous, there is no alternative to resection, taking care not to untwist the torsion. Resection of gangrenous bowel is done, with creation of an end colostomy and a Hartmann's or mucus fistula being the safest option in absence of formal mechanical bowel preparation. There are insufficient trials comparing patients treated with or without a primary anastomosis in this condition. Similarly, on – table lavage has not been widely employed for volvulus.If the bowel is of questionable viability, derotation usually in counterclockwise manner with observation for the return of adequate perfusion may avoid resection. Often the use of Doppler probe or wood's lamp following intravenous administration of fluorescein can help in further evaluating for bowel viability.[13] If the colon remains viable following derotation, primary resection and anastomosis may be performed in favorable circumstances. However, if there is slightest fear of a leak, exteriorizing both the ends is safest option.

Outcomes following treatment -Operative mortality rates for emergent surgery for sigmoid volvulus are considerably higher in presence of intestinal gangrene or failed non operative reduction, approximating 40%. In comparison, the mortality rate for an elective resection following successful endoscopic reduction is less than 10% [13].

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Obstetrics forceps

To Arthur H. Bill (1877-1961), obstetrics in the early 20th century confronted a two-fold challenge. First, obstetrical procedures required substantial improvement and second, individuals needed to be trained in the application of these methods. As Bill noted at the end of his career, "instead of bringing our art down to the level of general practitioners, let us bring our art up to a higher level and educate those who do obstetrics to that point." Bill promoted the methods of the "new obstetrics" pioneered by Joseph Bolivar De Lee of Chicago. As Bill observed, "the old plan [which] allowed nature to take its course, even in the face of abnormalities with the hope that eventually the abnormalities might correct themselves, has given way to far more scientific and humane methods of correcting abnor-mal conditions and thus assisting natural forces." The approach endorsed by De Lee, Bill, and their adherents revolved around such proce- dures as the use of scopolamine or "twilight sleep," prophylactic forceps delivery, and episiotomy. The "new obst-etrics' was highly interventionist in character. When entering the field of obstetrics, Arthur Bill was alarmed that many physicians lacked adequate training in the proper use of forceps. The consequence: damage to both mother and child. He worked to remedy this by carefully teaching his students the technique. Bill structured the obstetrics training program at Case Western Reserve University so that students did not simply witness confinements, but helped deliver as many as 40 babies and attend innumerable labors. By his retirement in 1948, Bill had trained over 2,000 obstetricians-gynecologists in this manner. Bill also developed the Bill Axis Traction Handle, which further reduced the chance of damage to the child and lacerations to the cervix of the mother. This attachment was placed over the front of the forceps handles and made the instrument more accurate in delivery by reducing and determining the force needed for a forceps delivery. Arthur Bill employed the forceps and axis traction handle seen here during his tenure at MacDonald House, the maternity hospital of University Hospitals of Cleveland. For obstetricians, the choice of forceps could be overwhelming; some six hundred variants had been devised since the introduction of the instrument by the Chamberlen family in early-17th century England. For most forceps assisted deliveries, Bill preferred the Tucker McLane forceps, introduced around 1880-85: They were especially appropriate for outlet and low forceps deliveries, where the head is less than 45 degrees rotation from the occiput anterior position. The Tucker-McLane is distinguished from other forceps by its solid or non-fenestrated blades, prominent pelvic curve, and overlapping shanks, and articulating lock.

Traumatic sinus venous thrombosis -a case report

We report a case of severe head injury with depressed fracture fragments causing sinus venous thrombosis. On day of admission C.T. scan head plain study revealed depressed comminuted fractures of high biparietal bones. Patient developed paraplegia on second day, hence MRI brain with MR venography was performed. It revealed dissection of superior sagittal sinus causing thrombosis and bilateral high parietal venous infarcts. Timely clinical suspicion of sinus thrombosis in cases of depressed bone fragments located over major venous sinus and importance of MRI in its diagnosis are discussed.

The first case of post-traumatic cerebral venous thrombosis is reported by Holmes and Sargent in 1915. Cerebral venous thrombosis is a challenging condition because of its variability in clinical signs and symptoms. It is often unrecognized at its initial presentation. Amongst the various etiologies of it head injury is the commonest non-infective cause.Though angiogra-phy is considered Gold standard, MRI & MR venography is currently preferred technique for diagnosis. It can show consequences of thrombosis such as cerebral edema, infarction and haemorrhage as well as anatomy of disturbed venous circulation[1].

Case Report -A 40 Yrs old male patient with history of severe head injury brought in an unconscious state for CT scan brain. He had earlier episodes of convulsions and vomiting. Physical examination revealed depressed comminuted fractures of biparietal bones with soft tissue swelling.On neurological examination, eye opening to painful stimuli and incomp-rehensive sounds were present counting to Glasgow Coma scale of 8.CT brain plain study revealed depressed comminuted fractures of high biparietal bones. The displaced fracture fragments were causing impingement on underlying brain parenchyma. (Fig.-1). There was minimal acute subdural haemat-oma present along fronto-parietal conv-exity.On second day of admission patient developed paraplegia with absent lower limb deep tendon reflexes. Radio-graphs of cervical, thoracic and lumbar spine were done which did not reveal any fracture. To find out the cause of paraplegia MRI brain was performed. It revealed extensive sinus venous thrombosis involving superior sagittal sinus, right sigmoid and transverse sinus (Fig-2,3,4) with acute haemo-rrhagic venous infarcts in bilateral high parietal regions.(Fig-5, 6). Findings of sinus venous thrombosis were confirmed with MR venography (Fig-7,8). The depressed comminuted fractures of high biparietal bones caused dissection of superior sagittal sinus. Thrombosis of superior sagittal sinus leading to bilateral high parietal venous infarcts caused paraplegia. Patient received conservative manage-ment in the form of anticonvulsants, antibiotics and diuretics without revision of fracture fragments. Over period of one month, he regained grade IV power in both lower limbs.The follow-up MRI showed resolution of parasagittal venous infarcts. MR venography showed partial recanalization of superior sagittal, right transverse and sigmoid sinuses (Fig- 9,10).

Discussion -Thrombosis of cerebral veins and dural sinuses has a wide spectrum of clinical presentation. So the develo-pment of acute neurol-ogical symptoms in predisposing clinical settings should make clinician to consider the sinus venous thrombosis as possible diagnosis[2].Mechanism for development of thrombosis after trauma may include bone fragments, sinus dissection or distortion creating obstruction to flow that in turn induce thrombus formation[3].In case of depressed skull fracture located over the major venous sinus in which there is an unexplained worsening of the patient's neurological condition should suggest possibility of obstruction to venous flow caused by depressed bone fragments [4]. A

depressed fracture overlying part of superior sagittal sinus can cause persistent intracranial hypertension. If the signs and symptoms due to such condition persist or progress despite conservative management, surgical intervention is recommended [5].Radiological evidence of flow in the affected segment of venous sinus and presence / absence of thrombosis inside the lumen is of great clinical importance[4].In our patient CT scan showed only depressed fractures while MRI brain and MR venography revealed exact cause of paraplegia. MR venography being non-invasive imaging modality plays a vital role in detection of sinus venous thrombosis.

