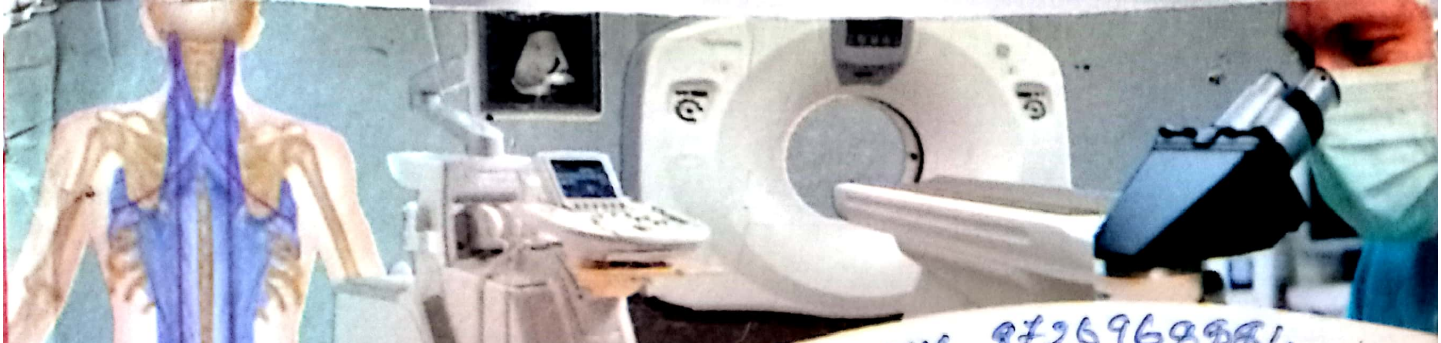

A Text Book For

Roga Nidana

And

Vikruthi Vijnana

Dr. Nisha Kumari

CHAUKHAMBHA ORIENTALIA
VARANASI

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Chapter-1
DOSHA DUSHYADI VIGYANA

1. Definition and Importance of Roganidana

The history of medicine is at the same time very old and very young field of study. It is not a mere history concerned of only historians, but is equally important for medical professionals too. Medical history tells us where we have come from and where we stand, in the art of heeling at present. Several generations of philologists and historians have made source available to us. Truely speaking, medical history is the compass that guides us into the future and shows the direction in which we are marching.

विघ्नभूता यदा रोगाः प्रादुर्भूताः शरीरिणाम्। तपोपवासाध्ययनब्रह्मचर्यव्रतायुषाम् ।।
तदा भूतेष्वनुक्रोशं पुरस्कृत्य महर्षयः। समेताः पुण्यकर्मणः पार्श्वं हिततवतः शुभे ।।

(च.सू. १/७)

Ancient acharyas were always with following of rituals, religious duties like performing of homa, tapa, adhyayana, upavasa and so on. This needs high concentration, strong determination and a sharp vision which was deteriorating gradually. The destraction or deterioration in human ability started once suffering or disease evolved. The physical strength, stamina, life span and overall performance was totally decreased in human which became a big obstackle for their achievement. That obstacle was called as ROGA which gave pain, suffering, and unusual death.

व्याधयो हि समुत्पन्नाः सर्वप्राणिभयंकर। तद् ब्रूहि मे शमोपायं यथावदमरप्रभो।। (च.सू.१/२२)

Roga or vyadhi was prevalent among all living creatures which created havoc and all creation was in verge of complete destruction. Then Maharshi Bharadwaja approached Lord Indra and receieved knowledge of trisutra ayurveda which is the only remedy for all sorts of suffering, pain or roga.

हेतुलिंगौषधानां स्वस्थानुपरायणम्। त्रिसूत्रं शाश्वतम् पुण्यं बुबुधे यं पितामहः।। (च.सू. १/२४)

Definition of Roga

रुजतीति रोगः शरीरयात्रां मृह्नातीति

The one which gives pain or let the body suffer from pain is called as roga. The science which describes roga by means of its cause, pathogenesis, signs, symptoms, the means to diagnose and prediction of prognosis is called as Roga nidana and vikruti vijnana.

The fascinating history of vikruti vijnana/pathology & its many magnificent personalities with their out standing contributions in the opening pages of history of medicine is meant to pay our obeisance to those great personalities who have laid glorious foundations of our specialty.

Life & works of those whose names are seen in history are linked to some disease process-the aim being to stimulate the inquisitive beginner in pathology as to how this colorful specialty emerged.

Every aspect of disease says to know its nature before treatment and for prevention.

"Disease is very old, nothing about it has changed. It is we who changed, as we learn to recognize what was formerly imperceptible."

"Jean Martin Charcot"

Importance of roga nidana

The clinical significance of morphologic & functional changes together with results of other investigations help to arrive at an answer to what is wrong (diagnosis) what is going to happen (prognosis) what can be done about it (treatment) and finally what can be done to avoid a disease, its complication & spread (prevention). Therefore before treatment there are lot many steps to go where knowing rogavijnana becomes really essential.

रोगमादौ परीक्षेत ततो अनन्तरमौषधम्। ततः कर्म भिषक् प्राज्ञानपूर्वं समाचरेत्।। (च.सू.२०/२०)

A disease has to be examined first and then based on the examination appropriate treatment has to be administered. A treatment can be successful only if disease diagnosis is right.

यस्तु रोगमविज्ञाय कर्माण्यारभते भिषक्। आर्थौषधविधानज्ञस्तस्य सियिहच्छया।। (च.सू.२०/२१)

After knowing the science of disease i.e. that is, its etiology, pathogenesis, form, severity and its components the treatment has to be planned. The selection of drug, procedure, dosage, duration etc are all planned after knowing everything about the disease.

नास्ति रोगो विना दोषैर्यस्मात् तस्माद् विचक्षणः। अनुक्तमपि दोशाणां लिंगैर्व्याधिमुपाचरेत्।।

A disease will not manifest without involvement of doshas. The disease will present based on nature of dosha vaishamya. Again doshavaishamya is manifested in the form of lakshanas. A complete knowledge in roga vijnana will pave a way for right diagnosis.

भिषजा प्राक् परीक्ष्यैवं विकाराणां स्वलक्षणम्। पश्चात्कर्मसमारम्भः कार्यः साध्येषु धीमता।।
साध्यासाध्यविभागज्ञो यः सम्यक् प्रतिपत्तिमान्। (च.सू. १०/२१)

A physician should know to examine any disease throughly by its features and later plan the treatment on the basis of its curability. The classification of 8 branches of ayurveds is based on disease forms, disease varieties or mere roga vijnana.

The treatment explained in our classics are to correct the dosha vaishamya. The pathology of dosha vaishamya should be understood before correcting it. Therefore perfect knowledge on roga nidana is required to understand the disease cure the disease & provent the disease.

2. Samanya Nidana and Samany Lakshana of Dosha Vriddhi, Kshaya and Prakopa

Introduction

Doshadi vijnana is the fundamental and most essential science of Ayurveda. The dushti of dosha manifest in various forms i,e : Vriddi, Kshaya, Prakopa, Pradosha, Utlishta, Hrushta, leena avastha of dosha dhatu and, mala in various intensities in various ashayas produce disease.

Various avastha of dosha, its combination with respective dhatu, association with mala, in an ashaya and involvement of srotas that triggers sroto dushti and avayava dushti. Gamana of doshas to various sites, spreading the pathogenesity. All these gives rise to various symptoms. Therefore before knowing any vikruti or vyadhi one should know all viktuta avastha of components of disease. Amongst them Dosha is the primary component.

Dosha Nirukti

- दूषयन्तिमनःशरीरचेतिदोषाः। (सिद्दान्तनिदान, तत्वदर्शिनी)
  One which does dushti of mana and shareera
- "शरीरदूषणाद्दोषाः" (शा.पू.ख्)
  Which undergo vikruti due to any cause and affects shareera
- दूषयन्तिइतिदोष।
  Which does the dooshana of shareera
- दोषाइत्यर्थदुषवैकृत्येइतिदोषधातोर्दृष्यन्त्येभिरितिवाक्येन.....(भा.प्र.पू.ख.)

Dosha Sankhya

- तत्रयःशरीरदोषावातपित्तश्लेष्माणः, तेशरीरंदूषयन्तिः, द्वे पुनःसत्वदोषौरजस्तमश्च, तौसत्वंदूषयतः। ताभ्यांचसत्वशरीराभ्यांदुष्टाभ्यांविकृतिरुपजायते, नोपजायतेचाप्रदुष्टाभ्याम्। (च.शा. ४/३४)
- वायुःपित्तंकफश्चेतित्रयोदोषासमासतः। विकृतअविकृतादेहंघ्नन्तितेवर्तयन्तिच। (अ.ह.सू. १/६)
- रजतमश्चमनसोद्वे च दोषावृदाहृतौ। (अ.ह.सू. १/२१)
- वायुःपित्तंकफश्चोक्तःशरीरोदोषसंग्रहः। मानसः पुनरुद्दिष्टोरजश्चतम एव च। (च.सू. १/५७)
- वातपित्तश्लेष्मण एव देहसम्भवहेतवः। (सु.सू. २१/३)

Shareerika dosha

1. Vata 2. Pitta 3. Kapha

Manasika dosha

1. Satwa 2. Raja 3. tama

General concept of dosha

- विसर्गादानविक्षेपैःसोमसूर्यानिलायथा। धारयन्तिजगद्देहंकफ़पित्तानिलातथा। (सु.सू.२१/७)
- पित्तंपन्नुकफ़ंपन्नुपन्नवोमलधातवः। वायुनायत्रनीयन्तेतत्रगच्छन्तिमेघवत्।। (शा.स.पू.५/२५)
  Visarga- destruction, separation, going away
  Adana- creation, coming together, addition
  Vikshepa- circulation
- All functions in universe are carried out by moon, sun and wind. Similarly kapha, pitta and vata are performing all the functions in living body.
- Pitta is immobile, Kapha dosha is also immobile. Thus the Vata dosha carries Pitta & Kapha where ever necessary just like cloud is carried by wind.

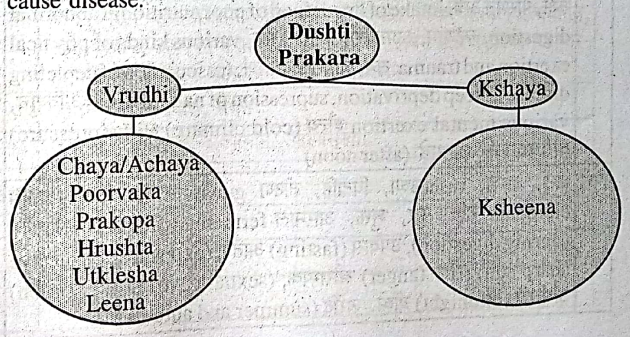
1. विकारोधातुवैषम्यंसाम्यंप्रकृतिरुच्यते। सुखसंज्ञकमारोग्यविकारोदुःखमेवच।। (च.सू. ९/३)
   Disease is caused by dushti or abnormal fluctuation of dosha

beyond physiological limitation. Dosha dushti causes dhatu vaishamya & lead to disease. On the other hand if doshas are in equilibrium then dhatus functions properly leading to health.

2. रोगस्तुदोषवैषम्यंदोषसाम्यमरोगता।। (अ.ह.सू. १/२०)
   Dosha vaishamya causes disease dosha samya causes health.
3. दोषा एव हि सर्वेषां रोगाणां एक कारणम् । यथा पक्षि परिपतन् सर्वतः सर्वमप्यहः।। छायामत्येति नात्मीयां यथा वा कृत्स्नमप्यदः। विकारजातं विविधं त्रीन् गुणान् नातिवर्तते।। (अ.ह.सू.१२/३२)

Vaishamya of dosha is always required for the formation of disease. Dosha dushti and vyadi are always associated and dependent just like the flying bird is always followed with its shadow.

- दुष् वैकृत्ये। (अ.को. ३/४/६)
  Any abnormalcy is called as dushti
- वैकृतं च यस्य यथा स्वरूपं तत् वैपरीत्यम्।
  Fluctuation from normal quality, exhibiting fluctuation from normal quality or exhibition of opposite quality is vikara or vipareeta. This is seen when there is dushti of any component in the body.
- दुष्टत्वम् अशुद्धत्वम् च। (वाचस्पत्यम् १/३६४८)
  Any component in its impure and abnormal form is dushta
- दोषाणामेव अयं स्वभावो यत् दूषकत्वं न धात्वान्तराणाम्। (च.वि. ५/९)
  It is the very basic quality of dosha to undergo dushta and cause disease.



Dosha Prakopaka Hetu

- **प्रकोपः क्षोभे । चांचल्ये।** (वैद्यकशब्दसिन्धु/६९८)
  The one which is aggressive, and having tendency to move is prakopa
- **दुष्टं प्रकर्षेण।** (च.चि.१९/८) The one which is in bad form
- **प्रकृष्टः कोपः प्रकोपः।** (च.सु. १७/११२) The one which tends to move out of anger
- **प्रकृष्टमेव जनयन्तीति प्रकोप हेतवः।** (सु.सु. २१/२०)
- **क्रुद्धं प्रकोपं गतम्।** (सु.सु. २१/३८) Going away with anger or aggression.
- **कुपिता विकृतिमापन्ना।** (मा.नि. १/१४)
  Dosha in prakopavastha have tendency to produce vikruti.
- **विलयन रूपा वृद्धिः प्रकोपः।** (सु.चि. ३३/३)
  It is the avastha in which the doshas increase in quality and quantity so that it gets dissociated from its normal site.
- **स्वस्थानं त्यक्त्वा दोषस्य यत् पुनः मार्गान्तरगमनं स कोप इत्युच्यते।** (अ.हृ. १२/२३)
  Prakopa is that stage of dosha which increases and then spills out from its original place and circulate through channels as result of overflow
- **उत्क्लेश्य प्रकोप्य।** (सु.सु. २०/२०)
- **उदीरणं अधिकं दोषाणाम्।** (सु.उ. १०/४)
- **कोपस्तु उन्मार्गगमिता।** (अ.हृ. १२/२३) gets aggressive and goes else where
- **प्रकर्षेण कोपः स्थानान्तर गति लक्षणः।** (अ.हृ.३/४४) gets aggressive & mores to another place.

दोष प्रकोप कारणा (Causes of dosha prakopavastha)

वात	कषाय, तिक्त, कटु, Rasa रूक्ष, परिणत अन्ने, intake of dry or poor nutrition at abnormal digestion, व्यायाम, अपतर्पण, प्रपतन, भंग, various kinds of physical exertion and trauma. क्षय, जागर, वेगधारणं, (association of depleting disease), sleep deprivation, supression of natural urges अतिशुच, various mental exertion शैत्य (cold climare) क्षोभ, (pressure) वारीधरागमने, अपराह्ने (after noon)
पित्त	कटु, अम्ल, rasa उष्ण, विदाहि, तीक्ष्ण gunas. लवण, तिल, अतसि elements. दधि, सुर, शुक्त, आरनाल fermented fluids. भूक्तेजीर्यति, (during digestion), उपवास (fasting) आतप, (exposure to excesive sharp sun) क्रोध (anger) स्त्रीसम्पर्क, (sexual trive) मध्यान्ने, (noon) अर्धरात्रि (midnight) ग्रीष्मे, शरदि (summer and autumn).

कफ	गुरु, (heavy to digest) मधुर (sweet) अतिस्निग्ध, दुग्ध, इक्षु, दधि milk, milk products, sugar cane products, anoopa mamsa सर्पि (clarified butter) प्रपूरै, दिननिद्र, (sleep during day time) तुहिनपतनकाले, दिवसादौ, भुक्तमात्र, वसन्ते (day time, during food ingestion and during spring).

Aaharaja Nidana

वात	रस	कटु, कषाय, तिक्त
	गुण	रूक्ष, लघु, शीत
	द्रव्य	मसूर, कलाय, यव
पित्त	रस	कटु, अम्ल, लवण
	गुण	तीक्ष्ण, उष्ण विदाहि
	द्रव्य	कुलत्थ वाराहिकन्द लशुन दधि
कफ	रस	मधुर, अम्ल, लवण
	गुण	गुरु शीत स्निग्ध पिच्छिल अभिष्यन्दि
	द्रव्य	कदलीफलं नारिकेलं दधि

Sevana Vidhi

काल	अतिद्रुतं, अतिविलम्बितं
मात्र	अतिमात्र, हीनमात्र, अतियोग of पिप्पली, लवण, क्षार
प्रयोग	भय, शोक, क्रोध, लोभ, मोह, मान इर्ष्या, दु:ख शय्या, प्रजागरै, अनभ्यास - दधि, माष, गव्य, माहिषमांस, मत्स्यं, अपथ्यतम in each वर्ग

Viharaja Karana

वात	वातमूत्रपुरीष शुक्रछर्दिक्षवथुउद्गारवाष्प वेगविधात विषमचेष्टाबलवत्विग्रहात्, अति व्यवाय, अध्ययन प्रपतन प्रधावन अभिघात लंघन प्रतरणरात्रिजागरणभार-हरणगजरथपदातिचर्या अनशन विषमशन अध्यशन
पित्त	आतप सेवा, उपवास, मैथुन
कफ	दिवास्वप्र, आलस्य, अव्यायाम, आस्यसुखा।

Manasika Karana

वात	चिन्ता, त्रास
पित्त	क्रोध
कफ	अचिन्ता

Kala as cause of prakopa:

हेमन्त	शीत, स्निग्धं	कफ चय
वसन्त	उष्णं, स्निग्धं	कफ प्रकोप
ग्रीष्म	लघु, रूक्ष	वात चय
वर्ष	शीतं, रूक्ष अम्लता	वात प्रकोप पित्त चय
शरत्	उष्णं, तीक्ष्णं	पित्त प्रकोप

Aagantuja Karana (External cause)

- **ये भूतविषवाय्वग्निक्षतभंग्रादि संभवाः। रागद्वेषभयाद्याश्च ते स्युरागन्तवो गदाः।**
  आगन्तवोऽपि रोगा दोषकोप अन्तरेणैव उत्पद्यमाना दृश्यन्ते ।
  तस्मात् रोगसम्भवादपिदोषकोपो अयं निश्चितः।। (अ.ह.सू. १२/२३)

External physical agents, toxins or poisonous substances, thermal injuries, injury due to fall or accident, psychological trauma all these can produce dosha prakopa.

चिकित्सा व्यापत् Improper (Improper treatment)

उच्चैः भाष्यं रथक्षोभं अतिचंक्रमणासने। अजीर्णाहितभोज्ये च दिवास्वप्नं स मैथुनम्।।(च.सि.१२/११)

Speaking loudly, travelling in unevenroad or jerky movement, too much mobilization of body, abnormal food habits, sleep during day time. All these if done during panchakarma therapy then it causes dosha prakopa especially vata.