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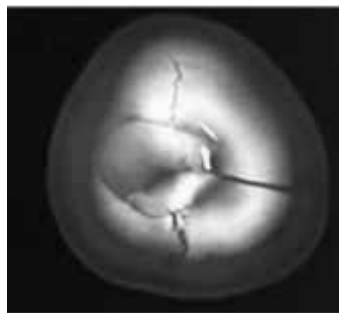Department of Radiology
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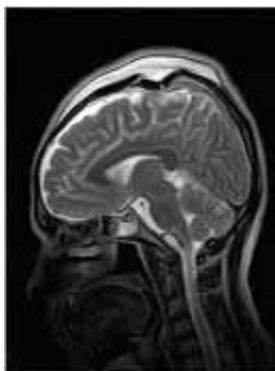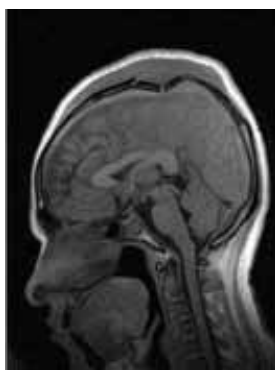Fig- 1,CT - Fractures of biparietal bones

Fig- 4, MRI Flair Cor : loss of flow void at Rt. Sigmoid S

Fig. - 2, 3, MRI T1WI ,T2WI Sag; Loss of Flow void in SSS with depressed fracture fragments
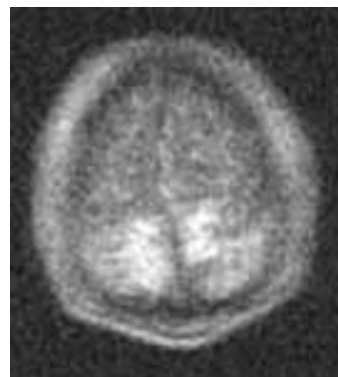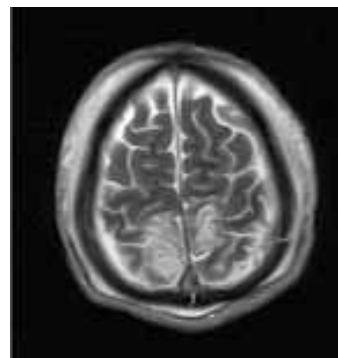
Fig - 5, 6, MRI T2 Axial, DWI BL high parietal parasagittal haemorrrhagic infarcts

Fig -9, 10, Partial recanlization of SSS, Rt. Transverse, Sigmoid Sinus

Fig -7,8, MRI veno- absent flow in SSS, Rt. Transverse, sigmoid sinus

- Talk presented by senior doctor or an expert in the field on a topic of GI specialty.

The sessions are conducted with interactive manner with balance of support and challenge to students to develop clinical reasoning in case based format. The sessions were attended and all teachers and students find these very useful and of great value. I feel this format may be used by many institutions which are situated closer to each other physically.There are also concerns about appraiser visits, its logistics as well as time constraints. I suggest a new approach for this. Mini-CEX popularized by John Norcini (References attached) is now well accepted all over the world and is useful tool that can be adopted by all of us. In Mini-CEX residents are assessed in various clinical settings with a diverse set of patient problems. It tests the postgraduates in clinical- real patient's scenarios and has simultaneous feedback and assessment. The tests are conducted by skilled clinician educators. Residents receive the ratings in the physical exami-nation and ratings in professionalism. Comparisons over the year of training their ratings are to be rated to assess improvement in all aspects of competence. Furthermore, the Mini-CEX has higher fidelity and permits evaluation based on a much broader set of clinical settings and patient problems, and is administered on site and it

Group Learning and MINI CEX

Super specialty departments usually have very few residents and hence the interactive learning opportunities are less. To overcome this and provide more exposure to our postgraduates departments of GI Surgery at Mumbai (namely Seth GS Medical College & KEM Hospital, Lilavati Hospital, Jaslok Hospital, Jagjivan Ram Hospital, Wockhardt Hospital), have come together to conduct monthly meetings. The meetings are conducted every month by rotation in all hospitals and are attended by all teachers and postgraduates. The format of the meeting is

- Update of a common interest topic by the resident (student) doctor, which is discussed at length.
- Case presentations followed by discussion as done in Practical examination.

involves feedback from the students during our monthly meets rather than conducting appraisers at six monthly intervals. MiniCEX can be conducted 4-6 times a year and is time and cost efficient. I feel we all should explore this new method to replace appraised system. MINICEX sessions can be standardized and recorded.

I therefore urge national board to promote group learning wherever possible and accept these as learning experiences as well as shift to MINI CEX system to appraiser system as it is more valid and convenient.

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Sciatic nerve block causing heel ulcer after total knee replacement in 36 patients

Femoral and sciatic nerve blocks are commonly used for postoperative analgesia following total knee replacement. We present cases of heel ulcers following a peripheral nerve block for knee replacement surgery. Heel ulcers have resulted in delayed rehabilitation in these patients. Pressure points in the foot should be protected after the nerve blocks to prevent occurrence of pressure sores. Awareness of this uncommon complication is necessary to prevent its occurrence and avoid delayed rehabilitation of patients[1]

Case report- We report a series of patients who developed pressure ulcers over heel following peripheral nerve block given for post operative analgesia after total knee replacement. This study was carried out over three years and included around 3000 patients. 36 patients developed ulcers over heel in post operative period which could be attributed to nerve blocks. All these patients had femoral and sciatic nerve blocks for post operative analgesia. The skin over the heel initially developed blisters few days after the surgery and later formed ulcers. The ulcers were dressed in aseptic manner and heel was covered with protective padding. The ulcers were inspected on regular basis. The advice of tissue viability nurse was sought as soon as possible. The ulcers took around 3-4 weeks to heal. During this period patent experienced pain while mobilising and rehabilitation was delayed. This delayed discharge of patients from the hospital.

Discussion-Peripheral nerve blocks are commonly used for postoperative analgesia following total knee replacement. They reduce the requirement of morphine in post operative period [2,3] The effect of nerve block lasts for around 48–72

hours after surgery. During this period patient experiences anaesthesia in the operated leg and can not actively move his foot or ankle. Patients lie in bed till mobilisation begins. Mobilisation of patient is delayed till the effect of nerve block wears off i.e patient regains sensations in leg and starts to move ankle and foot. After total hip replacement surgery operated leg is protected in a trough to avoid dislocation of the hip. The trough keeps heel of the foot elevated and thus avoids development of pressure sores. After knee replacement surgery the trough is not used in order to avoid flexion deformity in knee. The leg is wrapped in compression bandage and kept flat in bed. This puts pressure points in foot at risk of developing sores. Sores are painful and delay the mobilisation and rehabilitation of the patient. This increases the duration of hospital stay as well. Peripheral nerve blocks are very effective for postoperative analgesia and also reduce requirement of morphine in post operative period. Proper measures should be undertaken to avoid development of heel ulcers e.g. two hourly position change, protecting the pressure areas with appropriate hydroxypolymer dressings, monitoring the changes in skin and seeking early help of tissue viability nurse and plastic surgeons. A comprehensive 12-step detailed protocol for treatment of pressure ulcers is described; this includes recognizing that every

patient with limited mobility is at risk for developing a sacral, ischial, trochanteric, or heel ulcer; daily assessment of the skin; objective measurement of every wound; immediate initiation of a treatment protocol; mechanical debridement of all nonviable tissue; establishment of a moist wound-healing environment; nutritional supplementation for malnourished patients; pressure relief for the wound; elimination of drainage and cellulitus; biological therapy for patients whose wounds fail to respond to more traditional therapies; physical therapy; and palliative care. Availability of the described treatment modalities, in combination with early recognition and regular monit-oring, ensures rapid healing and minimizes morbidity, mortality, and costs[4]. If not, the development of heel ulcer hampers the very purpose of nerve block i.e. painless early mobilisation after surgery.

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Evaluation of postgraduates in viva voce examination

It is well known that basic needs of the society are food, clothing, shelter, health and education. Hence the medical persons are one of the main pillars of the society. So, on 'Medical Examinations' as a whole (Theory, Oral and Practical) giving passport to a postgraduate student, to treat, to teach or to go for research and higher education must be genuine and truthful. The goal of the examination is to show green signal to the deserving candidates only, so that he will be beneficial to himself, the medical education, research and the society with some contributions. The undeserving candidates should be made understood about their deficiencies and how to improve

for future. Thus to make a candidate pass the verdicts should not be on compassionate ground, for popularity, to maintain certain percentage of success, or to avoid bad reputation of the institution or the examiner. There may be various other factors also for leniency. But the examiner must not be responsible directly or indirectly to certify any undeserving candidate, who will downgrade the medical education, disorganize research or disrupt the society or any family. All the modes of medical examinations including viva – voce examination bear the same connotation and inner sense.

Evaluative value of viva and practical examination is more important than theory because in the former theoretical knowledge, practical experience, related mental faculties and a total audiovisual assessment of the student in general can be done. In viva voce examination there are three components viz. Environment, Examiner and the Examinee (the three E's). These three 'E's' must be congenial for a rational, beneficial cognitive analysis of the students and not only the examination of the theoretical knowledge. The examination must not be only as per regulations and formal. It should be lively, methodical, disciplined, systematic, meaningful, useful and practical oriented. In the viva part of the examination particularly, the students thought process, confidence, mode of interaction and communication, analytical quality, experiences and senses,

readiness, reception and reproduction of various aspects of his knowledge and extempore answers are judged and assessed along with personality and other general factors viz, clear pronunciation, audibility of the voice, language facial expression, temperament, etc. the knowledge delivered by the student should not be an amalgamation of sundry of linkless, ideas, imagination and knowledge. He must know his limits. At the end he should be convinced about his achievements and failures.

Environment-The examination must be conducted in a separate room and not in a crowded hall. The environment should be easy, congenial and comfortable. It is better to welcome them all in the waiting hall before starting the examination to allay fear of unknown, to make them confident and to assure them. While the examination is going on too many persons must not enter or leave the examination room frequently. Unrelated persons must not remain by the side, interrupt, comment, ask questions or talk irrelevant topics within the hearing of the examiner and the examinee and cause distraction of both.

Examiner-The examiner should not be by virtue of post only. He should be knowledgeable, well-versed and well-experienced. From the onset the attitude of the examiner should be, to make the student understand that he is not hostile, vindictive or too hard, rather helpful. The approach of the examiner should be encouraging, impartial, sympathetic and student friendly. He

should not be biased, clumsy in his language, gloomy, laughing, rebuking or sullen. He should know the difference between teaching room and examination room, particularly if he feels to guide the student while interrogating. He should talk freely in conversation style and should form the conversation a dialogue and not a monologue. He should not use uncommon words, terms or phrases or long, complex and compound sentences. To get the answer or response from the student, he should not reiterate endlessly. He should not try to show his knowledge, ask rare, uncommon or unimportant questions at the onset and at mid progress. The questions should be for short answers and in a ladder pattern from basic to higher strata. Some questions must be straight, direct and simple. The question pattern should be changed according to the depth of the knowledge of the student. Gradually questions should be analytical, problem related or inviting some instant thought process. The examiner should give pause in between questions. Questions in the same topics should be with links. A brief note, introduction, a short pause or a signal is preferred before starting a separate topic. While asking, the examiner should assess the bad, the better and the best and categorise accordingly on overall assessment. He should maintain same standard and time and behave equally with all throughout. Marks should be awarded in gradations and not lavishly, generously or in a flat

rate and as already mentioned not on compassionate ground, to maintain certain percentage, to favour someone or to avoid bad reputation of the examiner or the institution.

Examinee-The mode of entry of the examinee should be gentle, impressive and graceful. He should be at ease, comfortable and confident, but not over-confident. He must not be consternated, nervous or timid, casual, thoughtful, unmindful and as if 'taken aback'. He should not have apprehension, anxiety, phobia, suspicion, uneasiness, dismay and worries. He must be respectful and should show his readiness, prompt and intelligent responsiveness. He must be prepared with good knowledge and have common sense and must not be obsessed or preconceived with rumours or certain ideas. He must know his limits and where to start, how to proceed and when to stop. He must listen carefully what the teacher says and wants. He must not rush through or jam his presentation, deliver amalga-mated, linkless facts and a bizarre idea of his knowledge. He must not argue unnecessarily, but should keep balance in interaction. His verbal expression must not be entangled or with incomplete words and sentences, half expressed and half rolling in the mouth. He should not be repetitive with too many pauses and require too many leading and helping questions.

Objectives of viva on thesis- The main objectives should be exploration of the aptitude, sincerity, perfection, analytical quality, correlation, acumen,

spectrum of knowledge and depth of active participation in the project, as well as assessment of maintenance of format and the study as a whole, and separately on different chapters. Assessment should also be of validity of topics, fulfillment of aims and objectives and study of relevant works in review. Types of reviewed articles should be checked: whether recent, related or not. Number of Indian authors should be accounted. The quality of material / patients and methods / procedures should be judged. The statistical analysis and its significance should be verified. Bibliography should be quantitative and qualitative including regional, national and international authors. Examiner should judge rationality of analysis of results and observations. He should assess inner attitude regarding the work, whether the student is formal, interested, decisive or casual and unconcerned. In the discussion part the approach, self observation and interpretation, comparative aspect, style and analytical quality should be judged. In summary and conclusion part, correlation of the idea of the topics, aims and objectives, observation, ratio-nality and the outcome should be expected.