General Features of Prakopa

- **तेषां प्रकोपात् कोष्ठतोद सन्चरणं अम्लिकापिपास परिदाह अन्नद्वेष हृदयोत्क्लेदाश्च जायन्ते।** (सु.सू. २१/२७)

Pricking pain in the koshta, gurgling sound due to movement, sour taste in mouth, excessive thrist, burning sensation in the body, aversion towards food, nausea are samanya prakopa lakshanas.

Vata Vriddi lakshana

- **.....वृद्स्तुकुरुतेऽनिलःकार्श्यकाष्ण्योष्णकामित्वकम्पानाहशकृद्ग्रहान् ।**
  बलनिद्रेन्द्रियभ्रंशप्रलापभ्रमदीनताः।। (अ.ह.सू. ११/५-६)

Increased vata causes physical weakness or turns the body lean. Dark color of skin, patients longs to have warm or hot food. Presence of fine to course tremors, abdominal destension, dryness of stool and thus constipation, the capasity of sense organ decrease. There will be diminision of higher mental function, therefore irrelevant talk and behavior due to disorientation are seen.

Vata Kshaya Lakshana

- **लिंङ्गंक्षीणेऽनिलेऽङ्गस्यसादोऽल्पंभाषितेअहितम्।**
  संज्ञानाशस्तथाश्लेष्मवृद्युत्कामयसम्भवः।। (अ.ह.सू. ११/१५)

Features of decreased vata are: physical debility, general malaise. Patient hates to talk or speaks less, poor sensory function and feature of kapha dosha vriddi are seen.

Pitta Vrudhi and Kshaya Laxanas

- **पीतविण्मूत्रनेत्रत्वक्क्षुत्तृड्दाहाल्पनिद्रताः।** (अ.ह.सू. ११/७)

Discoloration of urine, conjunctiva, skin into yellow color. Excessive hunger and thirst. Burning sensation all over the body. Due to all these discomfort there is less sleep in the patient. These are pitta vriddi lakshanas.

- **पित्तेमन्दोऽनलःशीतंप्रभाहानिः।** (अ.ह.सू. ११/१६)

Pitta kshaya causes diminision of agni, diminision in function of vayu. Cold and clamy body, loss of complexion

Kapha Vrudhi and Kshaya Laxanas

- **श्लेष्मऽग्निसदनप्रसेकालस्यगौरवम्।।**
  श्वैत्यशैत्यश्लथाङ्गत्वंश्वासकासातिनिद्रताः। (अ.ह.सू. ११/७)

Sleshma vriddi causes decrease in quality, intensity and function of agni, excessive salivation, lazyness and heaviness of body. Color of skin turns pale, cold on touch, compact joints. Symptoms of respiratory distress like cough, dyspnoea. Excessive sleep. Are seen in kapha vriddi lakshana.

Kapha Kshaya lakshanas

.....कफेभ्रमः। श्लेष्माशयानांशून्यत्वंहृद्रवःश्लथसन्धिता।। (अ.ह.सू. ११/१६)

Giddiness, emptiness of sleshma sthana, palpitation loose joints.

3. Dosha Dhatu Ashraya Ashrayi Bhava

Introduction

The body is Organised due to permulation and combination or methematical configuration of Pancha Mahabutas. The tridoshas Originated following with utpatti of Dhatus. Later there is a need of interaction between dosha, dhatus for the functional integrity of

the body. The doshas stay in dhatus that means the dhatus provide ashraya to doshas (ashrayi) in specefic areas or sites. The selectivity of ashraya and ashrayi is purely based on sharing similar qulities of mahabhootha between ashraya and ashrayai (dhatus and doshas respectively) eg:- Kapha is predominant of jalamahabhootha, thus naturally it has intimacy towards rasa dhathu which is also predominant of jala mahabhootha.

वायुवाकाश धातुभ्यां वायुः, आग्नेय पित्तम् । अम्भः पृथिवीभ्यां श्लेष्मा । (A.San.Su. 20)

Vata is originated from combination of Vayu and Aakasha mahabuta. Pitta is originated from Agni and Sleshma is originated from Jala and Pritwi.

तत्र वायोर्वायुरेव थोनिः पित्तस्याग्निः कफस्यापः, रक्तं तेजोजलात्मकं मांस पार्थिवं, मेदो जलपृथिव्यात्मकम्, अस्थि पृथिव्यनिलात्मकं मज्जा शुक्रं चाप्यं, मूत्र जलानलात्मकं, पुरीषं पार्थिवम् आर्तवमाग्नेयं, स्वेदः स्तन्यं चाप्यम् (चक्रपाणि- Su.Su. 24/8)

Among the saptha dhatu Rasa is predominant of Apmahabuta, Rakta by agni and jala, mamsa predominant of jala and prithvi mahabootha, Asti made by pritwi and vayu, Majja and Shukra is predominant of jala mahabootha. Mootra predominant of jala, Pureesha by pritwi, Artava and sweda is predominant of agni, Sthanya is dominated by jala mahabuta.

Ashraya; Ashrayi Sambandha

तत्रास्थिनी स्थितो वायुः पित्तं तु स्वेदरक्तयोः श्लेष्मा शेषेषु तेनैषामाश्रयाश्रथिणां मिथः यदेकस्य तदन्यस्य वर्धनक्षपणौपधम् अस्थिमारुतयोनैवं प्रायो वृद्धिहि तर्पणात् । श्लेष्मानुगता तस्मात् संक्षयस्तद्विपर्ययान् । वायुनानुगतः । (A.Hri.Su. 11/26-28)

There is mutual interactin, dependence and influence between ashraya (dhatus) and doshas (ashrayi). It can be eloberated as follows:

1. Vata dosha stays in Asthi Dhatu. (as vayu and Aakasha is common)
2. Pitta dosha stays in Rakta Dhatu (as agni is common)
3. Kapha dosha stays in rasa, mamsa, meda, majja, sukra, mutra and purusha (as jala is common)

This relation will be maintained during the normal physiology. Since the doshas and dhathus are related in the form of adheya and adhara, any changes in any of them will reflect on other in same fashion.

Vriddi (Qualitative and Quantitative increase) is caused due to over nourishment (atisantarpana) and usually associated with kapha. Kshaya (quantitative decrease or qualitative decreases) is caused due to depletion (apatarpana) which is associated with vata dosha. Vriddi of doshas will causes Vriddi of dhatus in which they reside. Similarly kshaya of doshas will also influence dhatus.

1. Vata Vriddi will cause Asthi kshaya.
   Vata Kshaya will cause Asti Vriddi
2. Pitta Vriddi will cause Sweda and Rakta Vriddi
   Pitta Kshaya will cause Sweda and Rakta Kshaya.
3. Kapha Vriddi will cause Vriddhi of rasa, mamsa, meda, majja sukra, mutra, purusha and kshaya of kapha will cause kshaya of the same.

Usually Vriddi of Dosha will cause Vriddi of its dependent dhatus, kshaya of Dosha will cause Kshaya of its dependent dhatus. But there is an exeption to this Rule. Even though vata is residing in asthi, Vriddi of Vata causes Asthi kshaya, and vice Versa. So Vriddi of Vata should be treated with Brunhana and Kshaya of Vata with Langhana with respect to Asthi.

4. Dhatu Kshaya Vriddhi Lakshana

Introduction

- **पूर्वः पूर्वोऽतिवृध्यत्वाद्वर्धयेधिपरंपरम्। तस्मादतिप्रवृध्यानांधातूनांह्रासनंहितं।** (सु.सू. १५/२३)

Increased quantity of a bodily principle gives rise to a similar increase in the quantity of immediately succeeding component in the order of enumeration, hence an increase in any of the fundamental principles of the body should be checked and reduced to its normalcy or normal quantity.

- **तेषांयथास्वंसंशोधनःक्षपणञ्जक्षयादविरुधैःक्रियाविशेषैःप्रतिकुर्वीत।** (सु.सू.१५/१७)

The abnormal excess of the humors and principles etc. of the body should be checked and remedied with cleansing or pacifying measures that would be indicated by their respective natures, so as not to reduce them.

- दोषः प्रकुपितोधातून्क्षपयत्यात्मतेजसा। इद्धः स्वतेजसावन्हिरुखागतमिवोदकम्।(सु.सू.३६/६)

The aggrevated dosha diminishes dhathus by its innate power as kindled fire dries up the water kept in a sauce pan by its own heat.

- दोषादीनांत्वमतामनुमनेलक्षयेत्। अप्रसन्नेन्द्रियंवीक्ष्यपुरुषंकुशलोभिषक्। (सु.सू. १५/३९)

An expert physician should know the disequilibrium of doshaadi (dosha, dhathu, mala) by inference on observing the person's feelings & symptoms.

- क्षपयेद्बृंहयेच्चापिदोषधातुमलान्भिषक्तावद्यावदरोगः स्यादेतत्साम्यलक्षणं (सु.सू. १५/४०)

The healthy should be preserved and in unhealthy the physician should diminish or promote doshas, dhathus, and malas till he becomes free from disorders which is the indicator of equilibrium.

- अतऊर्ध्वंईषत्परिहार्णिर्यावत्सप्ततिरिति। सप्ततेरूर्ध्वंक्षीयमाणधात्वेन्द्रियबलवीर्योत्साहमहन्यहनि।

The dhathus are by themselves incapable of any functions and are made to function by the tridoshas vata, pitta, kapha which are normally residing in them. Thus tridoshas are the activators of dhathus.

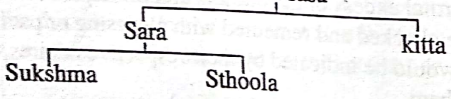
Dhathus

In our body the dhathus are found in two states.

- Asthayi- unstable/circulating.
- Sthayi - Stable/static

1. Asthayi- Essence of food which are required for the nourishment of the dhathus.

Poshakamsa of saptha dhatu present in the Sara paka
↓ reaches
Other dhathus by circulating with rasa dhathu
↓
That is the Ahara rasa
↓
Dhatwagni
↓
Ahara rasa



The poshakamsa is called as poshaka dhathu

1. They are the sthoola dhathus or the gross tissues and they are the poshya dhatus or the one which gets nourishment.

Dhathu Vaishamya and Dhathu Samya

The condition in which the dhathus have normaley in their specific pramanas (quantity), gunas (quality), and karma (functions) is known as dhathu samya. Where as vridhi and kshaya in respect of any of these three aspect is known as dhathu vaishamyatha.

Beyond these 2 aspect of vaishamyatha one more form exists that is vyapath.

If dhatus are neither increase nor decreased. If any presence of visha, krimi and others are found, it is not coming under vrudhi and kshaya

Vrudhi and kshaya is abnormal.
Upachaya and apachaya is normal

Karanas for Vaishamyatha

Dhatu Vaishamya → Doshavaishamyatha
→ Ahithaahara vihara

धातुः धारणात्धातव् :।। पुरुषधारणात्धातव्:।।

- Dhathu means one which supports or withholds the body is called dharana.
- They are the principles of our body and are seven in number - Rasa, raktha, mamsa, meda, asthi, majja, sukra
- They are also called as dushyas because they get dushti by the doshas.
- All the dhatus are getting nourishment from the food. Then they maintain themselves in respect of their Pramana, Guna, Karma.

Dhathu	Lakshanas	Charaka	Sushrutha	Vaghbata
रसक्षय	रौक्ष्य			✓
	भ्रम			✓
	ग्लानि			✓
	शब्दासहिष्णुताहृत्पीडा			✓
	कम्पा		✓	
	शून्यता	✓		

Dhathu	Lakshanas	Charaka	Sushrutha	Vaghbata
	तृष्णा	✓		
	क्षीणा		✓	
	हृदयताम्यता by स्वल्पचेष्ट		✓	
रक्तक्षय	अम्लशिशिरप्रीतिसिराशैथि			✓
	ल्य			
	रूक्षता			✓
	त्वचपारुष्यता	✓		
	स्फुटितत्वचा	✓	✓	
मांसक्षय	अक्षग्लानि		✓	✓
	गण्डिस्फकशुष्कतासन्धिवेद	✓		✓
	ना	✓		✓
	ओष्टशुष्कता		✓	✓
	उपस्थ		✓	
	उरुकक्षवक्षशुष्कता		✓	
	पिन्डिकशुष्कता		✓	
	उदरशुष्कता		✓	
	ग्रीवशुष्कता		✓	
	तोद		✓	
	गात्राणांसदनं	✓	✓	
	धमनीशैथिल्य	✓	✓	
मेद	स्वपनंकटिप्लीहोब्रुधिक्कुशा		✓	✓
	न्डतासन्धिशून्यतारौक्ष्यमेदूर			✓
	मांसप्रणीतसन्धिस्फुट्नग्ला-	✓	✓	
	निरक्षणंआयसतनुउदरक्षीणता	✓	✓	
	ता	✓	✓	
अस्थिक्षय	अस्थितोद			✓
	दन्तशतन	✓		✓
	केशशतन	✓	✓	✓
	नखशतन	✓	✓	
	लोमशतन	✓		
	शम्श्रुशतन	✓		
	श्रमा			
	रूक्षता	✓		

Dhathu	Lakshanas	Charaka	Sushrutha	Vaghbata
	सन्धिशैथिल्य			
मज्जक्षय	अस्थिसौषी			✓
	भ्रम			✓
	तिमिरदर्शनंअल्पशुक्रता		✓	
	पर्वभेद		✓	
	अस्थितोद		✓	
	अस्थिशून्यता		✓	
	दुर्बल	✓		
	लघुनि	✓		
	वातरोगिणि	✓		
शुक्रक्षय	चिरातप्रसिच्येत		✓	✓
	शुक्रशोणितं		✓	✓
	तोद in ब्रुषण			
	मेढ्रधूपन			
	दौर्बल्यं	✓		
	मुखशोष	✓		
	पाण्डुत्वं	✓		
	सदन	✓		
	श्रमं	✓		
	क्लैब्य		✓	
	शुक्रविसार्गक्षीण		✓	
	मेढ्रवेदना		✓	
	अशक्तिमैथुने		✓	
रसवृद्धि	अग्निसदन		✓	✓
	प्रसेक			✓
	आलस्य			✓
	गौरवं			✓
	शैत्यं			✓
	श्वैत्यं			✓
	श्लथांङ्गता			✓

Dhathu	Lakshanas	Charaka	Sushrutha	Vaghbata
	श्वास			✓
	कास			✓
	अतिनिद्राहृद्योत्क्लेशता		✓	✓
रक्तवृद्धि	विसर्प			✓
	प्लीह			✓
	विभ्रतीन्			✓
	कुष्ठ			
	वातास्र			✓
	गुल्म			✓
	उपकुश			✓
	कामल			✓
	व्यङ्ग			✓
	अग्निनाश			✓
	संम्मोह			✓
	रक्तत्वक्			✓
	रक्तनेत्र			✓
	रक्तमूत्ररक्तास्य			✓
	सिरापूर्णत्वं			
मांसवृद्धि	गण्डार्बुद			✓
	ग्रन्थि			✓
	गण्डा			✓
	उरुउदरवृधि		✓	✓
	कण्ठअतिमांसवृधि			✓
	स्फिक्वृधि			✓
	ओष्ठवृधि		✓	✓
	उपस्थबाहुजन्घवृधिगुरुगात्र		✓	✓
	ता			
मेदोवृद्धि	श्रमं			✓
	अल्पचेष्ट gets श्वास		✓	✓
	स्फिक्स्तनलम्बनं		✓	✓
	उदरलम्बनं			

Dhathu	Lakshanas	Charaka	Sushrutha	Vaghbata
	स्निग्धाङ्गतापार्श्ववृधि		✓	
	कास		✓	
	दौर्गन्ध्य		✓	
अस्थिवृद्धि	अधिदन्त		✓	✓
	अध्यस्थि		✓	✓
मज्जावृद्धि	नेत्रगौरवं		✓	✓
	अङ्गगौरवं		✓	✓
	पर्वसुस्थूलमूलानिअरुंषि			✓
शुक्रवृद्धि	अतिस्त्रिकामताशुक्राश्मरी			✓
	अतिप्रातदढर्षावं			✓

Till the age of 60 years, life is best with all the dhathus having full complement and abundance of strength, conferring upon the person the full capacity to withstand every sort of trouble and lead a healthy and active life. With the setting of old age 60 years , the dhathus begin to undergo slow depletion, becoming poor in their quantity, qualities and function.

Significance of Dhathu Vrudhi and Kshaya

- Knowledge of vrudhi, kshaya of dhathus are needed in knowing many diseases

Eg:-1) In pandu, rasa kshaya lakshanas are seen.
2) In arbuda, mamasa vrudhi lakshanas are seen.

- In every disease there will be dosha dushti and dhatu dushti lakshanas. There for knowing a disease is knowing dosha, dhatu vrudhi kshaya lakshanas
- It implies dosha kshaya cannot produce a disease where as dhathuvrudhi as well as dhathu kshaya can produce disease.

Chikitsa Aspect

- Vridha dhatu should be treated with kshapana karma (like ruksha, shodhana, apatharpana)
- Ksheena dhathus are treated by dhathu vrudhikara karma (like tarpana, preenana, rasayana)
- By the pancha karma treatment one can maintain the level of dosha, dhathu that can prevent the disease on set.

5. Mala Kshaya Vriddhi Lakshana

Introduction

- **दोष धातु मल मूलं हि शरीरम्।।** (स.सू. १५/३)

Like दोष's and धातु's, मल's also help in the maintenance of health. Therefore it is considered as moola of sareera.

Definition

मलिनीकरणान्मला

One which contaminates the शरीर is मल.