Conclusion-To make the viva fruitful and useful

- There should be pre-viva rehearsals in the institutions
- The examiner should feel that he had been truthful all along
- The examinee must know his limits, achievements and fallacies

- The environment should be friendly and not disturbing
- The success of the viva is satisfaction on either side

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Repair of inguinal hernia by a simple technique-a preliminary observation

Inguinal hernia is classically a challenge in terms of posterior wall repair to prevent recurrence. There is more than one technique for repair of inguinal hernia with the advantages and disadvantages of their own. Repair of inguinal hernia is an art of restoration of the anatomy and normal physical function with a definite object of lowering the rate of *recurrence* in the long term follows up.

Hypothesis behind the procedure-Hernia is the protrusion of a loop of bowel, organ or omentum through weak anatomical opening. Since intra peritoneal fat, connective tissue and loop of bowel abut against the lower abdominal wall, the protrusion of tissue through superficial ring or through deep and superficial ring constitute the direct and indirect inguinal hernia.[1] Anterior abdominal wall muscles namely external oblique (EO), internal oblique (IO), rectus abdominis(RA) and transverses abdominis(TA) take part in the shutter mechanism of

the inguinal canal. The inter actions between the intra abdominal pressure and the integrity of the four muscles are extremely important in maintaining the position of the viscera within the abdominal cavity. Three arches formed by the muscles, TA, IO and the EO help in prevention of hernia formation by the Clamp down mechanism.[1] It is evident that a patient with inguinal hernia might have any combination of pre formed sac and weak posterior abdominal wall. In the genesis of inguinal hernia, the preformed sac, the intra abdominal pressure and clamp down mechanism come into play with varying combinations. Intra abdominal contents are always under a constant waving pressure which is normally accepted and opposed by the semi circular shape of the lower abdominal wall. When the force of intra abdominal viscera is exerted upon the semicircular arc of the lower abdomen it falls perpendicularly to the curved anterior abdominal wall uniformly. But when the semicircular abdominal wall is weak there is uneven pressure on port like inguinal ring and canal. The direction of the propagated force tends to pass through this opening making the sac conical and dragging it through the opening to form a hernia. So, one force is intra abdominal, second is dragging force by weight and the third one is weak anterior abdominal wall work in combinations to generate

hernia.In our procedure the sac was twisted at the neck after reduction of the content. The twisted neck was fixed at the deep ring. The redundant part of the sac was not excised but was fixed over the posterior wall for reinforcement. The conical sac was expected to align again on the curved line of the abdominal wall. The procedure of twisting the neck causes multiple radiating folds of the sac. The radiating folds might offer the advantages of distribution of the pressure evenly on the lower abdominal wall to maintain the semicircular form in addition to the obliteration of the ring.

Procedure- Most of the operations were undertaken by the local anaesthesia and intra muscular sedation as a day case procedure with advice to attend the OPD on the 3rd day. A transverse incision was made over the deep ring about 3 inches in length. The spermatic cord was hooked gently by the finger dissection without damaging the pampiniform plexus and vas differens .The neck of the sac was tied after the reduction of the content. The twisted unexcised neck was fixed at the deep ring by the vicryl. The sac was spread over the posterior abdominal wall, when available, without making it taut. Its margins were fixed by the interrupted vicryl sutures. When sac was not available or it was very adherent to the posterior wall repair was done by the apposition of conjoint tendon with the inguinal ligament

without tension. It was done in 2 patients. Skin closure was made by the silk in most of the cases. Application of cyanoacrylate glue was also tried in few selective cases. There were wound infections in our total series. Three in the silk sutures and one in the patient with applied glue. All the cases were controlled with antibiotics.

Result analysis-We operated upon 78 patients in between the age of 12years and 58 years. There were 75 male and 3 female patients. Two patients developed wound infection but ultimately controlled with the ugly skin scar and one with hypertrophied scar but did not develop any sign of recurrence. One patient developed bulging over the incision resembling recurrence but there was no impulse on coughing and subsequent follow-up showed no sign of recurrence.

Discussion-The aim of repair of hernia is to prevent strangulation and recurrence so that normal physical activity remains unaltered. Conventional method of repair of inguinal hernia is associated with a recurrence rate of 10 to 12% as described in most series.[2] Combination of Shouldice technique with insertion of polypropylene mesh in the pre peritoneal layer can reduce the recurrence rate 0.4% as described by the National Ambulatory Hernia Institute technique (NAHI).[2] Mohan P. Desarda described a new technique of repair of posterior abdominal wall by the undetached strip of

external oblique apponeurosis. In his series of 400 patients there was only one recurrence after two years and had no such after 10 years follow up of 80 patients[3]. In our series the total number of patients was only 78 in between the age of 12 and 58.It is believed that the hernia sac is very thin and it is very weak membrane to support the posterior abdominal wall. So, repair with the sac of hernia is not unconditionally recommended. We tried to keep a regular contact with the operated patients by their available telephone number and address for communication. Far living patients are not included in this series.

Conclusion -In this preliminary study it is very difficult to comment on the applied technique in terms of recurrence but it is very clear that this procedure is very simple to perform even in the rural set up as a day case surgery. It is cost effective and acceptable to the patient. Conclusive results are yet to come after long follow up. No recurrence however has been recorded as yet after 6 years of follow up of 58 patients.

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Anesthetic Management of an Elderly Obese patient with Parkinson's Disease for Incidental Surgery – a case report

Parkinson's disease is a neurodegenerative disorder of unknown cause that affects all ethnic groups[1]. Age is the single most consistent risk factor, and with the increasing age of the general population, the prevalence of Parkinson's disease (PD) will rise steadily in the future[2]. Patients with Parkinson's disease most commonly present for urology, ophthalmological, or orthopedic procedures, and elderly surgical patients may have undiagnosed Parkinson's disease[3].Significant drug interactions, intraoperative exacerbation of Parkinson's disease, post operative respiratory dysfunction are the major problems during the anesthetic management of these patients. Here we report an elderly obese patient with Parkinson's disease who underwent surgery for carcinoma of penis with inguinal metastases.

A 66-yr-old man weighing 105 kg with Parkinson's disease was scheduled for partial penectomy with inguinal dissection for metastatic disease from cancer of penis. He was a known diabetic on oral hypoglycemic agents and a known hypertensive on anti hypertensive medications; however, his blood sugar and the blood pressures were well under control in the pre operative period. He had been suffering from PD for 6 yrs and had been treated with oral administration of Carbidopa/Levodopa (25/250), a L-Dopa (LD) preparation in tablet form, four times daily along with Selegiline 5mg twice daily and Ropinirole 0.5mg once daily. He manifested fluctuations of motor nerve disturbance, whenever the effects of LD disappeared. In the preoperative period the endocrinologist started insulin therapy for better control of blood sugar. The history, clinical examination and laboratory investigations revealed no other systemic illness in the patient. However, the ECG showed left bundle branch block and the chest X-ray showed bilateral basal infiltrates. The echocardiography showed normal LV function and an ejection fraction of 0.5 (50 %). The airway examination showed a Mallampatti grade of 3, normal thyromental distance and mild restriction of neck movements. He had undergone two operations 3 yrs ago for diabetic

foot under spinal anesthesia which were uneventful. The duration of surgery was expected to be long as the patient was obese and inguinal metastasis were anticipated to be adherent. Regional anesthesia was contemplated in this patient despite the expectation of a long surgery for the specific advantages especially in Parkinson's disease and the surgeons plan was to operate in the lithotomy position. The patient was explained about advantages of the regional anesthesia and he gave the consent for the same. On the day of the surgery the dose of all antiParkinsonian drugs including L dopa was given 30 min before the surgery with a small sip of water. On the Operating table a wide bore canula was secured and the patient was preloaded with 0.5 L of Ringers lactate solution. With the patient in the sitting position a combined spinal and epidural anesthesia was administered with dual space technique. Though the anatomy of the spine was difficult to appreciate due to obesity, an epidural catheter was successfully inserted in the L2 – L3 interspace and a combination of 12mg of 0.5% hyperbaric bupivacaine and 25ìg of fentanyl was administered in the subarachnoid space at the same interspace. The level of sensory blockade was at T 8 and the patient was positioned in the lithotomy position. The surgery lasted for 8 hours and the blood loss was within allowable limits. The patient was given L dopa

orally intraoperatively with a sip of water twice as per the schedule. Throughout the intraoperative period the patient did not have any exacerbation of Parkinsonian symptoms. The post operative recovery was uneventful and the analgesia was given with epidural boluses of morphine in 0.125% of bupivacaine mixture for the next 3 days. The patient received low molecular weight heparin for DVT prophylaxis and the epidural catheter was removed on the 4th day after stopping LMWH for 12 hours Parkinson's disease (PD) is a disorder of the extrapyramidal system resulting from the deficiency of dopamine in the basal ganglia.

Discussion-The classic triad of major signs of Parkinson's disease is made up of tremor, rigidity, and akinesia[4]. Respiratory dysfunction has been noted in patients with PD and multiple causes have been proposed including impaired central control of respiration, abnormal extrapyramidal control of respiratory muscles, excessive secretions, decreased chest wall compliance and upper airway obstruction[5]. Respiratory complications, particularly aspiration pneumonia, are the most common causes of death in these patients[6]. An obstructive ventilatory pattern has been observed in up to one-third of patients with Parkinson's disease[7.] Upper airway dysfunction is an important factor in the retained secretions, atelectasis, aspiration, and respiratory infection. Other

potential complications include post-extubation laryngospasm and postoperative respiratory failure. Dysautonomia is often a feature of Parkinson's disease. There can be abnormalities in salivation, micturition, temperature regulation and gastrointestinal function which is more commonly involved[8.] The cardiovascular manifestations are orthostatic hypotension, dependent edema and cardiac arrhythmias[9]. Therefore the clinical assessment should include an assessment of these problems with measurement of blood pressure in lying and standing positions. It may be unclear that an autonomic symptom is a manifestation of age, disease or therapy, or any combination of these factors. Regional anaesthesia has several advantages over general anaesthesia as it avoids the masking of tremor effects of drugs used for general anesthesia and the drug interactions associated with those drugs. Preoperatively, so that their symptoms may be observed[8]. We chose to administer regional anesthesia for our patient by combined epidural and spinal anesthesia technique despite the long duration of surgery for the concerns regarding the Parkinson's disease, obesity and difficulty with airway management, to avoid PONV, for excellent postoperative analgesia and to allow communication of the subjective feelings accompanying Parkinson's disease attacks, thereby

prompting earlier treatment. The muscle-relaxing effects of some general anesthetics and neuromuscular blockers will probably mask the tremors. Consequently, residual general anesthesia or neuromuscular blocker may delay diagnosis and treatment. The high incidence of nausea and vomiting associated with general anesthesia prevents effective administration of oral medications. Regional anesthesia avoids the effects of drug interactions associated with the drugs used for GA. Inhalational anesthetic agents have complex effects on brain dopamine concentrations, inhibiting synaptic reuptake of dopamine, thereby increasing its extracellular concentration and affecting both spontaneous and depolarization evoked dopamine release.[10,11] Among the intravenous induction agents ketamine is theoretically avoided to prevent exaggerated sympathetic responses.[12] Propofol has been associated with dyskinetic effects and abolition of tremor and infact it is recommended that it should be avoided in patients who are scheduled for stereo tactic surgery as they have their anti-Parkinsonian medication stopped for 12 ± 24 h.[13,14]Much of the evidence about the safety of various anesthetics drugs or techniques is based on single case reports or small case series. The usual drug regimen was administered as close to the beginning of anaesthesia as possible.

L-DOPA can only be administered enterally and its half-life is short (1 ± 3 h). It is absorbed from the proximal small bowel and therefore cannot be given as a suppository. Patients should be able to take L-DOPA either with sips of water or by nasogastric tube. We administered our patient a dose of L-DOPA, 6 hours after the beginning of the surgery. The patient never had any tremors throughout the surgery. Furthermore, it is essential to ensure that patients do not miss medication doses postoperatively. There are no randomized controlled trials evaluating various anaesthetic techniques or drugs in patients with Parkinson's disease. Careful preoperative assessment of these often elderly patients for coexisting medical illness, maintenance of drug therapy up to the time of anaesthesia if required intraoperatively and continuing them postoperatively, using a regional anesthetic technique in preference and avoiding known precipitating agents are the steps for the optimal management of these patients.

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Hirschowitz fiberoptic Endoscope, 1960

Fiberoptic endoscopy entered the realm of practicality in February 1957 when Basil Hirschowitz passed the first prototype instrument down his own throat and, a few days later, down that of a patient. Hirschowitz began work on the "fiberscope" in 1954 when he was on a fellowship with Marvin Pollard at the University of Michigan. After reading an article by Hopkins and Kapany describing recent advances in fiberoptics, Hirschowitz visited the authors in Britain and discussed the application of fiberoptics to endoscopy. Over the next three years, Hirschowitz and his associates in Ann Arbor, physicist C. Wilbur Peters and his student, Larry Curtiss, devised a makeshift, but effective method of drawing out their own glass fibers. In late 1956 Curtiss succeeded in producing the glass-coated fiber with the optical qualities required for the fiber bundle of a gastroscope. Following the demonstration of the new fiberscope incorporating this advance in 1957, Hirschowitz collaborated with ACMI (American Cystoscope Manufacturing Inc.) to produce a practical instrument. Finally, in October, 1960, Hirschowitz received the first production model, and presented it in *Lancet*, confidently asserted that "the conventional gastroscope has become obsolete on all counts."

Basil Hirschowitz exam-ining a patient with the fiberscope, 1961.