मल in प्रकृतिअवस्था ⟶ leads to धारणा of शरीर।

If they are contaminated , then leads to वृद्धि/क्षय and causes मलिनीकरणा of शरीर।

मल can be -

1. ब्रहत्मल 2. खमल 3. धातुमल

ब्रहत्मल

1. ब्रहत्मल–घनमल; पुरीष
   द्रवमल, स्वेद, मूत्र
2. खमल-नेत्र विट् (Nasal mucosa, ear wax)
3. धातुमल– पित्त (pitta) is mala of रक्त, कफ is mala of rasa

धातुमल

कफंपित्तंमलंखेषुप्रस्वेदोनखरोमच। नेत्र विट्त्वक् चक्षुषस्नेहधातुनां क्रमशोमल।।(अ.ह.शा.३/६३)

- रस-कफ
- रक्त-पित्त
- मांस-नासिक, कर्णमल
- मेद-स्वेद
- अस्थि-नख, रोम
- मज्जा-नेत्रविट्
- शुक्र-ओजस्

According to वाग्भट ⟶ ओजस् is the मल of शुक्रधातु

According to शारङ्गधर ⟶ He explained मल for all धातु

According to भावप्रकाश ⟶ शुक्र does not have मल

Because as is acted by different अग्नि. At last when formed it is pure, so there is absence of मल for शुक्र

In शरीरपक्रिय ⟶ परिणाम of आहार

- सारभाग → उत्तरोत्तर धातु पोषण → If वृद्धि or क्षय leads to → रोग
- किट्टभा → eliminated through 13 vegas → if not eliminated leads to आम → आम which combines with दोष and धातु → Leads to आम दोष ⟶ रोग

Elimination of मल through 13 vegas

- वात-अधोवात-flatus
  उर्ध्ववात-belching
- विट-feces
- मूत्र-urine
- क्षवथु-sneeze
- तृट्-thirst
- क्षुधा-hunger
- निद्रा-Sleep
- कास-cough
- श्रमश्वास-breathing on exertion
- जृम्भा-yawning
- अश्रुज-tears
- च्छर्दि-vomiting
- रेतस्-ejaculation of शुक्र

Contents of elimination

- Undigested food material
- Residual products
- Toxins, other bio-chemical and microbial waste
- Heat

क्षय of खमल

- **मलानंअतिसूक्ष्माणांदुर्लक्ष्यंलक्षयेत्क्षयम्। स्वमलायनसंशोषतोदशून्यत्वलाघवैः।।**(अ.ह.सू.११/२३)
- Decrease of मल's which are सूक्ष्म are to be assessed from शोष, तोद, शून्यत्व, लाघवता- felt in their respective मलायन's

वृद्धि of खमल

- **....एवंचलक्षयेत् । दूषिकादीनापिगलान्वाहूल्यगुरुतादिभिः।।** (अ.ह.सू. ११/१४)

The state of increase of other malas such as दूषिक (विट् of eye) are to be assessed from their increase and heaviness felt along the respective मलायन's

क्षय of pureesha :-

पुरीष- हृदयपार्श्वपीडः सशब्दवायोरुर्ध्वगमनं कुक्षोसंचरणम् (सु.सू. १५/११)

Produces pain in chest planks, flatus, upwardmovement of vayu, circulation of vayu in Kukshi.

According to अ.हृ.सू. ११/२१

- **पुरिषेवायुरन्त्राणीसशब्दोवेष्टयन्निव । कुक्षोभ्रमतियात्यूर्ध्वंहृत्पार्श्वेपीडयन्भृशम्।।**

According to सु.सू. १७/७०

- **क्षीणोशकृतिचान्त्रणिपीडयन्निवमारुतः। रुक्षस्योन्नमयन्कुक्षिंतिर्यगूर्ध्वंचगच्छति।।**

मूत्र kshaya lakshana :-

- **मूत्र क्षये बस्तितोदो अल्पमूत्रता च।।** (सु.सू. १५/१५) Decrease of मूत्र causes pricking pain in bladder ans seanty micturition.

According to अ.हृ.सू. ११/२२

- **मूत्रअल्पंमूत्रयेत्कृच्छ्राद्विवर्णांसास्रमेववा।।** Seanty urine, difficulty in passing urine due to pain discoloration of urine, blood in urine.

According to च.सू. १७/७१

मूत्रकृच्छ्र	
मूत्रविवर्णाता	तृष्णाधिक्यत
	मुखशुष्कता

स्वेद (सु.सू. १५/१५) Kshaya Lakshana

स्तब्धरोमकुपता	
त्वक्शोष	स्पर्शवैगुण्य
	स्वेदनाश

According to अ.सू. ११/२१

- **स्वेदोरोमच्युतिःस्तब्धरोमतास्फुटनंत्वचः।।**

पुरीष वृद्धि :-

- **पुरीषमाटोपंकुक्षोशुलंच।।** (सु.सू. १५/२०)

According to अष्टाङ्ग हृदय ११/१३

आध्मानआटोपगौरव वेदनाशकृत्

मूत्र वृद्धि :-

मूत्रवृद्धिमुहुर्मुहुप्रवृत्तिबस्तितोदआध्मान

According to- astanga

मूत्रंतुबस्तिनिस्तोदंकृतेअप्यकृतसंज्ञताम्। (अ.हृ.सु. ११/१३)

स्वेद-Vriddi lakshana

स्वेदस्त्वचोदौर्गन्ध्यंकण्डूंच।। सु.सू. १५/२०

According to- (अ.हृ.सू.११/१४)

अतिस्वेददौर्गन्ध्यकण्डू

चिकित्सा

- Diseases due to पुरीषवृद्धि ⟶ pacified by अतिसारचिकित्साक्रिया
- Diseases due to पुरीषक्षय ⟶ pacified with meat taken from middle part of the body of मेष, अज and intake of यव, माष, राजमाष
- Diseases due to मूत्रवृद्धि ⟶ treated in the lines of प्रमेहचिकित्सा
- Diseases due to मूत्रक्षय treated by इक्षुरस, मण्ड, द्रव, मधुररसअम्ल-लवणरस leads to उपक्लेद
- Diseases due to स्वेदक्षय ⟶ treated through व्यायाम, अभ्यङ्ग, स्वेद
- Diseases due to स्वेदवृद्धि ⟶ शीतवीर्ययुक्तद्रव्य's

Importance

मल also acts as media for elimination process like -

- In विरेचन where the पूरीष acts as media for expulsion of dushta dosha
- In स्वेदन where sweat acts as media for elimination of dosha
- **वृद्धिमलानांसङ्गाच्चक्षयंचातिविसर्गतः।**
- **मलोचितत्वाद्देहस्यक्षयोवृद्धस्तुपीडनः।।** (अ.हृ.सु. ११/२५)

The decrease of mala can be accessed through excess elimination and increase can be identified by undesirable accumulation.

The increase is not considered as trouble as the decrease because the body being accustomed to accumulation of waste products.

6. Hetu, Bheda and Lakshana of Agni Dushti

Agni is the principal component of the body for every physiology. The metabolism, catabolism, transformation, digestion, destruction of toxins all are brought about by agni. In short agni is life, when agni is lost there will be an end of life. Its functions at various levels and intensities bring normal continuity of life.

आयुर्वर्णो बलं स्वास्थ्यमुत्साहोपचयौ प्रभा। ओजस्तेजोऽग्नयः प्राणाश्चोक्ता देहाग्निहेतुकाः।।१।। शान्तेऽग्नौ म्रियते, युक्ते चिरं जीवत्यनामयः। रोगी स्याद्विकृते, मूलमग्निस्तरुमाग्निरुच्यते।।२।।

(ca. chi. 15)

As long as agni functions normally inside the body the person continues to live. Normal and optimized property of agni provide good health, life span, strength, nourishment. The goodness of agni is expressed by proper prabha, varna and right functioning of ojus. Ojus gives vyadhikshamatwa, utsaaha and good growth of body with energy. If the functioning of agni is deteriorated, all the goodness in the body will be lost. The body gets open to various diseases and lifc ends due to deterioration.

अन्नस्य पक्ता सर्वेषां पकृणामधिपो मतः। तन्मूलास्ते हि तद्वृद्धिक्षयात्मकाः।।३।।
तस्मात्तं विधिवद्युक्तैरन्नपानेन्धनैर्हितैः। पालयेत् प्रयतस्तस्य स्थितौ ह्यायुर्बलस्थितिः।।४।।

Agni digests the food when in optimum stage. Due to various reasons there may be fluctuation in nature or intensity of agni in the form of vriddi, kshaya or agni vishamata. Therefore to compensate these fluctuation one has to follow proper anna sevana vidhi to maintain agni.

A good agni only can provide energy, good health and long life.

Hetu of Agni Dushti

अत्यम्बुपानाद्विषमाशनाच्च संधारणात्स्वप्नविपर्ययाच्च।
कालेऽपि सात्म्यं लघु चापि भुक्तमन्नं न पाकं भजते नरस्य।।५।।
ईर्ष्याभयक्रोधपरिप्लुतेन लुब्धेन रुग्दैन्यनिपीडितेन।
प्रद्वेषयुक्तेन च सेव्यमानमन्नं न सम्यक्परिपाकमेति।।६।।

Following are the causes of alteration in the intensity, quality and functioning of agni:

1. **Excessive water intake:** Jala is drava guna and sheeta which posses opposite quality of agni. Therefore excessive water intake before and soon after meal is contraindicated.
2. **Untimely food intake:** Agni has its biological time to get intensified. Otherwise it stays latent or inert. Food intake, quantity, quality of food all must be in accordance to agni. Otherwise the quality of agni changes. For example if one takes more food when agni is in less intense then there will be incomplete digestion as the available agni is not sufficient to digest huge or bulk quantityof food. Similarly consuming food before digestion of previous food, late consumption of food all these causes agni dushti.
3. **Vegadharana:** Suppression of natural urge especially kshudha, pipaasa, nidra causes agni dushti.

4. **Nidra viparyaya:** sleep is body's natural compensatory mechanism to revitalize the body from fatigue and exhaustion. The body needs time for relaxation from continuous work. This is achieved from sleep. Sleep deprivation decreases the quality of agni.
5. Psychological causes like hatredness, anger, fear, grief

Dosha vaishamya causes disease and the same causes agni vaishamya. Long standing disease will also deteriorate the quality of agni.

Classification of Agni Dushti

मन्दस्तीक्ष्णोऽथ विषमः समश्चेति चतुर्विधः। कफपित्तानिलाधिक्यात्तत्साम्याज्जठरोऽनलः।।७।।

There are 4 varieties of agni

1. Mandagni - Dominated by kapha dosha
2. Tikshnagni - Dominated by Pitta dosha
3. Vishamagni - Dominated by Vata dosha
4. Samagni - A normal agni doing proper pachana with the involvement of all the three doshas in samavastha

विषमो वातजान् रोगान् तीक्ष्णः पित्तनिमित्तजान्। करोत्यग्निस्तथा मन्दो विकरान् कफसंभवान्।।८।।

Vishamagni leads to vata vikaras, tikshnagni leads to pittaja rogas and mandagni causes diseases of kapha dosa.

आमं विदग्धं विष्टम्भं कफपित्तानिलैस्त्रिभिः। अजीर्णे केचिदिच्छन्ति चतुर्थं रसशेषतः।।९।।
अजीर्णं पञ्चमं केचिन्निर्दोषं दिनपाकि च। वदन्ति षष्ठं चा जीर्णं प्राकृतं प्रतिवासरम्।।१०।।

The disease caused by agni dushti is primarily named as ajeerna. Ajeerna is a state of abnormal incomplete digestion. This ajeerna causes formation of ama. Ama is moola for all rogas. Ama jeerna, Vidagdhajeerna, Vishtabdajeerna is caused by Kapha, Pitta, Vata respectively 4th variety is Rasasheshaajeerna 5th variety is दिनपाकी अजीर्ण 6th variety is Normal jeerna.

Lakshanas of Amajeerna

तत्रामे गुरुतोत्क्लेदः शोथो गण्डाक्षिकूटगः। उद्गारश्च यथाभुक्तमविदग्धः प्रवर्तते।।१०।।

Amajeerna is dominated by kapha dosha there fore there is incomplete digestion and stasis of food particals. This leads to gurutwa, or fullness. Excessive accumulation of kleda, manifestation of sotha in ganda and akshi koota are present in amajeerna. Due to stasis of food in stomach the udgara smells like that of ingested food

Lakshanas of Vidagdhajeerna

विदग्धे भ्रमतृण्मूर्च्छाः पित्ताच्च विविधा रुजाः। उद्गारश्च सधूमाम्लः स्वेदो दाहश्च जायते।।११।।

Giddiness, thirst, loss of consciousness and various pittaja lakshanas like osha, chosha. Patient will have sour and fuming sensation in belching, excessive sweating and burnig are features of vidagdhajeerna.

Lakshanas of Vishtabdhajeerna

विष्टब्धे शूलमाध्मानं विविधा वातवेदनाः। मलवाताप्रवृत्तिश्च स्तम्भो मोहाङ्गपीडनम्।।१२।।

Pain and abdominal distension, features of vata prakopa like toda, bedha. Absence of elimination of mala, adhovata due to sthambha or stagnation. Diminished higher mental functions, pain all over body are features of vistambhajeerna.

7. Definitions and Samanya Lakshana of Ama

- **परिणामतस्त्वाहारस्त गुणाः शरीरगुणभावमापद्यन्ते यथास्त्वामविरुद्धाः।**
  विरुद्धाश्च विहन्त्युर्विहिताश्च विरोधिभिः शरीरम्।। (च.शा. ६/१६)

Digestion is nothing but the process of transformation. During the process of transformation the gunas of ingested aahara should turn homologous with shareera gunas and turn as part of shareera bhavas if they are not having viruddha gunas. If they are viruddha to shareera gunas then they act against dhatus during the process of transformation.

This results in the onset of various abnormal decaying process in the body. This decaying process is nothing but impact of ama production. In the process of digestion pitta takes active roll where as others passive roll. Pitta can be taken as ushma

If ushma diminishes then the undigestion, maldigestion, improper and incomplete digestion, absorption, excretion takes place.

- **जाठरेणाग्नि रसः कट्भावेन कृत एव। किंतु धात्वग्निभिरपाकादाम इत्युच्यते।।**
  (डल्हण) (सु.सू. १४/३२)

When the food is not digested completely the nutrient remain in large chains or large molecular weights. They are non permissible through villi and hence remain unabsorbed. The undigested diet fibers may pass as loose stools but the dhatwagni mandhya produces biochemical wastes which are non homologous to body, but does not get excreted. This stasis causes various kinds of ailments. The component that stays undigested and non excreted is called as Ama.

दुष्यत्यग्निः, स दुष्टोऽन्नं न तत् पचति लघ्वपि ।अपच्यमानं शुक्तत्वं यात्यत्रं विषरुपताम् ।।
(Ca.Chi. 15/44)

The agni in Koshta will undergo dushti and convert the ingested anna into dushta anna. This type of agni and anna will not undergo further digestion and does not attain finality. This undigested anna will stay in amashaya to undergo fermentation. After this procers the anna attains visharupa.

It is a state where a substance undergo process of paaka or transformation improperly or incompletely without attaining finality; either in the form of catabolism or anabolism. The remnant residual bye product of improper digestion can be names as ama.

ऊष्मणो अल्पबलत्वेन धातुमाध्ययमपचितम्। दुष्टमामाशयगतम् रसमाम प्रच्क्षते। (a.hru.Su13/25)

Pitta which is in ushma roopa attains alpabala and agni diminishes. This results in incomplete digestion. If digestion is not proper or complete the ahara rasa is not produced which causes diminision of adhya rasa dhatu. This undigested improperly digested compound is no more fit for further transformation or absorption. Thus it is called as dushta which stays in amashaya to associate with dosha, dhatu, mala, avayava and produce various diseases. This is called as Ama.

- **न तु आमाशयस्थ कायाग्नेर्दौर्बल्यदविपाचितः।**
- **.....इत्यादिनोक्तः तस्य रोगहेतुतथा आमशयत्वेन च।**
- **एतवता धातुभूताग्निनां मान्ध्यत्वे अनामसम्भवात्।**
  शोषवृणविद्रध्यादि रोगाणां तज्ज्यत्वमुक्तं भवति।। (वाचस्पति)

Interpretation of Ama

- Ahaara which is not properly digested. Which is stagneted.
- Annarasa not properly formed in amashaya due to impaired kaayagni
- Imperfectly digested intestinal contents
- Residual aahara rasa after absorption.
- The first phase of dosha dusti or dosa dushhya sammorchana.
- Vrina or vidhradhi until it gets pakwavastha (inflammatory process)

Causes of Production of Ama

1. **Agnimandhya:-** Impaired kayagni, dhatwagni. Agni not merely digests the ingested food but also transforms them into such state where the body has to accept the absorbed food into tissue elements. There may be thousands of

properties in ingested food but all are converted into single form that is homologous to body. The foreign particles, micro organisms, toxins are all detoxified at every level of the gut. When agni diminis has ama can be produced.

2. Dosha avastha:- state of doshas

समप्रकोपौ दोषाणां सर्वेषामग्निसंश्रितौ तस्मादग्निं सदा रक्षेत्। (Ca.chi.5/156)

1) Vaata:
- Stimulation to nervous system
- Closure and opening of sphincters
- Initiation of peristaltic waves
- Muscular contraction of stomach
- stimulation to glands to secrete secretions
- Absorption of digested material and separation of waste.

2) Pitta : HCL, bile, pancreatic juice, gastric enzyme, salivary enzyme, all that is responsible for breakdown of food partials from complex to simple and homogenous state called Chyme is done by pitta.

3) Kapha: Gastric mucosa to compensate high acidic or alkaline nature of digestive juices and prevent corrosion.

- It also felicities food propulsion by lubrication.
- Change the consistency of food
- Felicitate transportation and absorption
- Thus the dushta avastha of any single above dosha or their combination definitely interrupts the mechanism of digestion.
- Vriddi, kshaya, dushti of doshas may bring up formation of Ama leading to toxic manifestation from the end products of digestion.
- This toxic undigested harmful product leads to agnimandhya and produce Ama.

3. Status of avayava

Structural abnormality of site of digestion- Grahani, amashaya e.g - stricture of CBD, gallbladder, obstructed gall stones, Ca of head of pancrease, duodenal ulcer, inflammation etc. Any structural or functional disturbance in stomach, duodenum disturbs the normal digestion & results in production of undigested substance.

4. Previous excess accumulation of metabolites

The anabolic and catabolic phenomenon goes on continuously among the dhatus and avayavas. As a result large amount of waste

products in the form of sthoola and sookshma malas are produced. Every biochemical reaction ends up with byproduct with bio waste which has to be excreted or disposed off regularly and efficiently. If person does vegadharana they remain inside the body which are harmful and toxic taking the form of Ama.