This instrument diminished patient discomfort by enhancing flexibility and by reducing bulk. Notable endoscope refinements of the late 1960s and early 1970s included re-positioning of

Basil Hirschowitz examining a patient with the fiberscope, 1961.

lenses for wider field of vision, addition of channels for biopsy forceps, suction, air, or water, and four-way controlled tip deflection.

Fiberoptic technology transformed gastrointestinal endoscopy in ways even more profound than its most ardent advocates might have imagined. Endoscopic procedures become safer and hence more commonplace, and virtually no region of the gastrointestinal tract remained unexplored. William Haubrich, editor of *Gastrointestinal Endoscopy*, recalled that "improvements in endoscopic design were so numerous and rapid during the early 1970s that one could hardly purchase a new instrument and become acquainted with its use before that instrument was rendered obsolete by a new model." The expanding diagnostic capabilities of endoscopy were soon complemented by new therapeutic applications, including colon polypectomy with a wire loop snare (1971), cannulation of the pancreatic duct (1972), removal of biliary stone (1975), and placement of feeding tubes by gastrostomy (1979). The range of technical developments in gastrointestinal endoscopy was so extensive across a broad front that John F. Morrissey was prompted to claim that "I think we are approaching a plateau in instrument development."

Pathological Aspects of Tumors of Bone and Related Recent Advances

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Bone tumors are very rare. Unless the specialist dealing with bone tumors is sufficiently experienced in a referral center, correct diagnosis is difficult.Since primary bone tumors affect mostly children & adolescents,it is all the more necessary to avoid unnecessary sugery,chemo or radio therapy.A multi disciplinary approach by the clinician, radiologist & pathologist ,the traditional diagnostic triangle is the best way to arrive at a "Final Diagnosis". There are changing trends both at the diagnostic aspect & at the therapeutic aspect.Newly introduced adva-nced imaging techniques have helped in staging the disease.Limb sparing surgery and neoadjuant chemotherapy have improved the quality of life and survival of the patients. Immunohistochemistry & molecular genetics have helped in understanding histigenesis ,classification, pathogenesis and biological behaviour of bone tumors.So now another angle is added to the triangle ,making it a quadrangle in the analytical approach to diagnosis of bone tumors- namely the molecular data in addition to clinical, radiological & microscopic features of bone tumors.[1]

Analytical approach to bone tumors

Clinical

- **Age**-Malignant tumors like osteosarcoma & ewings sarcoma are common in children and young adults. Benign tumors & tumor like lesions are common in adolescents(second & third decade). Giant cell tumor is common in skeletally mature adults in the age group between 20 and 50. C h o n d r o - s a r c o m a, Malignant fibrous histio-cytoma, plasma cell myel-oma, metastatic tumors &secondary osteosarcoma are common in the sixth to eighth decade.

- **Sex**-Some tumors show a distinct male predominance for e.g. Ewing's sarcoma[2]

- **Bone involved**-Next to age, bone involved gives a valid clue to correct diagnosis. Osteosarcoma commonly involves the lower end of femur, Adamantinoma & osteofibrous dysplasia the tibia & fibula Chondr-osarcoma involves femur, pelvis and ribs and never affects short tubular bones of hands & feet.Chordoma arises in base of skull and sacrum.Benign chondroma occurs exclusively in the short tubular bones.

- **Location**-Most tumors arise from the metaphysical region of the bone. Giant cell tumor and chondro-blastoma arise from the epiphysis. Ewings sarcoma, chondrosarcoma and metastatic deposits involve the diaphysis.

- **Symptoms**- Pain is the first symptom in rapidly growing malignant tumors. So, osteosarcomas present with pain first followed by swelling. Locally aggressive lesions like giant cell tumor & adamantinoma also give rise to pain. Osteoid osteoma is characterized by intense pain relieved by taking paracetamol.

- **History**-Previous history of malignancy or irradiation is very important to recognize secondary tumors. Very rarely a benign lasion can be a seat of malignant transformation, e.g. osteoch-ondroma and ench-ondr-oma.

- Clinical parameters and examination can help to arrive at a possible diagnosis, but it has to be followed by radiological and histopat-hological examination. While the patient can be examined by the surgeon and the radiologist, on many an occasion the pathologist may not have access to the patient, so all the relevant clinical details and radiol-ogical findings must be provided to the pathologist at first instance of submitting the biopsy itself in order to shorten the time interval between biopsy and

getting the report to plan surgery.It is also ideal to have discussion among the surgeon, radiologist and pathologist in difficult cases.

Radiologial data

- Plain X-ray.
- Computed tomogram.
- MRI.
- Bone scintigraphy.

Plain X-ray –It is the important basic method to evaluate patterns of bone destruction, periosteal reaction and matrix mineralisation.

Patterns of bone destruction-This gives an important clue regarding the growth rate of tumor cells, best observed in the interphase between tumor cells and host bone.

- A permeative pattern is seen in rapidly growing tumors, where the marrow cancellous bone is destroyed by osteoclastic stimulation, resulting in fine rarefactions less than 1 mm in diameter, with multiple small ovoid lucencies in the cortex.This pattern is seen in osteosarcoma, ewings sarcoma, lymphoma, leukemia, myeloma, metastatic deposit and also in infections.
- A moth eaten pattern of bone destruction is seen in tumors with an intermediate rate of growth.Here the lucencies are 2-5mm in diameter, with a tendency to coalece destroying the cortical and cancellous bone. e.g. of this moth eaten appearance are chondrosacoma, eosinophilic granuloma and myeloma.

- A geographic pattern of bone destruction is seen in slow growing tumors charecterised by lucencies more than 1cm in diameter, this also stimulates osteoblastic repair resulting in a sclerotic border. Benign tumors are also charecterised by this pattern. E.g., fibrous dysplasia, non ossifying fibroma,chondromyxoid fibroma,solitary bone cyst, and low grade chondro sarcoma.

Periosteal reaction

- Buttress-Dense thickening of the cortex indicates an irritative lesion, mostly benign.e.g., infection, trauma, oteoid osteoma.
- Codman's triangle is produced by a rapidly growing tumor which lifts up the periosteum, a definite indication for biopsy.e.g. osteosarcoma, it can also be produced by osteomyelitis, sub periosteal hematoma & aneurismal bone cyst.
- Onion skin arrangement of periosteum is usully seen in Ewings sarcoma where there is alternating tumor growth ,infarction and host response.
- Spiculated or sun burst appearance is common in very aggressive tumors like osteosarcoma.

Matrix mineralisation

- Osteoid formation is seen in bone forming tumors, malignant osteoid appears as cloud like amorphous density with irregular mineralisation in osteosarcoma. Mature osteoid of

benign tumors like osteoid osteoma shows regular trabecular pattern of ossification.

- Cartilage is easily identified by the presence of stippled calcification.It may also be flocculant or ring or arc like densities.Pattern of calcification does not indicate malignancy in cartilaginous tumors.