- Stages of Ama :-

अपच्यमानं शुक्तत्वम् यावत्यन्नं विषरूपताम् (च.चि. १५/४४)

Ingested food
↓
Mandagni
↓
Apakwa Ahara rasa
↓
Stasis : as it is unabsorbable & not transportable to next phase
↓
gets shuktatwa on stasis
↓
Ama rasa
combines with dosa to cause disease | **on further stasis**
↓
Ama Dosha
acquires toxicity | getting more shuktatwa, visid on stasis
↓
(Concentrated form)
Ama Dosha
↓
Ama Visha

यथान्नमपि तेनैव अग्निना पक्कममृततां याति अपक्कम् च विषताम्।

Mode of formation & Ama Swaroopa

1. Undigested annarasa will have Dravatwa (fluidity)
↓
Attain Gurutwa + Snigdhatwa

↓

Piccilatwa (increassed viscosity)

↓

(further stasis)

↓

Tantumaya (further increased visidity)

↓

The tantumat anna rasa (ama) does not undergo further transformation. Hence it remains apakwa

↓

Undigested and Asamyukta (non acceptable or does not get combined with any for transformation)

↓

Further increased viscosity & Putrification causing Dourgandya

↓

Fermentation & Putrification

↓

continues to attain Abhishyanda(murtha rupa)

↓

Sarva Shareera sanchara + Combination with avayava

↓

SROTORODHA leading to →VYADHI

Stasis of anna rasa leads to fermentation and increased concentration with impact of time, enzymes, secretions and abnormal chemical reactions. The matter turns unacceptable to tissue ellement. The continuation of stasis causes toxicity. "Thus undigested Annarasa possessing odor with high viscosity causing gatra, shaithilya is called "Ama" which produce-

- Dosha dushti first
- Dhatu pradosha next
- Combine with element and circulates in srotas
- Srotodushti
- Manifestation of vyadhi

Ama can take place in bhootagni level or jataragni level or dhatwagni level.

Location of Ama & Manifestation of Symptoms

1.	Amashaya	Chardi, hrillasa, amlapitta ajeerna, sula, aruchi
2.	Pacchamanashaya	Grahini, sula, adhmana, atopa, alasaka, kaamala
3.	Pakwashaya	Atisara, pravahika, vishuchika, anilamoodata
4.	Asthi Sandhi	Amavata, vata rakta krostuka-sheersha
5.	Twak, Maamsa	Sheetapitta, udardha, kota, kusta, visarpa
6.	Marma	Pakshagata (पक्षाघात), stroke, embolism (shiras)
		Hridroga, pulmonary embolism (hridaya)
		Ashmari, Avila motra (basti)
7.	Sira Dhamani	Dhamani praticaya therosclerosis), varicose veins
8.	Higher mental function	Cerebral hypoxia, poor concentration, lethargy, drowsy, un satisfaction
9.	Respiratory system	Kaphaja kasa, shwasa
10.	Haemopoitic system	Paandu, Kaamala
11.	Reproductive system	Loss of libido, infertility
12.	Muscular system	SLE, rheumatic fever, myopathy

General features of Ama

- क्षुन्नशो हृदयाशुद्धिः तन्द्राजटर गौरवै दोषप्रवृत्तिर्नो यक्षव्याधिभ्यान्वितं वरेत् (वन्गसेन २२/५३१) स्त्रोतोरोध बलभ्रंश गोग्रवानिलमूढताः। आलस्य अपक्ति निष्ठीव मलसंघारुचि क्लमाः।।

Loss of hunger, unclear chest, lassitude, slow bowel and heaviness, dushti of dosha causes various disease. Absence of dhatu poshana.

Srotorodha : Due to piccila, abhisyandi and tantumat swabhava amd sticks to the wall of circulation and slows down circulation initially. Later on there is manifestation of obstruction. Sroto rodha causes many disease and discomfort.

Balabramsha : Due to agni mandhya and ama the ahara rasa and adhya rasa dhatu is not properly produced. Due to lack of uttarottara dhatu poshana there is balakshaya or decrease in strength and stamina.

Gourava : Due to stasis of past food in incomplete form, sluggishness in circulation, obstruction of srotas and of doshas, malasanchaya there is increase in heaviness of body along with alsaya.

Anila moodhata : Cala guna of vata gets hyper activated and disoriented.

Apakti : Agni mandhya and presence of previously ingested and undigested food will cause indigestion further. The mixture of such compound leads to abhishyandi and utklesha. This results to nisteevana or excessive salivation.

Agni dushti also causes aruchi, abnormal incomplete stool formation. Absence of nutrition and Accumulation of ama causes klama.

8. Sama and nirama Dosha, Dushya Lakshana

Ama produced in amashaya gets associated with doshas independently or coolectively. This condition is called as Sama doshavastha.

Sama Vata Lakshana

वायु सामोविबन्धो अग्निसादस्तन्द्रान्त्रकूजनैः। वेदनाशोफनिस्तोद क्रमशो अंगानिपीडयन्।।
विचरेद्युगपच्चापि गृह्णाति कुपितो भृशम्। स्नेहाद्यैर्वृद्धिमाप्नोति सूर्यमेघादयो निशि।। (अ.स.सु. २१)

Due to anila moodhata and rookshata there is vibhanada or non excretion of malas and gurgling sound in abdomen. Agni dushti and indigestion continues. Vata in dushta avastha with ama circulates haphazardly and aggressively in srotas. It reaches asthi and sandhi to produce pain, stiffness and swelling. There is aggravation of symptoms if oil massage, or snehana karma is done. It also aggravates during early morning time, cloudy climate and during night hours.

Nirama Vata Lakshanas

निरामो विशदो रूक्षो निर्विबन्धो अल्पवेदनः। विपरीतगुणैः शान्तिं स्निग्धैर्य।

The normal functions of vata are clearly seen, dryness of body,

less pain or less sick symptoms. The quality opposite to vayu that is snehanadi karma can be administered in nirama vata stage.

Saama Pitta Lakshana

दुर्गन्धम् हरितं श्यावम् पित्तम्लम् सरम् गुरु। अम्लीक कण्ठ दाहकरम् साम विनिर्दिशेत्।।

Foul smell from body, oral cavity, feces emerge. The discoloration of stool, vomitus will be green or dark color. There is increase amlata or acidity in the stomach. Stasis of undifested food gives guruta (heaviness). Sour belching, burning epigastrium are features of sama pitta.

Nirama Pitta Lakshana

आताम्रपीतमत्युष्णं रसे कटुकमस्थिरम् । पक्वम् विदग्धम् विज्ञेयं रुचिपक्तिर्बलप्रदम्।।

The normal color of pitta is gained that is peeta varna from harita, haridra varna. Pitta attains its ushmata. Amlata is optimized from excessive sourness. Atiamlata is now replaced by katuta. The accumulated pitta during amavastha starts to move or sarata is achived. Vidagdhata due to ajeerna is repaired by samyak pachana. When pachana is attained aruci disappears. Patient feels ruchi and consumes food. Consumed food is digested normally and this leads to normal dhatuposhana. When there is poshana to the body by dhatus, bala is naturally gained.

Saama Kapha Lakshana

आविलस्तन्तुलः स्त्यानः कण्ठदेशेअवतिष्ठति। सामे बलसो दुर्गन्धो क्षुदुद्गार विघातकृत्।। (मधुकोष)

Kapha turns abnormally sticky, gumy and thready. Such kapha goes to kanta desha (throat or laryngeal region). This produces durgandha of oral cavity and obstruction to hunger reflex (because stomach is full of ama and kapha), obstruction to belching.

Nirama kapha lakshana

फेनवान् पिण्डितः पण्डुनिःसारो अगन्ध एव् च। पक्वः स विज्ञेयश्छेदवान् वक्त्रशुकृित्।।
(मधुकोश व्याख्या)

The hard and dense form of kapha is turned to light and frothy form which is its natural form. It achieves its original mass form, original pale color (mucoid form turns to mucus form) and clear non polluted form. There is clear throat, due to which there is absence of foul smell. Person regains normal digestion and no accumulation or excessive saliva is seen. Thus there is vaktra shuddi.

Conclusion

- In ahara parinamakara bhavas pridominently agni is essential for paka of aahara, visha, oushada.
- Agnimandya leads to apaka of all above.
- The reminant, residual bye product of improper digestion is Ama
- Ama stasis brings shuktatwa and undergoes putrifaction, combines with body elements.
- Ama with dosa dhatu circulates all over body. Where there is weakest point in the body it stays there to produce disease.
- Ama not only brings disease but also fastens the aging process, decreases immunity, hampers normal wel being.

9. Dosha Paka and Dhatu Paka Lakshana

दोषपाक

It is a form of vyadhi avastha where doshas attain pakwata and reverse to their prakrutavastha with the advent of samagni.

दोषपाक अवस्था : It is a stage favorable to treatment of disease because aama gets separated from doshas.

- **दोषप्रकृतिवैकृत्यं लघुताज्वरदेहयोः। इन्द्रियाणांचवैमल्यं दोषाणांपाकलक्षणम्। ।**(मा.नि.२/६६)

This condition is stated to be nirama stage of dosha as a result, diseases either cures completely or symptoms start diminishing gradually or agitated doshas starts coming towards koshta. It is essential stage of recovery of disease.

During dosha paka, certain symptoms will manifest.

- The symptoms of the doshas involving in the development of disease start diminishing.
- In case there was fever or incresed temperature of affected part, it starte subsiding.
- As the doshas and aama starts separating from dhatus and srotases, the body is releived from heaviness and laghuta or sense of relaxation is produced.
- In the dusta state of doshas and aama, the desire of sense organ to respond to sensation remain depressed, but in the stage of dosha paka, this depression is removed. The sense organ gets refreshed and become ready to respond to the sensation.

धातुपाक

On the contrary dhatupaka is pathological and worsening stage of disease.

- **धातुपाकाद्धन्ति, मलपाकाद्विमुञ्चतीतिव्यवस्थितविकल्पः, धातुमलपाकविकल्पेचदैवमेवहेतुः। उत्तरोत्तरोगवृद्धिबलहानिभ्यांशुक्रादिधातुसहितमूत्रादीनांचधातुपाकोज्ञेयः, अन्यथातुमलपाकः; यदुक्तंनिद्रानाशोहृदिस्तम्भोविष्टम्भोगौरवारुचि । अरतिर्बलहानिश्चधातूनांपाकलक्षणम्।।**

(मा.नि. २/६६-७३ (मधुकोश)

धातुपाक & दोषपाक are the two different process responsible for the prognosis of diseases. in dhatupaka the symptoms manifested are sleep deprivation, depression of cardiac activity. sluggishness in circulation and slow elemininationn due to abnormal metabolism. the bstasis of metabolic waste produces heavyness and pain in the body. As the dhatus are diseased the poshana karma to the body is absent. hence there is balakshaya.

In dhatu paka all the preceeding dhatus cause increase in symptoms of disease. Due to dhatu paka the poshana to be produced by dhatus are absent. Therefore there is bala hani.

10. Concept, classification, diagnosis and general complications of Avarana

आवरियते देह चैतन्य आवरणे आच्छादन साधन मात्रे। (VACHASPATI)

- The word 'Avarana' means to envelop, to mask, to obstruct, to overlap or to cover.
- Entity which gets obstructed is called Avarya which obstructs the Vata is Avaraka.
- In Avarana process either visible or invisible entities are in Avaraka form.
- Avarya is always invisible i.e. Amoorta Vayu.
- The obstructing entity (Avaraka) will be strong enough to diminish the functions of obstructed entity (Avarya).

Synonyms of Aavarana

- Anvita
- Baddhamarga
- Pratighata
- Samanvita
- Samishraha
- Samyukte
- Samvita
- Uddhuya
- Upastambhita

Charectars of Avarana

''यस्य यस्य कर्मवृद्धिः स आवरको, यस्य यस्य च कर्महानिः स आवृत'' इति।
अतः प्रकोपानन्तरं वायुः आवरणं करोतीति संबद्यते। (जेज्जट च.चि. २८/२१८)

The dosha which enhances its karma is the avaraka, by virtue of which the avruta wil have diminished functioning. So as to say, vata after its prakopa causes avarana.

Pathophysiology of Avarana

धातुक्षयात् इति सारक्षयात्। मार्गावरणेन वेगप्रतिबन्धात् एव कुपितो भवति।
अथ भवतु मार्गावरोधात् वातकोपः, आवरकेण तु पित्तेन कफेन चास्य कथं न संबन्धो भवतीत्याह-वातपित्तेत्यादि। त्रितयमेलकेऽपि वायोः प्रधान्यमाह-वायुरेव हि सुक्ष्मत्वात् इति; सुक्ष्ममार्ग अनुसारितया प्रेरकत्वात्। (चक्र.च.चि. २८/५६-६०)

Dhatu kshaya refers to sara kshaya, there will be vata prakopa due to marga avarana.

Sequele of Avarana

एषां स्वकर्मणां हानिः वृद्धिः आवरणे मता। (च.चि. २८/२१६)

Whenever there is increased or decreased functioning of vata, avarana may be inferred.

Due to overpowering of avaraka dosha, avruta dosha functioning will be limited or reduced, on the other hand, avaraka dosha wil exhibit vriddi lakshanas.

Classification of Avarana

According to	Amoorta	Moorta	Paraspara	Total
Charaka	20	11	11	42
Vagbhata	20	12	10	42

Charaka - 42+5 types of pittakapha (mishra) avarana = 47

1. Anyonya varana (Amurta Avarana) - (20) 12 vata by vata.
2. Samanya avarana (Murta avarana) - (22) vata by pitta, kapha, dhatus, malas & anna.

Doshavrita vata -	2
Doshavrita pancha vata -	10
Dhatwavrita vata -	6
Sarvadhatwavrita vata -	1
Malavrita vata -	1
Mutravrita vata -	1

Annavrita vata - Innumerable (Cha. Chi.28/227, As. Hri.Ni.16/30).

Samanyavarana

1. Pittavruta vata
2. Kaphavruta vata
3. Raktavruta vata
4. Mamsavruta vata
5. Medavruta vata
6. Astavruta vata
7. Majjavruta vata
8. Sukravruta vata
9. Sarva dhatwavruta
10. Annavruta vata
11. pittavruta prana
12. pittavruta udana
13. pittavruta samana
14. pittavruta vyana
15. pittavruta apana
16. kaphavruta prana
17. kaphavruta udana
18. kaphavruta samana
19. kaphavruta vyana
20. kaphavruta apana
21. Mootravruta vata
22. purishavruta vata

Anyoonyavarana

1. Pranavruta udana
2. Pranavruta vyana
3. Pranavruta samana
4. Pranavruta apana
5. Udanavruta prana
6. Udanavruta vyana
7. Udanavruta samana
8. Udanavruta apana
9. Vyanavruta prana
10. Vyanavruta udana
11. Vyanavruta samana
12. Vyanavruta apana
13. Samanavruta prana
14. Samanavruta udana
15. Samanavruta vyana
16. Samanavruta apana
17. Apanavruta prana
18. Apanavruta udana
19. Apanavruta vyana
20. Apanavruta samana

Lakshanas of Avarana

Avarana	Lakshana	Treatment
Pittavruta vata	Daha,trishna,sula, bhrama, tama,vidaha by taking katu amla lavana ushna and sita kamita	Shitamushna kriya, Jivaniya ghrita, Dhanvamamsa, yava, Shali, Yapana ksheera Basti, Virechana, Panchmoola sidhha ksheera.
Kaphavruta vata	Saitya, gourava, shoola, katvadhyaupashyo, langhanayasa usnakamita	Swedana, niruha, vamana, virechana, tila, purana ghrita, sarshapa
Raktavruta vayu	Daha, arati, pain in between mamsa and tvaka, saraga swaythu.	Vatrakta treatment
Mamsavruta vayu	Kathina vivarna, pidikas and swaythu, harsa, pipeelika sanchara.	Sweda, abhyanga, Rasa ksheera sneha.
Medavruta vayu	Chala, snigdha, mridu, sita, sophanga, Aruchi.	Medohara chikitsa and prameha chikitsa
Asthiavruta vayu	Feeling comforte while pressing and hot touch pidana, priking type of pain	Mahasneha
Majjavruta vayu	Vinama, jrumbhana, pariveshtana, shula, Feeling comforte while pressing	Mahasneha
Sukravruta vayu	Avega and ativega, Nisphaltvam	Mahasneha
Annavruta vayu	Shula after intake pf food, during jirana avstha,	Ullekhana, Pachana, Dipana, Laghu ahara
Mutravruta vayu	Apravruti and vsti adhmana.	Sweda, Uttar basti
Vidavruta vayu	Vibandha, parikruntati, anaha, Shuska dukha chirat pidita while defecation, pain in shroni, vakshana and prishta and discomforte in hridaya.	Eranda taila, Snigdha and Udavarta chikitsa

Clinical types of Avarana

1. Avarana of Sama Vata by Vriddha Dosha and or Dooshya
2. Avarana of Vriddha Vata by Sama Dosha and or Dooshya
3. Avarana of Vriddha Vata by Vriddha Dosha and or Dooshya

Diagnosis of Avarana

Diagnosis of avarana can be done by differentiating with the kshaya, vriddhi, gata vata with profound knowledge of lakshanas. It is done by exclusion & reasoning.

Exclusion by Nidana:

E.g.

- Madhumeha - due to avarana. Cha.Su.17/78-79
- Madhumeha - of kevala vatajanya. Cha.Ni.4/36
- Though the disease is of vata there is no vata nidanas or dhatu kshaya.
- But the history of indulgence in nidanas of avaraka is appreciable.

Exclusion by Lakshana

- In kevala vatajanya madhumeha- mootra is kashaya and madhura rasa, pandu in varna.
- In avaranajanya madhumeha- Dhatu kshaya lakshanas are absent in intial stages. (Cha. Su. 17/81)

Exclusion by Upashaya

- The condition of avarana will not respond to upashaya of vata rather he feels sukha with the upashaya of the avaraka.

E.g.

- In pittavruta vata - upashaya is sheeta but not the ushna.
- In kaphavruta vata - upashaya is katu etc & he feels preeti to langhana, aayasa rukshoshna kamita. (Cha Chi 28/61-63)

Upadrava of Avarana

- Hridroga
- Vidradhi
- Pleeha
- Gulma
- Atisara

Saadhya- asadyata of avarana

- श्लेष्मपित्तावृत प्राण उदान गम्भीर (प्राण जीवन, उदान बलाश्रय)

  Life lies in prana vata, strength in udana.

- परि संवत्सर उपेक्षित आवरण दुरुपक्रम

  Among dhatu avarana medavruta vata is gurutara to treat.

Importance of Avarana

- क्षयं वृद्धि समत्वं च तथैवावरणं भिषक् विजेय पवनादीनां न प्रमुह्यति कर्मसु। (Ch.Chi.28/47)
- The concept of avarana forms the route of pathogenesis for many diseases. It mainly help us to understand the actual samprapti and to do the samprapti vighatana thus helping for treatment.
- Chakrapani has made great effort in better understanding of different configuration of Doshas, Dooshya and Malas in process of Avarana.
- Understanding Avarana will enhance the quality of the treatment in various diseases forms and take to better prognosis.
- Bringing back the doshas from its unmatched marga turning its direction to the prakrita either by increasing or decreasing, optimizing the speed one can regulate doshagati.
- In modern science, doshagati can be measured by sphygmomanometer, Galvanometer, Stethoscope, EEG, ECG, EKG which are expressed in the form of osmotic pressure, threshold, impulse, conduction etc.
- The objective understanding of Doshagati is possible today by using advanced imaging techniques (USG, 3D Doppler study etc.)
- The concept of svamargaharana treatment, pratimargaharana treatment all are built up on Doshagati and Rogamarga.
- The concept of aavarana is not only conceptual and hypothetical but also practically evident.
- If the conventional treatment fails the reconsideration of the case w.r.t avarana has to be made
- Setting of objective parameters for aavarana is a task of challenge.