Disadvantages of plain x-ray

Though plain x-ray is the first step among the imaging techniques it has got certain limitations[4]

- Partial destruction of the cortex & small lytic lesions can be missed
- Deeply located bones cannot be studied due to superimposition of soft tissue
- Poor detection of soft tissue involvement

Computed Tomography (C T)-CT also uses x-rays but the image is an indirect image of a slice of the body.(4). Sensitive detectors are used to obtain the matricial image of the slice which appears on a television screen.A selected part of the image can be reproduced on film.Being a computer image distances & dencities can be easily measured.Multiple contiguous slices allow reconstruction in different planes.Main advantages over plain x-rays will be-greater contrast resolution,central bones can be studied better, and it helps in interventional radiology to take CT guided biopsies and in intra tumoral injections.

Magnetic Resonance Imaging (MRI)-MRI is an advanced

imaging technique because of its excellent soft tissue contrast and multiplanar imaging capability.(4). By placing the body in a homogenous magnetic field, and measuring the protons (h+) of the body which act like small magnets and its interactions with the magnetic field, images are measured.Contraindications are cardiac pacemakers, ocular magnetic particles,recent surgical vascular clips and claustrophobia. MRI is used to assess the local extent and in staging of bone tumors. Intramedullary extension of tumors ,skip lesions and depiction of soft tissue and vessel involvement are studied by MRI.

Bone scan-Bone scanning uses intravenous injection of technetium 99 m diphos-phonates. (4) in the study of bone tumors. The radionucleotide uptake of the lesion depends on reactive or reparative response to the tumor .The advantage is, the ability to study the whole body and its high sensitivity. A negative examination rules out a lesion.Exceptions are: fastly growing lesions and purely lytic lesions.

Histopathological exam-ination-Biopsy confirmation of a suspected malignant tumor is essential to proceed with a definite line of treatment. Ewing's sarcoma and osteomy-elitis resemble clinically where histopathological confir-mation is mandatory. Biopsy also helps in histological typing,and grading and with neoadjuvant therapy tumor mass can be reduced before surgery. Ancillary techniques like immunohi-stochemistry, molecular genetics and electron microscopy can be done with the biopsy material.

Types of biopsy- closed & open-Open biopsy and frozen section to assess adequacy of material have been replaced now with closed biopsy techniques, namely Fine needle aspiration cytology (FNAC) and core needle biopsy.(CNB). FNAC has the advantage of being minimally invasive with little discomfort to the patient ,being an OP procedure, immediate report can be given especially in cases like Ewing's sarcoma,metastatic deposit and giant cell tumor.Absolute requirements are the radiological investigatory results, to enable the pathologist to aspirate from the representative site of the lesion. Deep seated lesions require the radiologist's help under CT guidance. There are no complications or risk of tumor spread.A preoperative diagnosis of a high grade primary bone tumor enables the patient to undergo chemotherapy or combined chemo & radiotherapy before surgery. Core needle biopsy is a more invasive and traumatizing procedure so it is done under anaesthesia, in a theatre.It has the advantage of providing enough material with preserved tissue architecture for special techniques.So the accuracy rate is higher than the FNAC . When FNAC or CNB fail to establish a definitive diagnosis,open biopsy is done. This should be done by the surgeon who is going to do the definitive surgery, so as to plan the site of biopsy which should be within the incision of definitive surgery. The accuracy of open biopsy is 100%in ideal conditions and open biopsy must immediately be followed by

definitive surgery as the risk of spread of tumor cells is maximum with open biopsy technique.

Specimen handling-Most of the malignant tumors have soft tissue extention and permeation and they entirely are made up of unmineralised tissue. Slides can be prepared after processing within 24 hours. But some tumors may have mineralized hard material which has to be teased out and decalcified before sections can be prepared.This takes another 24 hours. Excessive decalcification may wipe out cellular details so one has to be careful to avoid this.Formic acid or Nitric acid are used as decalcifying agents. In a resection specimen margins must be sampled for clearance. Assessment of percentage of necrosis after chemotherapy in resected specimens of osteosarcoma helps in predicting prognosis. So entire tumor in the longest axis should be sampled.The standard approach of formalin fixed,paraffin embedded,haematoxilin eosin stained sections give adequate details in most of the malignant tumors of the bone ,but in an uncommon clinical presentation or when a rare neoplasm is encountered, the H & E sections must be supplemented by special techniques to substantiate the diagnosis. Special techniques also help in knowing the histogenesis & absolutely essential for assessing prognosis and in research methodology.

Special techniques-Special stains, Immunohistochemistry, Flowcytometry, Electron micro-scopy, Cytogenetics.

Special stains

- **Periodic acd-schiff stain-** This stain demonstrates intracytoplasmic glycogen , it is useful in Ewing's sarcoma,clear cell chondrosarcoma and also to demonstrate fungi & parasites.

- **Mucin stains-** These stains are useful in demonstrating mucins in metastatic adenocarcinoma.They are also used in chordoma which has intra and extra cellular mucin, especially when chordoma presents in abnormal locations like the nasopharynx and in the neck. Though some of the special stains like reticulin,trichrome and histochemical stains like acid phosphatase (for osteoclast) are still used, they are mostly replaced by the more specific and sophisticated immun-ohistochemical stains.

Immunohistochemistry- In this method, the cell of origin can be ascertained by detecting the particular antigen charecteristic to the cell. This is accomplished by adding highly specific monoclonal antibody to the section and visualizing the bound antigen antibody complex by a colour based detection system. The pathologist can precisely localize the positive reaction under the microscope. The most frequently used method is the avidin- biotin and peroxidase based detection system.

Advantages- Retrospective study is possible as it can be applied to stored paraffin embedded tissue; Cytological material can be studied; Decalcified and stained

sections can also be studied by this method.

Uses- It helps in identifying the cell of origin in disputed cases for e.g in cases of chondrosarcoma vs chordoma; lymphoma vs small round cell neoplasms.It does not differentiate benign from malignant cell. So the main use is to classify accurately a neoplasm which is essential to choose the correct line of management.

Disadvantages- The exorbitant cost which smaller institutions may not opt for; False positive and false negative results which may result in ambiguous diagnosis.The pathologist must be familiar to the technical aspects and continuously exposed to reading the pattern of staining with positive and negative controles to be able to give an accurate interpretation.

Immunohistchemical markers in bone tumors

- **Intermediate filaments-** These are ultrastructural cytoplasmic microfilaments and tubules. Based on their chemical composition and function there are 5 types of intermediate filaments- Vimentin, Keratins, Desmin, Glial fibrrillary acidic protein, Neurofilaments. Of these, expression of keratins and desmin help in the work up of bone tumors.Keratins are a group of 19 polypeptides with varying molecular weights. They can be broadly classified as acidic or basic.Different epithelia express different type of keratin.Organ specific keratin profile must be known in the differential diagnosis of tumors. Though keratins are

markers of epithelial differentiation,some tumors of mesenchymal origin also express keratins.In skeletal neoplasms keratins are expressed in adamanti-noma, chordoma, chondrosarcoma, epithelioid hemang-ioendot-helioma, sarcomatoid carcinoma and in metastatic neoplasms.[1] Desmin is marker of muscle differentiation, and also expressed in cells with contractile properties like myofibroblasts. It is generally used in tumors with skeletal muscle differentiation - rhabdomyo sarcoma.In bone tumors it is useful in differentiating primary from metastatic spindle cell neoplasms. It helps in identifying rhabdomyoblastic differentiation in dedifferentiated chondrosarcoma.It is also used in the differential diagnosis of small cell tumors of the bone.Actin is another marker of muscle differentiation and it is also used in the DD of primary and metastatic spindle and round cell tumors.