11. Doshagati and Rogmarga

Introduction

Chalatva or gamana/movement, motility and locomotion is a chief expression of life from unicellular organism to multicellular organism, where gati signifies the presence of life, hence differentiating living from non-living.

Gati is required for sanchara of prana, sanchara of nutrition, sanchara of sanjna, sanchara of mala and such essential factors. The above concept is totally based on doshagati. The doshas are principle aspects of life, if at all gati have to exist.

The doshas travel carrying respective dhathus and corresponding elements along the margas/srotas. Therefore, for the physiological continuity how much doshagati is essential similarly for the vikritotpatti the association of doshagati is mandatory. In whichever marga the vikrita dosha moves that becomes Rogamarga

- गतिर्बहिर्निस्सरणम् तद्वन्ति गतिमन्ति पुरीषादीनि (च.सू. १८/४९)
- मार्गेण गच्छन् (च.वि. ५/९) any thing that moves in a marga is gati
- शरीरगतस्य वातेर्गतिः (अस.नि. १५/३१)

Prakrita Karma of Vata W.R.T. Gati

- प्रवर्तकचेष्टानामुच्चावचानां- Stimulant, initiator of any movement or reflex
- सर्वशरीरधातुव्यूहकर - Projected to all parts of body
- क्षेप्ता बहिर्मलानां - Quality of throwing or oozing
- नियन्ता प्रणेता च मनसः - Connects mind and body
- समीरणो अग्नि - Influences and maintains agni
- सन्धानकरः शरीरस्य - Connects and communicates with various organs and systems. (च.सू. १२)

उत्साहोच्छवास निश्वास चेष्ट धातुगतिः समाः। समो मोक्षो गतिमतां वायोः कर्माविकारजम्।। (च.सू. १८/४९)

Vata maintains gati of other dosha, other dhatu and body in the optimized form. If there is any change in speed, velocity, pressure, of movement of vata than normal (abnormal gati) then it causes changes in its physiological function leading to disease.

Causes of Vaikruta Dosha Gati

- Vegadarana
- Srotodushti
  (a) Atipravritti (c) Vimargagamana
  (b) Sanga (d) Siragranti
- Dosha Vaishamya– Various doshaavasta (vriddi, kshaya, hrushta, utklesha, leena, pradosha, prakopa)

Classification of Dosha Gati

क्षयः स्थानं च वृद्धिश्च दोषाणां त्रिविधा गतिः। ऊर्ध्वं चाधश्च तिर्यक् च विज्ञेया त्रिविधाऽपरा।।
त्रिविधा चापरा कोष्ठशाखा मर्मास्थि संधिषु। इत्युक्ता विधिभेदेन दोषाणां त्रिविधा गतिः।।
(च.सू.१७/११३)

There are three primary varieties of dosha gati as per charaka.

1. Kshaya (decreased), Stana (normal), Vriddi (increased)- Based on speed.
2. Urdwa (upward), Adha(downward), tiryak (sideward)- Based on direction.
3. Koshta (trunk, abdominal and pelvic organs), Shaka (exposed or external surface of body) Marmasthi sandhi (vital organs, bones and joints- Based on structures.

I. Based on Normalcy-Prakruta Dosha Gati

1. Vata : सर्वा हि चेष्टा वातेन स प्राणःप्राणिनां स्मृतः। तेनैव रोगा जायन्ते तेन चैवोपरुध्यते।।

The cala guna is from vata dosha. All kinds of circulation, locomotion, conduction of normal elements is brought by vata. If the element to be transported is diseased then by doshagati disease can also be carried to other organs or disease can be spread all over

2. Pitta : पित्तादेवोष्मणः पक्तिर्नराणामुपजायते। तच्च पित्तं प्रकुपितं विकारान् कुरुते बहून्।।

The radiation type of gati is done by pitta. Majority of its movement is in association with vata. As pitta is ushma the heat radiation is a method of homeostasis. The heat is produced as result of digestion, breakdown or metabolism. Due to abnormal pitta abnormal metabolism and chemical reaction takes place which does heat imbalence. The abnormal heat can be carried to bring various diseases.

3. Kapha : प्राकृतस्तु बलं श्लेष्मा विकृतो मल उच्यते।
स चैवोजः स्मृतः काये स च पाप्मोपदिश्यते।। (च.सु. १७/११६-११८)

The normal gati of sleshma builds up the body and provides or supplies strength and energy to various cells. Otherwise it causes accumulation of waste in the body.

II. Ritu Bedha Based Dosha Gati

चय प्रकोप प्रशमाः पित्तादीनां यथाक्रमम्। भवन्त्येकैकशः षट्सु कालेष्वभ्रागमादिषु।।
(च.सू. १७/११४)

Chaya, prakopa, prashama of three dosha takes place with respect to six ritus.

III. Based on Abnormality- Vaikruta Dosha Gati

There are 8 kinds of primary varieties of samprapti . Hence there are 8 chapters mentioned in charaka nidana stana, rest of the samprapties are derived from these primary 8 samprapties. It is elaborately explained in charaka chikitsa stana as 30 chapters. Again methodically enormous samprapties can be derived upon it based on permutation and combination of:

a) Doshavasta
b) Doshagati
c) Srotojanya vikriti
d) Sthaana samshraya w.r.t khavaigunya
e) Ashayapakarsha

Types of Dosha Gati (Vriddi, kshaya, sama)

१. वृद्धि

वृद्धिः स्वप्रमाण आधिक्यम्। (अ.ह.सू. १/१३)

Vriddi is qualitative or quantitative increase of doshas and other elements.

eg: - Vata Vrudhi Laxanas

- .....वृद्दस्तुकुरुतेऽनिलःकार्श्यकार्ष्ण्योष्णकामित्वकम्पानाहशकृद्ग्रहान्।
बलनिद्रेन्द्रियभ्रंशप्रलापभ्रमदीनताः।। (अ.ह.सू. ११/५-६)

Increased vata causes physical weakness or turns the body lean. Dark color of skin, patients longs to have warm or hot food. Presence of fine to course tremors, abdominal destionsion, dryness of stool and thus constipation, the capisity of sense organ decrease. There will be diminision of higher mental function, therefore irrelevant talk and behavior due to disorientation.

वर्धनम् वृद्धि हेतुः। (अ.ह.सू. ११/२८)

२. क्षय

Kshaya is is qualitative or quantitative decrease of doshas and other elements. in this decreased function of respective component is seen. absence of normal performance.

Eg ; pitta kshaya lakshanas.

- **पित्तेमन्दोऽनलःशीतंप्रभाहानिः।** (अ.ह.सू. ११/१६)

Pitta kshaya causes diminision of agni, diminision in function of vayu, cold and clamy body, loss of complexion.

क्षीणानां प्रकृतिलिंगं क्षय व्यतिरिक्तं विकारकर्तृत्वं नास्तीति दर्शयति।
स्वयमेव दुस्थितत्वात्। (च.सू. १७/६२)
यतो वृद्धो उन्मार्गगामिनो दोषा दूष्योदूषयन्तो ज्वरादीन्कुर्वन्ति न क्षीणाः।।

Vridda doshas can proceed to prakopa of doshas causing to get deviated to other marga than normal. (unmarga gamana) This produce various diseases. But ksheena doshas itself lose its presence and cannot make any disease as the doshas are weak by itself.

Based on direction dosha gati (urdwa, adha, tiryak)

1. **Urdwa gati ; Tendency of doshas moving in upward direction**

Eg; Rakta pitta

Pitta and rakta dushtikara Nidana
↓
Vridhi of drava guna
↓
Increase in volume
↓
As rakta is its ashraya, it goes to raktotpatti stana (yakrit & pleeha)
↓
Both rakta and pitta undergoes dushti
↓
Dushta rakta remains unutilized
↓
Further increase in volume
↓
Increased production of dushta rakta and decreased excretion or utility
↓
Association of Kapha dosha Urdwa gamana of Dosha.
↓
Urdwaga Raktapitta

2. **Adho gati : Tendency of dosha moving down wards:**

Eg ; Adhoga Amla pitta

Nidana
↓
Pitta dushti
↓
Agnimandya
↓
Pitta vidagdata and Ama formation
↓
Annavahasrotodushti
↓
Apakva anna reaches Adho amashayaVidagda anna as such will get excreted adho pravrutti of vidagda anna
↓
Adhoga amla pitta

3. **Tiryak gati : To in sideward direction**

Eg : Kushta

Ukta nidana
↓
Vatadi dosha prakopa
↓
Utklesha of saptavidha kushta dravyas
↓
Gamanapravritti of kushta dravyas
↓
Obstruction/avarodha of urdva & adho marga (supression of vegas or structural sanga)
↓
Tiryak gamana of dosha
↓
Gets lodged in tvacha
↓
Dushti of other dhathu's like rakta, mamsa etc (stanika dosha dushti)
↓
Mandalotpatti and vaivarnya etc
↓
Kushta

samavasta and pitta is brought back to its normal ashaya. Then symptoms automatically pacify.

Conclusion

- Gati can be self motivated or altered by intervention. Maintaining the speed in gati, direction of gati, pathway of gati is all about maintainence of health.
- Doshas may move in abnormal speed but in normal direction. Eg: high blood pressure.
- Doshas may move in normal speed but in abnormal direction Eg: Urdvaga amlapitta where pitta should move from amashaya to pachyamanashaya thus forward, but instead in amlapitta it moves in upward direction.
- Both speed and direction may be normal but the pathway/ Rogamarga may be altered. Eg: Kushta.
- Bringing back the doshas from its unmatched marga turning its direction to the prakrita either by increasing or decreasing, optimizing the speed one can regulate doshagati.
- In modern science, doshagati can be measured by sphygmo-manometer, Galvanometer, Stethoscope, EEG,ECG, EKG which are expressed in the form of osmotic pressure, threshold, impulse, conduction etc.
- The objective understanding of Doshagati is possible today by using advanced imaging techniques (USG, 3D Doppler study etc.)
- The concept of svamargaharana treatment, pratimargaharana treatment all are built up on Doshagati and Rogamarga.
- The concept of आशयापकर्ष is not only conceptual and hy-pothetical but also practically evident.
- If the conventional treatment fails the reconsideration of the case w.r.t आशयापकर्ष has to be made.
- Setting of objective parameters for आशयापकर्ष is a task of challenge.

Rogamarga :-

Introduction

Movements of doshas are called as dosha gati. But the channels which they select for their movement and finally settle in new abode is called as Marga. The pathway in which dushta doshas move and carry disease producing agents with them is called as Roga Marga. They are three in number. Generally srotas are the margas. But to understand the sthana of disease it can be catagorised under three divisions

1. Shakha: (external pathway.) Also called as bahya roga-marga
2. Marmasthi sandhi: (middle pathway) Also called as madhyama roga marga
3. Koshta: (Internal pathway) Also called as abhyantara roga marga

त्रयो रोगमार्गा इति-शाखा मर्मास्थिसन्धयः कोष्ठश्च।

Shaka roga marga: The superficial body components or uttana dhatus are part of bahya roga marga. It lies in the most pheripheral or external surface of the body. Rasa and twak, rakta, mamsa dhatus are components of shaka marga. The diseases of shakhamarga are manifested mostly in twak

तत्र शाखा रक्तादयो धातवस्त्वक् च, स बाह्यो रोगमर्गः

Marma asti, sandhi: The middle compartment of body which consists of marma(specially shira, hrudaya, basti), asti, sandhi (musculoskeletal system - muscle, tendon, ligaments, bones, joints) is madhyama roga marga

मर्माणि पुनर्बस्तिहृदयमूर्धादीनि, अस्थिसन्धयो अस्थिसंयोगास्तत्रोपनिबद्धाश्च स्नायु कण्डराः, स मध्यम रोगमार्गाः

Koshta marga: This is the inner most compartment of the body. This comprised of all the organs of thorasic, abdominal, pelvic region. Shareeramadhya, Mahanimna, antaraani are synonyms to koshta. Thus it forms abhyantara roga marga.

कोष्ठः पुनरुच्यते महास्रोतः शरीरमध्यं महानिम्नमामपक्वाशयश्चेति पर्याय-शब्दस्तत्रे, स रोगमार्गा आभ्यन्तरः।

Diseases pertaining to particular roga marga

Shakaanusari Roga

The diseases of bahya roga marga are:

Ganda (mumps) pidaka (popular rash) alaji (smaller than pidaka) apaci (oozing ulcer) Charmakeela (warts) adhimaamsa (mass formation) mashaka (macula popular rash) kushta (skin diseases), visarpa (eryseples) shwayathu (local oedema), gulma (swelling) arsha (external pile), vidhradhi (abscess). Vyanga (skin discoloration).

तत्र गण्डपिडकालज्यपची चर्मकील अधिमांस मषक कुष्ठ व्यङ्गदयो विकारा बहिर्मार्गजाश्च विसर्प श्वयथु गुल्म अर्श विद्रध्यादयः शाखानुसारिणो भवन्ति रोगाः।

Madyamaroganusari Roga

The diseases pertaining to madhyama roga marga are :

Pakshagata (hemiplegia) paksha graham (hemiparesis), apatanaka (tetanus), ardita (facial palsy), sosha (degenerating diseases) rajayakshma (depleting disease) asti sandhi shoola (arthralgia) gudabramsha (prolopse rectum) diseases of shiras and basti.

पक्षवध ग्रह अपतानक अर्दित शोष राजयक्ष्म अस्थि सन्धिशूल गुदभ्रंशादयः।

Koshtanusari Roga

The disease pertaining to inner most compartment of the body is called as abhyantara roga marga. They are: jwara (fever) atisaara (diarrhea) chardi (vomiting), alasaka (stagnant compound of ama) vishuchika (cholera) kasa, (cough) shwasa (dyspnoea) hicca (hiccups) anaha (abdominal distension) udara (ascitis) pleeha (spleenomegaly) visarpa (erysepeles) shwayathu (local oedema), gulma (swelling) arsha (pile), vidhradhi (abscess).

ज्वरातिसार च्छर्दि अलसक विसूचिक कास श्वास हिक्का आनाह उदर प्लीहादयो अन्तर्मार्गजाश्च विसर्प श्वयथु गुल्म अर्शोविद्रध्यादयः कोष्ठानुसारिणो भवन्ति रोगाः।

12. Detailed study of Srotomoola and Srotodushti Samanya andVishishta Hetu Lakshana of all Srotas, Differences between Sroto Dushti and Kha Vaigunya

ntroduction

The physiological and anatomical pathways that carry all the omponents, elements, signals, reflexes come under srotas. In some ontexts the srotas can be traced anatomically and hence understood s channels or passages eg: annavaha srotas, pureeshavaha srotas. Whereas in some anatomical traces may not be found instead they re identified by physiological pathways eg: manovaha srotas, wedavaha srotas. Every srotas has one ugama sthana or moolasthana rom which transportation or transmission begins and one prabhava thana from which the transported matter finally reaches its destiny. Srotas carry dosha, dhatu, upadhatu, mala in it. Therefore dosha ushti can produce srotodushti and vice versa.

efinition

स्रवणात् स्रोतांस्सि। (च.सू. ३०/१२)

It is the path which helps for transformation of substance.

स्रोतांसि खलु परिणामम् आपद्यमानानाम् धातूनां अभिवाहिनि भवन्त्ययनार्थेन। (च.वि. ५/३)

मूलात् खातान्तरम् देहे प्रस्रुतम् तु अभिवाहियत् स्रोतस्तदिति विज्ञेयम् सिरा धमनि वर्जितम्। (सु.शा. ९/९३)

Channels which carry the transformed dhatus to different destinations through the network to nourish the cells and tissues.

Srotas is defined as empty spaces spread to entire body which originates from root space except sira and dhamani.

roto Paryaya

स्रोतांसि सिराः धमन्यः रसायन्यः रसवाहिन्यः नाड्यः पन्थनः मार्गाः शरीरच्छिद्राणि-संवृतासंवृतानिस्थानानि आशयाः निकेताश्चेति शरीर धात्वावकाशानां लक्ष्यालक्ष्याणां नामानि भवन्ति।। (च.वि. ५/९)

Srotamsi, sira, dhamani, rasayani, rasavahi, nadya, panthana, margasareera chidrani, samvruta asamvritani, sthanani, ashaya, niketha based on the context and element that is carried inside srotas, apt term for srotas is used.

Structure of Srotas

imp.

- अहित सेवनात् तानि दुष्टाय रोगाय विशुद्धानि सुखस्य च । (अ.हृ.शा. ३/४२)

Orifices of the srotas are minute spreaded long and far away like lotus stalk. Through such channels 'rasa' circulates and nourishes cells and tissues.

Sroto Pramukhyata/ *Importance -*

imp.

- अहित सेवनात् तानि दुष्टाय रोगाय विशुद्धानि सुखस्य च। (अ.हृ.शा. ३/४२)

Improper foods, erratic behaviour and such other things which are not conducive to the body brings abnormality in srotas leading to manifestation of disease. Adoptance of normal foods and actions leads to happiness and sound health.

- वातपित्तश्लेषमाणां पुनः सर्व शरीर चराणां सर्वाणि स्रोतामि अयन भूतानि तद्वदतीन्द्रियाणां पुनः सत्वादिनां केवलं चेतनावतच्छरीरमयनभूतमधिष्ठानभूतं च।
  तदेतत् स्रोतसां प्रकृति भूतत्वान्नविकारैरूपसृज्यते।। (च.वि. ५/७)

The doshas vata, pitta and kapha moves inside the srotas to perform their normal functions at different places similarly things which are beyond perception of sensory organs like mind etc. move inside the srotas and are located in each part of the body. Healthy srotases perform their normal functions as a result body is free from diseases and unhealthy srotas become root cause for the development of pathogenesis.

Sroto Bheda

Srotas : Bahya and Abhyantara

1. According to Susruta

Srotas :– Bahya-Male- 9; Females- 12; Abhyantara- 11 pairs

2. According to Sarangadhara

Bahya Srotas- Male- 10, Female- 13

3. According to Charaka

Abhyantara Srotas- 13 in Number.

Bahir Mukha Srotas

Nasal cavity- 2
Ear path- 2
Eye opening- 2
Genital path-I
Anal canal- 1
Oral cavity- 1

Total nine channels are described as bahir mukha srotas.

Sarangadhara included one more to above nine ie, mastaka randra (brain canal)

In case of female three more srotas are included.

Breast channels- 2
Uterus path- 1

Abhyantara Srotas

According to Charaka

1. Pranavaha Srotas
2. Udadavaha Srotas
3. Annavaha Srotas
4. Rasavaha Srotas
5. Rudiravaha Srotas
6. Mamsavaha Srotas
7. Medovaha Srotas
8. Asthivaha Srotas
9. Majjavaha Srotas
10. Sukravaha Srotas
11. Mutravaha Srotas
12. Pureeshavaha Srotas
13. Swedavaha Srotas

According to Susruta

1. Pranavaha Srotas
2. Udakavaha Srotas
3. Annavaha Srotas
4. Rasavaha Srotas
5. Rudiravaha Srotas
6. Mamsavaha Srotas
7. Medovaha Srotas
8. Sukravaha Srotas
9. Mutravaha Srotas
10. Pureeshavaha Srotas
11. Artavavaha Srotas

Sroto Dushti Prakara

- Atipravrutti– Excessive action
  Eg:- excessive urination - prameha
  excessive watery stools - atisara
- Sanga- Excessive Obstruction
  Eg : - jwara
- Sira Granthi– Tumurs inside the srotas or new growths tortuous vessels.
  Eg :- arsas
- Vimarga gamanama– Leaving its own path and entering into other path.

Eg :- entrance of mala into mutra marga

Sroto Vidda Laxana (A.H.Sar. 3/47)

Clinical features that develops due to injury to srotas are called as vidda. Their lakshanas are unconsiousness, tremors, distention of sabdomen, vomiting, fever, delirium, obstruction of urine and stool and even leads to death. That is why it is said that physician should inform to the attender about its poor prognosis and then plan the treatment and manage the wounds as per its treatment.

1. Pranavaha Srotas

Pranavaha Sroto Moola : प्राणवहानां स्रोतसां हृदयंमूलं महास्रोतश्च

- Charaka-The channels which carry prana vayu are originated from hrudaya and mahasrotas (GIT)
- Susruta-They are two in number

  It originates from hrudaya and rasa vahini dhamani : प्राणवहे द्वे तयोर्मूलं हृदयं रसवाहिन्यस्य धमन्यः।

Dushti Nidana

क्षयात् संधारणात् रौक्ष्यात् व्यायामात् क्षुधितस्य च। प्राणावाहिनी दुष्यन्ति स्रोतस् अन्यैश्च दारुणैः।

- Decrease of dhatus
- Suppression of natural urges
- Intake of dry food
- Doing exercise when one is feeling hungry
- These causes dusthi & malfunctioning of pranavaha srotas

Laxana According to Charaka

तत्र प्राणवहानां स्रोतसां हृदयं मूलं महास्रोतश्च प्रदुष्टानां तु खल्वेषामिदं विशेषविज्ञानं भवति तद्यथा—अतिसृष्टमतिबद्धं कुपितमल्पमभीक्ष्णं वा सशब्दशूलमुच्छ्वसन्तं दृष्ट्वा प्राणवहान्यस्य स्रोतांसि प्रदुष्टानीति विद्यात् (Ca. ni. 5/8)

- Prolonged respiration
- Obstructed respiration
- Short breath with increased frequency
- Loud respiration associated with pain, overall respiratory distress.

Viddha Laxana According to Susruta

- Injury to pranavaha srotas leads to groaning
- Bending down of body

- Illusion
- Giddiness
- Tremors
- Ultimately leads to death

Pranavaha Sroto Vikara

- Kasa
- Svasa
- Hridroga
- Rajayakshma
- Swarabheda

The Cardio Vascular System :-

It is the system of heart and blood vessels that circulates blood through out the body.

- The blood circulating the body transports nutrients and oxygen to the tissues and removes Co_2 and waste products from the tissues.
- Heart is the central pump and blood vessels the series of distributing and collecting tubes.
- CVS constitute one of the major coordinating and integrating systems of the body.

Heart

- It is a muscular organ that pumps blood through out the circulatory system.
- Its situated in between two lungs in the mediastinum
- Its made up of two atria and two ventricles.
- The force of contraction of heart depends up on muscles of heart.

Layers of Walls of Heart

- Outer pericardium
- Middle myocardium
- Inner Endocardium

Right Side of Heart

- Right side has upper atrium and lower ventricle.
- Right atrium is a thin wall and low pressure chamber
- It has a pace maker known as sino atrial node that produces cardiac impulses and atrio ventricular node that conducts the impulses to ventricles.
- It recieves deoxygenated blood through two large veins
- A. Superior venacava that returns the blood from upper parts of the body.

B. Inferior venacava that returns blood from lower parts of the body

- Right atrium communicates with right ventricle through tricuspid valve.
- From right ventricle pulmonary artery arises which carries blood from right ventricle to lungs.
- In the lungs the blood is purified(oxygenated)

Left Side of Heart

- Left side of the heart has upper left atrium and lower left ventricle
- It is thin walled and low pressure chamber.
- Left atrium recieves oxygenated blood from lungs through pulmonary veins.
- This is one of the exception in the body where we can see artery carry deoxygenated blood and veins carry oxygenated blood.
- Blood from left atrium enters left ventricle through bicuspid valve.
- Left ventricle pumps oxygenated blood to different parts of the body through aorta.

The Respiratory System:-

- It can be divided into upper and lower respiratory tracts
- Functionally it can be divided into conducting and respiratory portion.
- The conducting portion consist of a series of inter connected tubes that circulate air through, nose, pharynx, larynx, trachea, bronchi and bronchioles till terminal bronchioles
- The respiratory zones consist of alveolar ducts, alveolar sacs and alveoli that take part in gaseous exchange.

Bronchial tree

- The trachea divided into right and left bronchi
  Primary bronchus (2 to left and 3 to right)
  Secondary bronchus
- Tertiary bronchi
  Bronchioles
  Terminal bronchioles
  Respiratory bronchioles
  Alveoli

New Pathological Condition

Bronchiectasis

Persistent and irreversible dilatation and distortion of medium sized bronchi by more than 2 mm. Types are cylindrical, saccular, varicose and fusiform. Clinical features includes persistent, recurrent cough and large quantity of purulent sputum production; haemoptysis; persistent coarse leathery crackles with or without bronchial breathing. Clubbing of fingers and toes present.

Copd (Chronic obstructive pulmonary disease)

It is characterized by airflow limitation that is not fully reversible. Usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases. Diagnosed by cough, sputum production, dyspnoea, ,H/O exposure to risk factors for the disease.

Emphysema

It is defined as distension of the air spaces distal to the terminal bronchiole with destruction of alveolar septa. Types include centriacinar, panacinar, paraseptal and irregular.

Bronchial Asthma

It is an inflammatory disease of the small airways, characterized by episodic, reversible bronchial obstruction due to hyper responsiveness of tracheobronchial tree to a multiplicity of intrinsic and extrinsic stimuli manifested clinically by paroxysms of polyphonic wheeze, dyspnoea and cough which may be relieved spontaneously or as a result of therapy.

2. Udakavaha Srotas :-

- Udaka– Jala, ambu, vaari, salila, water
- Vaha– Carrying, bearing, bringing, flowing
- Srotas– Channel, stream, processing
- Udakavaha srotas is the one which carry or controls the water in the body tissues

Definition

- The channel, passage or duct which transport the liquid substance of the body from one place to other for development and nourishment of the body.

Synonyms

• Ambuvaha • Jalavaha

Mula

• उदकवहानांस्रोतसांतलुमूलंक्लोमं च। (च.वि. ५/८)

Udakavaha srotas mula are taalu and kloma

• उदकवहेद्वेतयोर्मुलंतालुक्लोम च। (सु.स. ९/१२)

Udakvaha srotas originate from talu and kloma

Dusti Karana

• औष्ण्यादामादभयात्पानदतिशुष्कान्नसेवनात्। अम्बुवहिनिदुष्यन्तित्रुष्णाश्चतिपिडनात्।। (च.वि. ५/११)

Udakvaha gets vitiated by exposure to heat, indigestion, fear, encess intake of alcohol, excess intake of dry food and who is suffering from excessive thirst.

Dusti Lakshana

• प्रदुष्टानांतुखल्वेषमिदंविशेषविज्ञानं भवतिः तथ्यताजिह्वातल्वोष्ठक्लोमशोषंपिपासां प्रबुध्दांदुष्टोदकवहान्यस्यस्रोतांसिप्रदुष्टानितिविध्यत्। (च.विं. ५/८)

Characteristics of udakvaha srotus are dryness in tongue, palate, lips and kloma along with excessive severe thirst.

• तत्रविद्दस्यपिपासासध्योमरणंच।

Injury to udakavaha srotus develops thirst and patient dies instantaneously.

Vikara

Trushna, Shopha, Prameha

Water Balance in the Body

Decreased water content gives Stimulation to thirst center. Osmoreceptors in hypothalamus answers the reflex and express to increase water intake. On the other hand Increased ADH secretion from post pituitary orders to increase water retention by kidney to conserve normal water content.

Hypothalamus regulates water content of the body by two mechanisms.

Thirst Mechanism

• Thirst center present in lateral nucleus if hypothalamus
• There are some osmorecepters in the areas adjacent to thirst cente

• When the ECF volume decreases the osmolality of ECF increases.
• If the osmolarity increases by 1-2% the osmoreceptors are stimulated.
• Osmoreceptor in turn activate the thirst center thirst sensation is initiated now person feels thirsty and drinks water.
• Water intake increased ECF volume and decreases osmolality.

ADH mechanism

• When the volume of ECF decreases with increased osmolality the supra optic nucleus is stimulated and ADH is released.
• ADH causes retention of water by facultative re absorption in the renal tubules. It increases the ECF volume and brings the osmolality to normal level.

Pancreas

• Histology of pancreas-

Made up of small clusters of glandular epithilial cells.

About 99% of the cluster called acini constitute exocrine portion of the organ these secrete pancreatic juice.

1% of the cluster called pancreatic islets from the endocrine portion of the pancreas these secret glucogon, insulin, somatostanin and pancreatic polypeptide.

• Role of insulin and glucogon-

Insulin decreases blood sugar level it is the only antidiabitic hormone available in the body.

Glucogon increases blood sugar level.

What are Talu and Kloma

• Talu can be considerd as palate because the description of structure of talu resembles to palate exatly.

But kloma is the very controversial organ

• By seeing various reference we can tell that kloma the pancreas.
• Acharyas like charaka, vagbata, bela and kashyap have consider kloma as koshtanga therefore it should found in trunk that is thoracic or abdominal cavity.

By considering above view we can tell that kloma not present in head and neck.

Some classical references

- तस्यवामपार्श्वेप्लिहाफुप्फुसदक्षिणतोयकृत्क्लोमच।। (अ.स.शा. ५/७१)

  Tells about hrudaya which arises from the prasada bagha of rakta. In left lateral is pleeha and lung are present and in the right we have yakrut and kloma.

- तस्याधोवामतः प्लीहफुप्फुसाश्च दक्षिणतोयकृत्क्लोमच। (सु.शा. ४/३०)

  This shloka explains kloma present below hrudaya to left pliha and pupusa to the right kloma and yakrut.

- कालखण्ड (यकृतादधस्तात्) स्थितंदक्षिणपार्श्वस्यंतिलकमितिप्रसिद्धम्। अधस्तुदक्षिणभागोहृदयात्क्लोमतिष्टति। (डल्हण सु.नि. ९/९८)

  Kloma is the one which is present below the yakrut and which has shape of tila. Situated in right side below hridaya.

- By seeing all the references we can conclude that kloma is nothing but the pancreas.

3. Annavaha Srotas :-

Channels carrying अन्न is called as अन्नवहस्रोतस्

- अन्नवहानां स्रोतसो आमशयोमूलम् वामम् च पार्श्वम्। (च.वि. ५/८)

  चरक considered the मूल of अन्नवहस्रोतस् as- आमाशय and वामपार्श्व; stomach and left side of Abdomen.

- अन्नवहेद्वेतयोर्मुलम्आमाशयोअन्नवाहिन्यश्चधमन्यः। (...९)

  सुस्रुत told अन्नवहस्रोतस् are two in number and their मूल as आमाशय and अन्न वाहिनी धमनि।

निदान

- अतिमात्रस्य च अकाले च अहितस्य च भोजनात्। अन्नवाहिनिदुष्यन्तिवैगुण्यात्पावकस्यच।। (....५)

  अतिमात्र आहार - Excess intake of food
  अकाल आहार - Eating during improper time
  अहित आहार - Consumption of unwholesome food
  वैगुण्यात्पावकस्य- Due to dushti of agni.

According To Modern Science : Stomach (आमाशय)

- A muscular bag forming the widest & most distensible part of the digestive tube.

Dr. Hemant Kumar Maurya

- Connected above to the lower end of the oesophagus & below to the duodenum.
- Acts as a reservoir of food & helps in digestion of carbohydrates, proteins & fats.

Location

- Lies obliquely in the upper & left part of abdomen.
- Occupying epigastric, umbilical & left hypochondric region.

Shape & Size

- When empty - J shaped
- When partially distended - pyriform in shape
- In obese persons - more horizontal
- 25 cm long
- At birth- 30 ml capacity

External Features

- Stomach has 2 orifces
- 2 curvatures or borders
- 2 surfaces

Layers of Stomach

- Wall of the stomach is formed by four layers-
  Outer serous layer
  Muscular layer
  Submucus layer
  Inner mucus layer

Serous Layer– formed by peritoneum which covers the stomach except at the lesser and greater curvatures.

Muscular Layer– made up of three layers of smooth muscle fibres-

Inner oblique, middle circular, outer longitudinal

Submucous Layer– Formed by areolar tissue, blood vessels, lymph vessels and Messner's nerve plexus.

Inner Mucous Layer– Lined by mucus secreting columnar epithelial cells, gastric glands are situated in this layer.

Glands of Stomach

- Glands of stomach are gastric glands
- These are tubular structures made up of different types of cells
- Open into stomach cavity through gastric pits

Classification of Gastric Glands

- Fundic glands
- Pyloric glands
- Cardiac glands

Secretory Functions of Cells in Gastric Glands

- Chief cells
- Parital cells
- Mucus cells
- G cells
- Enterochromaffin cells
- Enterochromaffin like cells

Secretory Products

- Pepsinogen,renin,lipase,gelatinase,urease
- HCL, intrinsic factor
- Mucin
- Gastrin
- Serotonin
- Histamine

Functions of Gastric Juice

- pepsinogen $\xrightarrow[\text{to}]{\text{breaks}}$ pepsin

  pepsin - activated form, proteolytic enzyme
- Proteins $\xrightarrow[\text{to}]{\text{breaks}}$ proteases+peptones
- Gastric lipase- lipolytic enzyme

  Tributyrin (butterfat) $\xrightarrow[\text{to}]{\text{breaks}}$ fatyacid+glycerols
- Gelatinase degrades Type 1 & Type 5 gelatin & Type 4 & 5 collagen into peptides
- Urase- urea $\xrightarrow[\text{to}]{\text{breaks}}$ ammonia
- Gastric amylase degrades starch
- Renin- curdles milk, only in animals

Functions of HCL

HCL present in gastric juice-

- Activates pepsinogen into pepsin
- Kills some of the bacteria entering the stomach along with food substances. This action is called bacteriolytic action.
- Provides acid medium which is necessary for the action of hormones.

Abdominal Aorta (वामपार्श्व)

- It is a region of descending aorta , originating superiorly as a ***continuation of Thoracic aorta,*** as it passes through an opening in diaphragm & terminating inferiorly as the abdominal aorta bifurcates into left & right common iliac arteries.
- It is a large lumened , unpaired arterial vessel that is a part of the main trunk of the systemic arterial system.
- As such the abdominal aorta supplies oxygenated blood pumped by the left ventricle of heart, to the abdominal & pelvic organs & structures via visceral & parietal arterial branches.
- The abdominal aorta & its major arterial branches are highly elastic.
- During systole the aorta & arterial walls expand & accommodate the increased blood flow.
- Correspondingly, the vessels contract during diastole & elastin fibres assure that this contraction also serves to drive blood through the arterial vessels.
- As the thoracic aorta passes through the aortic hiatus it becomes abdominal aorta.
- The abdominal aorta , ultimately branches into lft & rt common iliac arteries.
- The common iliac arteries then branch into internal & external iliac arteris to supply oxygenated blood to the organs & tissues of the lower abdomen, pelvis & legs.

Major branches of the abdominal aorta include the coeliac branches & superior & inferior mesentric arteries.

- On the dorsal side of the aorta are the lumbar & median sacral branch arteris.
- Lateral to the aorta are the inferior phrenic, middle suprarenal, renal & ovarian or testicular arteries.
- Bcoz the branches from the abdominal aorta are large, the aorta rapidly decreases in size as it courses downward through the abdomen.
- The coelic trunk divides into 3 major branches;

Left gastric artery to stomach

Hepatic artery to lobes of liver

Splenic artery to -surrounded by a plexus ofnerves that ultimately Terminates in branches

Entering the hilus of spleen.

Superior mesentric artery

- Supplies oxygenated blood to small intestine below the duodenum & portions of the caecum & colon

Branches

Inferior pancreatoduodenal artery, jejunal & ileal branches, ileocaecal artery & right & middle colic arteries.

Inferior mesentric ar Supply the transverse colon, descending colon & rectum.

Branches: left colic artery, the sigmoid arteries

अन्नवहस्रोतोदुष्टिलक्षण

- प्रदुष्टानामुखल्वेषामिदम्विशेषविज्ञानम्भवति। तथ्यथाअनन्नभिलाषम्अरोचकम्अविपाकौछर्दिंच।। दुष्ट्वाअन्नवहान्यश्चस्रोतांसिप्रदुष्टानिनिविष्यात्। (च.वि. ५/८)

अनन्नभिलाष- Lack of interest towards food
अरुचि - Tastelessness
अविपाक - Indigestion
छर्दि - Vomiting

अन्नवहस्रोतोविद्दालक्षण

- तत्रविद्दस्यआध्मनाश्शुलोअन्नद्वेषछर्दिः पिपसस्आन्ध्यम्मरणम्च। (सु.शा. ९/२)

शुल – Pain in abdomen
अन्नद्वेष – Lack of interest towards food
छर्दि – Vomiting
आन्ध्य – Blindness
मरण – Death
पिपास – Thirst

अन्नवहस्रोतोदुष्टिविकार

- छर्दि
- अतिसर
- अजीर्ण
- अग्निमान्ध्य
- क्रिमि
- अरोचक
- गुल्म
- मुखरोग
- अम्लपित्त
- विसुचिक
- विलम्बिक

4. Rasa Vaha Srotas :

Introduction

Rasa is the first dhatu among the seven dhatus to be formed from the ahaara . The main function of Rasa dhatu is the nourishment

of tissues. Hence it could be understood that, it is present in each and every part of the body. That is why its function have been told as a rasa.