- **Vascular markers-** Factor VIII associated antigen or von Willibrand factor is a protein expressed by endothelial cells. In hemangioendothelioma, it is used to confirm the endothelial nature of the tumor and also in the differential diagnosis of spindle cell lesions.

- **Neural markers-** S-100 protein. Though originally identified in glial and schwann cells,it is also expressed in fat cells, melanocytes, myoepithelial cells, Lang-erhans' cells and cartilage cells,especially

developing cartilage .So it is used in demonstrating developing cartilage as in chondroblastoma, chondromyxoid fibroma and mesenchymal chondrosarcoma. Together with cytokeratin and epithelial membrane antigen it is used in the differential diagnosis of chordoma, chondrosarcoma and chond-roid chordoma.

- **Neuron specific enolase-** This is an enzyme with alpha ,beta and gamma subunits. Different isoenzymes have different tissue expressions. In bone tumors it is studied as a marker for Ewing's sarcoma.It is also used in neuroblastoma and metastatic tumors with neural differentiation.

Flow cytometry-Flowcytometry is a technique which combines physics,optics,electronics and computer science.Evolution of this technique has made it user friendly and it can be utilized for clinical applications in immunophenotyping of cell surface markers and in quantitation of DNA content and cell proliferation. A laminar flow of monodispersed suspension of cells is passed through a sensing region where optical and electrical signals are generated. A laser beam is used as a light source.Cells to be analysed are prelabelled with flourecsent dyes according to the cellular parameter for eg., cellular antigen or DNA content.Light focused on the cell stream interacts with each cell, excites the fluorescent dye and leads to light scattering and fluorescence. These signals

are electronically amplified and processed by the computer resulting in display of parameter lists or cytograms or histograms. The flowcytometric histogram of DNA content of normal cell is typical.In contrast malignant cells have abnormal DNA content due to chromosomal abnormalities. So, flowcytometry is a straightforward technique to detect abnormal malignant cell population with abnormal DNA content. Low grade tumors are DNA diploid, whereas high grade tumors are DNA aneuploid. DNA aneuploidy is also associated with shorter survival as observed by several workers, for eg., osteosarcoma, chondrosarcoma and ewings sarcoma. Chordoma and adamantinoma show DNA diploidy and better survival.

Electron microscopy-After the advent of immunohi-stochemistry and molecular genetics EM has lost much of its application. But immunoelectron microscopic techniques have been introduced recently which help to identify the histogenesis in metastatic tumors from unknown primary.While reading an electronmicrograph several subcellular elements are studied .Like the nucleus,nuclear membrane, cytoplasm,cell membrane, inclusions/granules,and extra cellular matrix.Electron microscopy has been applied in the different types of chondrosarcoma. It is also useful in round cell sarco mas and in identifying Ewings sarcoma.In difficult cases EM contributes in improving the diagnosis and in understanding the pathogenesis.

Molecular genetics-Karyotyping of tumors and clonal expression of a particular chromosomal anomaly in tumors have helped in understanding the biology of cell transformation in the last 2 decades. But development of cytogenetic studies in the recent years has helped in the diagnosis, assessing prognosis, classifying neoplasia, and in tumor pathogenesis. Tumors can be produced either by a distinct molecular alteration or by widespread genomic alterations which is developed in a multistep cumulative process. The following are the various genomic alterations that occur:

- By eliminating a suppressor gene eg-Rb gene.
- By activating a cellular oncogene
- By fusion of 2 genes resulting in a hybrid gene as a result of translocation or
- By the loss of a DNA repair gene.

Any of these genetic alterations or a combination of a few mutations can transform a normally dividing cell to an abnormally dividing and multiplying tumor cell. Identifying the the genetic alterations indicates the probable response to therapy as in the case of osteosarcoma; or classify/ subtype a particular tumor as in the case of Ewings family of tumors ;or asses prognosis/ dedifferentiation as in the case of chondrosarcoma.

Collection of samples for cytogenetic analysis - 0.5 – 1CM volume tumor tissue from a non necrotic area should be

obtained and placed in a culture medium with all aseptic precautions. The tumor specimen should be transferred to a cytogenetic laboratory at room temperature. Media maintaining stable pH , such as L 15 or HEPES- buffered media are recommended.

Cytogenetic methodologies

- **Karyotypic abnormaities-** This is the coventional cytogenetic method by which all known chromosomal abnormalities in tumors have been identified. It is time consuming ,not easily standardized and requires skilled personnel. Some tumor cells may not proliferate in tissue sections, with a low mitotic index. So metaphase chromosomes may not be available for karyotyping. These limitations have been overcome by in situ hybridization techniques.

- **Flourescent in situ hybridization (FISH)-** In this technique,a known probe of DNA sequence is added to the tumor tissue and allowed to bind with the chromosome regions expected to be rearranged.If positive the probe binds with the corresponding nuclei or chromosomes and visualized as fluorescent spots. FISH is a very sensitive technique and can be performed on interphase nuclei. Another advantage is that it can be done with paraffin embedded material.

- **Comparative genomic hybridization-**This is a recently developed technique in FISH. Here the tumor DNA is used as probe. And the gains,losses and chromosomal segments of tumor cells are studied . The tumor DNA is labled with green fluorochrome and the normal DNA with which it is mixed is labled with red fluorochrome.Both the DNA s hybrid competitively to the metaphases and the ratio of red and green are measured.The fluorescent image is captured, digitalized and processed. Amplification of abnormal genes appear green and deleted regions appear red.a software programme can give the ratio profile of each chromosome pair.This method has the advantage of exploring the whole chromosome material of the tumor. And it does not require mitosis. Most of the Ewing's sarcoma and peripheral neurectodermal tumors show a specific translocation t (11; 22)(q 24 :q 12). This translocation results in a fusion of EWS gene on chromosome 22 and FLI 1 gene on chromosome11 leading to a hybrid oncogenic chimeric protein. Finding this translo-cation helps in the differential diagnosis of small round cell tumors.

Thus cytogenetics plays an important role in the diagnosis of selected cases.The traditional karyotypic analysis was hampered by the difficulty of getting sufficient metaphases in tumors. This is successfully overcome by the recent introduction of FISH technique which an be used both on fresh tissue as well as paraffin embedded tissue. Increasing numbers of available commercial probes have popularized these techniques but it can be done only in a well equipped cytogenetic lab by skilled personnel.

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