Of the seven Dhatus, Rasa & Raktha Which have the function of Preenana & Jeevana karya's are continuously ejected into circulation by the Hridaya.

रस निरुक्ति

तत्र रस गतौ धातुः, अहरहर्गच्छतीत्यतो रसः।। (सु.सू. १४/१३)

The word rasa is derived from the term 'ras' means movement, since it is moving constantly it is called rasa. Ahar Ahar Gachati, if this character has to be fullfilled in the body only one organ can satisfy it i.e. Hridhaya, which never stops till the deathof the person. That's why Acharyas haveconsidered the Hridhaya as Moola of Rasa vaha Srotas. Rasa is the first and earlier part of garba, formed from ahararasa of the mother and circulates in hridaya and siradhamanies repeatedly in a cyclic manner.

यत् सादमादौ गर्भस्य यत् गर्भ रसात् रसः। संवर्तमानं हृदयं समाविशति यत् पुराः।।
(Ch.Su. 30/10)

Rasa Swabhava

- **स खलु द्रवानुसारी स्नेहन जीवन तर्पण धारणादिभिर्विशेषैः सौम्य इति अवगम्यते।**
(सु.सू. १४)

1. Dravanusari 2. Saumya (jalatatvatmak/kaphamaya)

Moola Sthana of Rasavaha Srotas

- **रसवहाना स्रोतसां हृदयं मूलं दश धमन्याः।।** (च.वि. ५/८)
- **रसवहे द्वे, तयोर्मूलं हृदयं रस वाहिन्यश्च धमन्यः।।** (सु.शा. ९/२१)

The moola of rasavaha srotas is hridaya, dashadhamani, rasavaha dhamani. It has been said by Sushruta that the word Dhamani, has to be related with Arteries only. As we have seen previously the circulatory system starts with Arteries continues as Capillaries and ends inVeins. So the structures before Capillaries can be considered as Rasavahini Dhamanis.

1. Hridaya

- **हृदस्ताः हृदय संबद्धाः।**
- **रसवतादि मार्गाणां सत्वबुद्धिन्द्रियात्मनाम्।**
प्रधानस्यौजसश्चैव हृदयं स्थानमुच्यते।। (च.चि. २४/३५)
- **रस वातादिवहनां हृदयं स्थानम्।** (चक्रपाणि)

- शोणित कफ प्रसादजं हृदयं– यदाश्रयाहि धमन्यः प्राणवहा (सु.शा. ४/३१)
- Hridaya as one among pranayatana.
- As pratyanga and koshta.
- As madyama rogamarga.
- As matruja bhava.
- As trimarma.
- As chetanadhishtana.

2. Rasavahavaha Dhamani and Dasa Dhamani

रसवाहनं कुर्वन्ति धमन्यः सततं तथा। (सु.शा.)
रसवहधमनीनां तु हृदयं स्थानं, तदुपघाताच्च मोह उपपन्न एव। (च.सू. २४/२५)

हृदय दश धमनी

- दशमूल सिरा हृत्स्ताः सर्वं सर्वतो वपुः। रसात्मकं वहन्योजस्तन्निबद्धं हि चेष्टितम्।। (अ.ह.शा. ३/१८)

There are 10 important siras in our body in relation to hridaya. It transports rasa to all angapratyangas in our body. All the cheshtas of the body are taken care by these siras.

The Circulatory System

Transports substances including oxygen, nutrients and wastes to and from cells responding to changing demands by diffusion (from high to low concentration along concentration gradient). Humans have a closed circulatory system. (cardiovascular system).

The human circulatory system consists of:

- The heart
- A series of blood vessels
- Blood that flows through

Types of Circulation

- Pulmonary circulation = from right side of the heart to lungs where carbon dioxide leaves the blood and oxygen is absorbed
- Systemic circulation = from left side of the heart to organs
- Coronary circulation = through heart tissue

Blood Vessels- They are network of tubes

Arteries: arterioles move away from the heart

- Elastic Fibers
- Circular Smooth Muscle

Capillaries: where gas exchange takes place.

- One cell thick
- Serves the Respiratory System

ins: Venules moves towards the heart

Skeletal Muscles contract to force blood back from legs

ood: Composed of plasma and blood cells

pes of Cells are:

- Red Blood Cells
- White Blood Cells
- Platelets

Plasma: they are straw yellow colored fluid that is mixed with lood. It contains 90% of water and 10% of elements. The elements are Organic, inorganic elements and dissolved gases.

Organic elements - albumin, globulin, fibrinogen, prothrombin

Inorganic elements - calcium, potassium, magnesium, sodium, iron, zinc

Gases- oxygen, nitrogen, carbon dioxide.

Others like:

- Glucose
- Amino acids
- Proteins
- Minerals
- Vitamins
- Hormones
- Waste materials like urea.

Lymphatic system: It is the One way system to the heart. There is Return of collected excess tissue fluid and Return of leaked protein

- "Lymph" is this fluid
- Edema results if system blocked or surgically removed
- Transports dietary lipids- Lymphatics vessels transport lipids and lipid soluble vitamins (A, D, E and K) absorbed by the gastrointestinal tract.
- Carries out immune response.

Sequence of lymph formation-

↓

Fluid flow in the blood capillaries

↓

Interstitial spaces (interstitial fluid)

↓

Lymphatic capillaries

↓

Lymphatic vessels

↓

Lymphatic ducts

Junction of internal jugular and subclavian vein

Absorption of nutrients in GI– 90% of all absorption of nutrients takes place in the small intestine. Absorption of Monosaccharides, Absorption of Amino acids, dipeptides and tripeptides, Absorption of lipids, Absorption of electrolytes (Caloium ions, iron, potassium, Magnesium and phosphate- active transport) Absorption of vitamins, Absorption of water takes place via osmosis.

Hepatic Portal Circulation:

- Carries venous blood from the gastro intestinal organs and spleen to the liver.
- After a meal hepatic portal blood is rich in nutrients absorbed from the gastrointestinal tract.
- Liver stores some of them, modifies others (converts glucose to glycogen) before entering into the general circulation.
- The superior mesenteric and splenic vein unite to form the portal vein.
- In this way liver receives nutrient rich deoxygenated blood which gets mixed with oxygenated blood from hepatic artery in sinusoids of liver and then bloods leaves the sinusoids through hepatic veins, which drains into inferior venecava.

दश धमनि can be taken as

1. Superior Vena cava
2. Inferior Vena cava
3. Rt & Lt Pulmonary Arteries
4. Pulmonary Veins (4),
5. Aorta
6. Coronary Sinus.

The explanation of Dhamanis, Srotas, Siras, indicating that these ten vessels are initially ten Dhamanis, & its branching behave as Srotas. This provides nutrition to nourish Dhatus. Siras are the channels which collects metabolic waste products from tissue back to circulation. It can be concluded that, anatomically Rasavaha srotas along with its moola can be correlated to Circulatory system. Rasa is all the components of food like protein, carbohydrates, fat, mineral,

vitamines which are present in the plasma as a whole i.e., every structure can be nourished by rasa and all dhatus can be formed from rasa dhatu tonly.

Continuous nourishment is necessary for the tissues by the supply of prana and rasa for the sustenance of life. For this hridaya is the only sthana which plays a role as moolasthana for rasa and pranavaha srotus. The difference between Caraka and Sushruta in rasa vaha srotas moola sthana is Rasavahinya Dhamanis and Dasha Dhamanis respectively.

The blood vessels which carry blood and nurients like hepato portal system with its tributaries, lymphatic vessels, the venacavas and umbilical vein can be considered as rasavaha srotas.

In short, prana and rasa are 2 essential nourishment factors involved in growth of other dhatus, as they circulate in a closed circuit, where they nourish all other dhatus.

The second moolasthana mentioned is rasavahi dhamani according to susruta, by which it is very clear that prana ,rasa and rakta move together through dhamani that is facilitated by heart which is nothing but circulatory system.

रसवहस्रोतो दुष्टि कारण

गुरुशीतमतिस्निग्धमतिमात्रं समश्नताम्।
रसवाहीनि....ष्यन्ति चिन्त्याननां चातिचिन्तनात्।। (च.वि. ५/१३)

Causes of dushti of rasavaha srotas are :

Excessive consumption of heavy, cold, oily food. Consumption of wholesome and unwholesome food together, grief, stress or worry.

रस प्रदोषज रोग

अश्रद्धा चारुचिश्चास्यवैरस्यमरसज्ञता। हृल्लासो गौरवं तन्द्र सांगमर्दो ज्वरस्तमः।।
पाण्डुत्वं स्रोतसां रोधः क्लैब्यं सादः कृशांगता। नाशोऽग्नेरयथाकालं वलयः पलितानि च।।
रसप्रदोषजा रोगाः, (च.सू. १८/९-१०)

While explaining Dhatu gata Jvara Charakacharya gives explanation about rasa Dhatu gata Jvara lakshana-

Nidana: गुरुत्वं दैन्यमुद्वेगः सदनं छर्दिरोचकौ। रसस्थिते बहिस्तापः साङ्गमर्दो विजृम्भणम्।।(च.चि. ७६)

Nidana Sevana leads to Agni Dusti which produces Ama form of Rasa. This Ama Rasa in one hand disturbs the normal functioning

of Vayu in Amasaya and on other hand blocks Swedavaha Srotasa. Due to blocking of the natural path in Amasaya, Vayu spreads in the whole body taking Agni (Amasayastha Pitta) with it. Blockage of Swedavaha Srotas creates Aswedanam. Thus overall Santapa increases which is termed as Jwara.

अन्न अश्रद्ध अरोचक अविपाक अङ्गमर्द ज्वर हल्लास तृप्ति गौरव हृत् पाण्डुरोग मार्गोपरोध कार्श्य वैरस्य अङ्गसाद अकालज वली पलित दर्शन प्रभृतयो रसदोषज विकाराः।(Su.Su. 25)

Here he added the Avipaka, Angamarda, Trpti. And he used the word Anna Asraddha instead of Asraddha, Pandu Roga instead of Pandutva, Margoparodha instead of Srotorodha. Along with this patient suffers from wasting or karshya, loss of interest in food, pain all over body, premature wrinkling of skin, and graying of hair.

रसवह स्रोतस् विद्ध लक्षण

तत्र विद्धस्य शोषः प्राणवहविद्धदच्च मरणं तल्लिङ्गानि च।।

Prana vaha sroto viddha lakshana- aakroshana, vinaman, mohana, bhramana, vepana and marana.

5. Raktavaha Srotas

The channels which carries शोणित is called as रक्तवह स्रोतस्

It can be correlated to the circulatory system & Haemopoictic system.

स्रोतो मूल

1) According to Caraka:

शोणितवहानां स्रोतसां यकृत्प्लोहा च। (च.चि. ५/८)

The Moola of Raktavaha srotas is Yakrut and pleeha

2) According to Susruta:

रक्तवहे द्वे तयोर्मूलं यकृत् प्लीहनो रक्तवाहिन्यस्च धमन्यः। (सु.सु. ९/१२)

The moola of raktavaha srotas is Yakrut, (Liver) Pleeha (spleen) and Raktavahi dhamani (Blood vessels).

Ayurvedic Understanding of प्लीह and यकृत्:

यकृत्

- It is one among the koshtangas.
- It is considered as a मातृज अवयव

- Its sthana is below the हृदय in right side and occupies most of the righthypochondrium and epigastric region. It is also considered as the seat of ranjaka pitta.

Synonyms:

ज्योतिस्थान, कलाखण्ड, अग्निस्थान

प्लीहा:

- Considered as one among the koshtangas. (acco. to Caraka and Vagbhata).
- Involved in the production of the blood during intra uterine life.
- It is located in the left side of the abdomen and is related with the production and destruction of blood.

Rakta Production

रसात् रक्तं प्रजायते। (च.चि. १५/५)

That is raktadhatu is formed from rasadhatu. The प्रसादभाग of rasa which is watery in colour reaches यकृत् and प्लीह acted upon by ranjaka pitta gives red colour to it and thus rakta dhatu forms. Sthana of ranjaka pitta is यकृत्, प्लीहा, आमाशय and हृदय।

Modern Aspect

- Related with haemopoiesis

Definition:

The production and maturation of the formed elements of the blood is known as haemopoiesis.

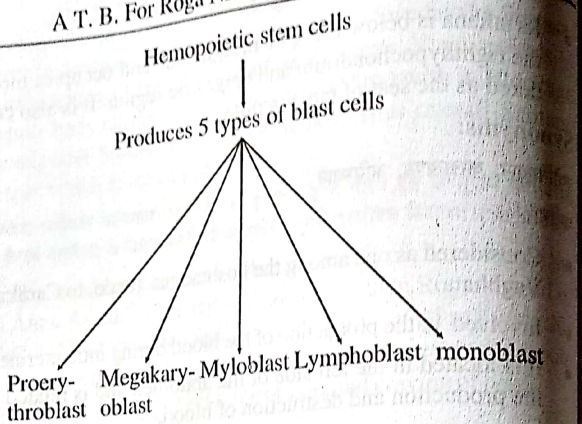
The formed elements are:

- RBC
- WBC
- Platelets

It starts from the third week of intra uterine life.

In the fifth month it takes place in red bone marrow (RBM); mainly in humerus and femur.

Haemopoietic stem cells are present in these bones which can renew themselves and can give birth to new cells.



The proerythrpblast differentiates and produces erythroblast (RBC).

- Myeloblasts differentiates and forms Neutrophils, oesinophils and basophils.
- Lymphocytes differentiates and produces mature lymphocytes.
- Monoblast differentiates to form monocytes and
- Megakaryoblast produces platelets.

Factors Required for Hemopoiesis

1) Colony stimulating factors
2) Interleukins

RBC Production ——→ Erythropoiesis

1) Production ——→ bone marrow
2) Growth and maturation ——→ by liver and spleen

Hemopoietic stem cells
↓
Proerythroblast
↓
Basophilic erythroblast
↓
Polychromatophilic erythroblast
↓

Acidophilic erythroblast
↓
Reticulocyte
↓
Red Blood Cells

RAKTA FUNCTION:

- जीवनं It is life of the living body.
- रक्तं वर्ण प्रसादं मंस पुष्टिं च करोति। (सु.स. १५/५)
  It gives complexion to the body and increases the bulk of the body.
- धातूनां पूरणं वर्ण स्पर्शज्ञानं असंशयम् ।
  It does poorana to dhatu's, gives complexion and sensation of touch.
- It gives nourishment to mala (malaroopi pitta) and upadhatu (kandara and sira) of it.

Modern Aspect

1) Nutrient function
2) Transport of respiratory gases.
3) Excretory function.
4) Transport of hormones and enzymes.
5) Regulation of water balance.
6) Acid base balance.
7) Regulation of body temperature.
8) Helps in the defense mechanism.

रक्तवह स्रोतो दुष्टि निदानः

विरुद्धान्नपानानि सिग्धोष्णानि द्रवाणि च। रक्तवाहिनी दूष्यन्ति भजता चातपानलो। (च.वि.५/१४)

- Intake of substances which induces burning sensation inside the body.
- Excessive consumption of .....
- Excessive exposure sun fire
- Injury by burn, heat, physical trauma.

दुष्टि लक्षण

दुष्यन्ते रक्तदोषजाः। कुष्ठविसर्पपिदका रक्तपित्तमस्रुग्धरः।।

गुदमेढ्रास्यपाकश्च प्लीह गुल्मोऽथ विद्रधिः। नीलिका कामला व्यङ्गः पिप्लुवस्तिलकालकः
दद्रुश्चर्मदलं श्वित्रं पामा कोटस्त्रमण्डलं रक्तप्रदोषाजायन्ते। (Cha.Su. 28)

The lakshanaas like kushta, visarpa, pidaka, raktapitta, asrukdara, gudapaka, medrapaka, asyapaka, pleeha, gulma, vidradhi, kamala, neelika, kamala, vyanga, pipplava, tilakalaka, dadru, charmadala, shwitra, paamaa, kota and asramandala will be observed.

विद्ध लक्षण

तत्र विद्धस्य श्यावाङ्गता शोणितागमनं रक्तनेत्रता च इति। (सु.स. ९/१२)

Blue colour to the body, fever, burning sensation, anaemia, bleeding, reddish discolouration to the eyes will be seen.

शोणितवह स्रोतो विकारः

मसूरिका, विसर्प, रक्तपित्त, रक्तधातुगतज्वर, विद्रधि, रक्तगुल्म, दाह, अर्श, कामला।

Conclusion

- Each dhatu is nourished by its on srotas only and each srotas will synthesis its own dhatu only.i.e,
- यदा स्वेदनोष्मणा पाकं शरीरं यान्ति धवतः स्रोतसा च यथास्वेन धातुः पुष्यन्ति धातुतः (च.चि. ८/...)

  Without the existence of srotas there will not be any synthesis or destruction/transportation of any biological elements in the body and thus nourishment.

6. Mamsavaha Srotas :-

- रसाद्रक्तंततोमांसंप्रजायते।

  Rakta is formed from rasa dhatu and maamsa from rakta dhatu
- वाय्वम्बुतेजसारक्तमूष्मणाच अभिसंयुतम्स्थिरतांप्राप्यमांसंस्यात्। (च.चि. १५/२८)

  Some portion of the rakta dhatu becomes solid by the effect of vaayu, teja and jala and this stira amsha is called as maamsa

मांसवहस्रोतोमूल

- मांसवहानां स्रोतसां स्नायुर्मूलं त्वक् च। (Ch.Vi. 5)
- मांसवहे द्वे तयोर्मूलं स्नायुस्त्वक्शोणितवाहिन्यश्च धमन्यः। (Su.sh. 9)

स्नायु

- These are the rope like structures.
- They are the binding material in the body to bind maamsa and ...
- As it helps in the function of maamsa like aakunchana and prasarana it is considered as one of the moola.

रक्तवहधमनि

- It is the structure in which rakta flows along with rasa.
- This helps in the nourishment of the maamsa.
- Hence, it is considered as one of the moola stana.

त्वक्

- मूलस्थानं इति प्रभावस्थानम्!
- As the seventh layer of twacha is maamsadhara and its continuation is maamsa in anatomy.
- The dusti of maamsa is expressed through twak.

  Therefore it is one among the moolasthana.

मांसस्वरूपः

- बहुल - large
- पेलव - small
- स्थूल - thick
- अणु - thin
- पृथु - flat
- वृत्त - round
- ह्रस्व - short
- दीर्घ - long
- स्थिर - firm
- मृदु - soft
- श्लक्ष्ण - slippary
- कर्कश - hard

मांसवहस्रोतो दुष्टिकारण

अभिष्यन्दिनिभोज्यानिस्थूलानिगुरूणिच। मांसवाहिनिदुष्यन्तिभुक्त्वाचस्वपतांदिवा।। (Ch.vi.5)

Intake of abhishyandi aahara, heavy food. After heavy meal sleeping during day time causes dushti of Maamsavaha srotas.

मांसवहस्रोतोदुष्टिलक्षण

अधिमांसार्बुदं कीलं गलशालूकशुण्डिके पूतिमांसालजीगण्डगण्डमालोपजिह्विकाः वेद्यान्मांसाश्रयान्
(Ca.Su. 28/14)

Excessive growth or mass formation, warts, fleshy projections, growth in root of tongue and palate, necrosed mass, sloughing, lymphadenitis, mumps, ranular growth.

मांसवहस्रोतोविद्धलक्षण

तत्रविद्धस्यश्वयथुर्मांसशोषःसिराग्रन्थयोमरणंच!

Symptoms of injury to mamsa vaha srotas are- oedema, piles, dystrophy, vascular tortion, cyst and death.

Functions of मांसवहस्रोतस

- Conduction Function
- Movement
- Tonicity

Formation of Muscle

it has similarities with moola sthana of maamsavaha srotas

Muscle

Skeletal (Snaayu) | Smooth (twak) | Cardiac (raktavaha dhamani)

7. Medovaha Srotas :-

- मेदोवहस्रोतस् is one of the स्थूलस्रोतस् and also अन्तर्मुखस्रोतस् explained in the ayurvedic treatise.
- Both charaka & sushrutha opines it as the 7th strotas in the order.
- It is the strotas which nourish & maintain the मेदधातु

स्रोतोमूल

मेदोवहनांस्रोतसांवृक्कौमूलंवपावहनं च। (Ch.vi.5/8)

१. वृक्क २. वपावहन

मेदोवहेद्वेतयोर्मूलं कटी वृक्कौ च। (Su.sa.9/12)

१. वृक्क २. कटी

स्रोतोविद्धलक्षण

तत्रविद्धस्यस्वेदागमनंस्निग्धाङ्गतातालुशोषःस्थूलताशोफःपिपासाच।।(su.sa.9/12)

a. स्वेदागमन : Excess sweat production
b. स्निग्धाङ्गता : More slimyness/oilyness of body parts
c. तालुशोष : Dryness of talu/palate
d. स्थूलता : Excess of fat deposition/obese
e. शोफ़ : Oedema (obstrustion in venous &lymphatic circulation)
f. पिपासा : Excess thirst/polydypsia

स्रोतोदुष्टिलक्षण

"निन्दितानिप्रमेहाणांपूर्वरुपाणि यानि च"। (Ch.su.28/15)

Ailments that cause decrease of ayu as told in atisthooladi asta nindita purusha, manifestation of prameha poorvarupa are the features of Medovaha Srotas dushti.

स्रोतोदुष्टिकारण

अव्यायमाहिवास्वप्नान्मेध्यानां चातिभक्षणात्। मेदोवाहीनिदुष्यन्ति वारुण्याश्चातिसेवनात्।।

- अव्यायमा : No exercise
- दिवास्वप्न : Day sleep

- अतिभक्षणात् : Excess intake of food like mamsa, and rich in fat
- वारुण्यतिसेवना : Excess intake of liquid items like madhya

What is vrukka, kati, vapavaahana? :-

Vrukka वृक्कौकुक्षिगोलकौमांसपिण्डब्दवंएकःवामपार्श्वश्चदक्षिणपार्श्वम्

Vrukka are two round structures made up of mamsa & it is present in पार्श्व ie; in lumbar region.

According to sharangadhara: vrukka is made up of प्रसादभाग of रस & मेद it nourishes the मेदधातु (mainly present in udara (abdomen).

Vapavaahana : Is a sheet like structure present in & around all the organs of body mainly in udara pradesha.

Kati: it is the region particular to the lower back part of abdomen.

Anatomical & physiological View

Assesment of Vrukka

- By above references we can say vrukka as kidney along with suprarenal glands.
- Kidney helps in the regulation of salts like sodium, potassium which are main component of bile juice secreated from liver, and does emulcification of fat which is digested and absorbed.
- Suprarenal gland secreates mineralocorticoids which acts on renal tubules in maintaining the level of sodium & potassium salts.

Assesment of Vapavaahana

- It is a sheet that is present in skin, blood, muscle, around umbilicus, heart & other internal organs which is made up of fat molecules & its function is to protect the under lining structures, this can be said as adipose tissue present in this area mainly omentum (policeman of abdomen), true & false capsule of internal organs.

Assesment of kati

It is a storage bag of fat present in lower back region .

8. Asthivaha Srotas :-

The channels which carries the substances which nourish asthi is called asthivahasrotas.

Which stays for long time, not easily get destroyed.

Formation of Asthi

Asthidhatu is formed by the medodhatu. In medodhatu so asthidhatusamanagunas are present. When medas comes and con with asthidharwagni. Those qualities are digested and convert into asthi, with the effect of agni, prithvi, app and vayu qualities medas become solid to form the asthidhatu.

Asthisankhya

Charaka-360 Sushruta-300 Modern classification-20

Asthivahasrotas is mentioned by charaka.

मूल

अस्थिवहानांस्रोतसांमेदोमूलंजघनंच।

The moola of asthivaha srotus is meda & jaganam

स्रोतोदुष्टिलक्षण

अध्यस्थिदन्तौदनास्थिभेदशूलांविवर्णता। केशलोमनखश्मश्रुदोषाश्चास्थिप्रदोषजा।।

Extra growth of bones, more growth in teeths, cracking pa in teeth and bones discolouration, deformities in kesha, loma, na

स्रोतोदुष्टिकारणा

व्यायामातिसंक्षोभादस्थ्यातिविघट्टनात्। अस्थिवाहिनीदूष्यन्तिवातलानांचसेवनात्।।

The karanas are excess indulgence in exercise, intake of va aggravating aharas,excessive exhaustion, excess of friction in bone due to injury.

Asthivaha Srotho Vikaras

Amavata	Sandhivata	Rajayakshma
Kushta	Jvara	Dantaroga
Vatarakta		

Bones

Bones are specialized types of connective tissues undergoing various changes and consist of cells and solid inter-cellular substance containing calcium salt.

Structure of Bone

Periosteum-outer covering
Compact part-outer part

Spongy part-inner
Medullary cavity-inner cavity or interior of long bones lined by endosteum

Composition of Bone

Organic Inorganic

Fibres, calcium and megnisium ions, crystals, carbonates, caphosphate (fine collagen).

Ossification

Process of formation of bone and it includes proliferation of collagen and ground substance with subsequent deposition of calcium salt.

Cell Types

Osteoblasts- concerned with bone formation
Osteocytes- maintaince of bone
Osteoclast- concerned with bone resorption

Medas as a moola of asthivaha srotus– Formatiom of asthi is from meda, so can be considerd as moola. i.e. the compact bone has a large marrow cavity called as medullary or marrow cavity which contains yellow bone marrow. The main function of this is in storage of fats. The spongy one has the bone marrow inside called saraktameda, site for production of blood cells.

Jaganam as moola of asthivaha srotus– The word meaning is pelvis or hip region. Jaganam in body has a main role in weight bearing. The function of asthi is also dharana and is controlled by this region. For a man to stand erect this region is very important and must be strong. Any injury makes it difficult to be erect.

9. Majjavaha Srotas :

Channels which carry the substance to nourish majja dhatu is called majjavaha srotas.

मूल

- मज्जावहानांस्रोतसामस्थिनिमूलंसन्धयश्च।। (च.वि. ५/८)

The moola of majjavaha srotas is asthi and sandhi.

Majjavaha sroto moola is only explained by Acharya Charaka. Acharya Sushruta haven't explained because each asthi is

मूर्च्छा- unconsciousness
तमदर्शन- darkness of eyes; अरुषं = Small wounds above joints
Kapha is ashrayee to majja, increased kapha causes tamas
And also the smaller/bigger joints are prominat.

मज्जवहस्रोतोविकार

सन्धिवात	आमवात	पक्षाघात
वातरक्त	कुष्ठ	राजयक्ष्मा
प्रमेह	मसूरिका	विषमज्वर

Modern co-relation of Majjavaha srotas:

Structure of Bone Marrow

The bone marrow measures about 4000ml in adults, 50% of this is RBM and the remaining is yellow bone marrow.

RBM:- The epiphysis of the femur, humerus and spongy bone of the skull bone and vertebra are filled with RBM.

YBM:- All the other parts like the shaft of the long bones contain YBM.

Functions of Bone Marrow

1. Destruction of old RBC's:- The old RBC's are destroyed by macrophages of RBM and the iron content of Hb of the destroyed Rbc's is split into two compounds i.e,Haemozederin and Ferretin. These 2 compounds are stored in liver, spleen and bone marrow for the re-synthesis of Hb.
2. Formation of blood cells:- All the 3 types of blood cells i.e, erythrocytes, leucocytes and platelets are formed by the RBM.
3. Storage of iron:- Iron in the form of haemosederin is stored in the RBM.
4. Macrophages destroy the toxins circulating in the blood.
5. Nourishment to the bone:- All the 3 types of bone cells i.e, osteocytes, osteoclasts and osteoblasts are produced from the bone marrow itself.

10. Sukravaha Srotas :-

शुक्र

It is one among the 7 dathus, it is a drava dathu.

"Majjath Sukram Prajaayate"

The prasada bhaga or essence of majja oozes out through small pores in bones & goes to the gonads, this later forms sukra.

According to Sushruta

सप्तमी शुक्रधरानाम, या सर्वप्राणीनां सर्वशरीरव्यापिनी।।

The 7th kala; sukradharakala is present all over the body. So that sukra is also considered to be present all over the body. He gives a simily like ghee is present in milk & jaggery in sugarcane, the sukra is present in our body.

Only difference he said was that in purusha it's in sthoola form & sukshma form in stree.

Sukravaha Srotas

Moola

शुक्रवहानां स्रोतसां वृषणं मूलं शेफश्च। (चरक)

He says that since vrishna helps in storage of sukra & shefa for the ejaculation these can be considered as moola.

शुक्रवहे द्वे तयोर्मूलंस्तनौ मूलं वृषणं च।। (सुश्रुत)

Vrishna has the same explaination for being the moola as above. Sthana even though not directly related to sukra its said to be a sensitive part & believed to stimulate the ejaculation.

शुक्रवहानां मूलः स्तनौ मुष्कौ मज्जा च। (वाग्भट)

Vaagbhatta adds majja to the list as its said that sukra is originated from majja. Mushka is another term for vrishna.

दुष्टि कारण

अकालयोनिगमनान्निग्रहादतिमैथुनात्। शुक्रवाहीनि दुष्यन्ति शस्त्रक्षाराग्निभिस्तथा।।(Ca.vi.5/19)

अकालयोनिगमनं– Intercourse at improper time

निग्रहाद– Veghadhaarana of shukra

अति मैथुनात्– Exessive coitus

शस्त्रक्षाराग्नि– Trauma due to instrument (shastra), kshaara karma, (alkalics or corrosives) agni karma (burn, thermal injury)

Also exess intake of अम्ल, तिक्त कटु रस, worry, grief, oldage, fear, anger, toxin etc. can cause morbidities of shukra.

विद् लक्षण

तत्र विद्स्य क्लीबता चिरात् प्रसेको रक्त शुक्रता च।। (सु.शा. ९)

Injury to moola cause,Impotancy,delay in ejaculation&some times blood mixed shukra is ejaculated.

दुष्टि लक्षणा

फेनिलं तनु रूक्षं च विवर्णं पूति पिच्छिलं। अन्यधातु उपसृष्टं अवसादि तथाऽष्टमम् ।। (च.चि. ३०/१३९-१४०)

Mainly 8 types of दुष्टि लक्षणाल are told

फेनिलं -frothy semen

तनु -thin semen

रूक्षं -dry

विवर्णं -discoloured

पूति-with putrid/offensive odor

पिच्छिलं - very much slimy (prostatic secretion-milky)

अन्यधातु उपसृष्टं -mixed with other dhatus like raktha mamsa etc.

अवसादि -semen sinking to bottom when placed over water.

Due to suppression of manifested urge for sex, semen get obstructed by aggravation of vayu, form knotty (grathitha) and अवसादि (ejaculated with difficulty) may be density increases.

Acc. to susruta, in shaareera sthana. 2nd chapter.:

Men with shukra vitiated with tridoshas, will have

Semen with cadaveric smell, knot formation, pus mixed (पूय शुक्र) deficient, smell of urine or faeces. Such people will be sterile and impotent.

शुद्ध शुक्र लक्षणा

स्फटिकाभं द्रवं स्निग्धं मधुरं मधु गन्धी च। शुक्रमिच्छन्ति के चित्तु तैलं क्षौद्रं निभं तथा।।

स्फटिकाभं– Crystal clear like quarts

द्रवं– fluid

स्निग्धं– Unctous

मधुरं– Sweet

मधु गन्धी– Smells like honey

शुक्रमिच्छन्ति के चित्तु तैलं क्षौद्रं निभं तथा– Also resemble like clear oil or honey in appearance.

Modern Co-Relation

- Vas deference
- Prostate

Vas deference: It is about 45 cm long. Ascends along the posterior border of epididymis, passes through inguinal canal & enters pelvic cavity.

Passes posterior to the uterus & forms a dilated part called Ampulla.

It joins with end of seminal vesicle to form the ejaculatory duct without terminating in prostate urethra, where sperm is ejected with the seminal vesicle, secretion & semen moves from urethra to exterior

Prostate: It is a cloughnut shaped gland, inferior to urinary bladder&surrounded by prostate urethra Secretions: milky-citric acid -sperm use it for ATP production.

Seminal plasma -antibacterial action

Spermatogenesis

(Spermatogonia A) Germ cell (2n diploid) [44+x+y]

↓ Mitotic division

Sprmatogonia B (2n) [44+x+y]

↓ Mitotic division 2

Primary spermatocytes [44+x+y]

↓ Meiotic division 1

Spermatocytes(22+x) (22+y)

(22+x) (22+x) (22+y)(22+y) [spermatid]

Mature into

Spermatozoa or sperms

11. Aartavavaha Srotas :

A channel which carry the aartava is called आर्तववहस्रोतस्. It is explained by sushruta only, giving two mula sthana for it.

Charaka did not explain as he already told about shukravaha srotus which include both shukra and aartava.

मुलस्थान

आर्तववहेद्धेत्योर्मुलंगर्भाशयआर्तववाहिन्यश्च धमन्यः (सु.शा. ९/१२)

1. गर्भाशय 2. आर्तववाहिन्यस्धमन्य

Uterus (गर्भाशय)

Uterus is child bearing organ in female and which protects and provides nutrition to a fertilized ovum.

Location

के Pelvis , between bladder and rectum.

Menstrual Cycle

It is a cyclic changes taking place in the structure of female reproductive tract, notably in ovary and endometrial wall of the uterus by the influence of progesterone and estrogen.

It consists of 3 phase:
1. Bleeding phase
2. Secretory
3. Proliferative

This phase occurs due to decline of both the hormons. It is also called as weeping of uterus as during this phase ovum is going to be released.

Duration: 5 - 7 days.

The functional layer of the endometrium is going to shed off i.e, from the endometrium becomes ischaemic and starts being shed. The vessel wall gets necrosed and blood enters the stroma and menstrual flow start.

So garbhashaya provide the pathway for the removal of menstrual of blood.

Fallopian Tube (Arthava Vahini Dhamani)

These are the hollow tubes present in female.

They carry ova from ovary and sperm from uterus to the tube for fertilization.

Location

Free upper border of broad ligament.

Parts

1. Uterine part
2. Isthmus
3. Ampula
4. Infundibulam

Physiologically

At the 14th day of menstrual cycle i.e. at ovulation day,there is the release of ovum from ovary, this ovum is carried through this fallopian tube and if sperm enters it is also carried through this for fertilization but if there is no entrance of sperm, then the released ovum degenarates and sheds down as a menstrual blood.

विद्धलक्षण

तत्रविद्धायावन्ध्यत्वंमैथुनासहिष्णूत्व.....(सु.शा.९)

वन्ध्यत्व- sterility or incapable for reproduction

मैथुनासहिष्णूत्वं- unbearable pain during coitus (dysperunia)

.....loss of aartava (amenorrhea)

For the वन्ध्यातव, Harita Samhita has told mainly 6 reasons:

1. गर्भकोषभंग - It can be considered as acquired disease
2. धातुक्षय - General condition of the body,due to improper nutrition, rakyashya,panduroga, vrdha avastha etc.
3. काकवन्ध्य - One child sterility i,e one child borne and got sterility due to infection after 1st delivary or any other disorder in fallopian tubes.
4. अनपत्या - Absolute sterility i,e those who have not conceive at all or lose the capiblity to reproduce from the young time,mainly as congenital disease.
5. गर्भस्राव - Delivary times come at 4th or 5th month of pregnancy.
6. ..... - At the 7th month of pregnancy delivary occurs of a dead baby. Or after delivary, there is no proper functioning of the heart of the baby.

12. Mutravaha Srotas

मूल

"मूत्रवहानां स्त्रोतसां बस्तिर्मूलं वंक्षणौ च"।। (च.वि. ५/२८)

The moola of the muthravaha srothas are basthi , vankshana

दुष्टि लक्षण

"अतिस्रुष्टं अतिबद्धं प्रकुपितं अल्पाल्पं अभीष्णं वा बहलं सशूलम् मूत्रयन्तं दृष्ट्वा मूत्रवाहिन्यस्
स्त्रोतांसि प्रदुष्टानि इति विद्यात्।।" (च.वि. ५/३२)

The dushti lakshanas of the muthravahasrothas are,

अतिस्रुष्टं - Increased frequency of urination

अतिबद्धं - Difficulty in urination

प्रकुपितं - change in the normal quality of the urine

अल्पाल्पं -Very less flow of the urine

बहलं - Large quantity of the urine flow

सशूलं -Painful micturition

मूत्रवह स्त्रोतोदुष्टि कारण

"मूत्रितोदक भक्ष्य स्त्रीसेवनान्मूत्रनिग्रहात्। मूत्रवाहिनी दुष्यन्ति क्षीणस्य अभिक्षतस्य च।।"

The dushti factors of the moothravahasrothas are,

- मूत्रितोदक भक्ष्य स्त्री सेवनं -A person who has a desire to void the urine, when he drinks the water, eats the food and during sexual intercourse.
- मूत्रनिग्रहात्–If suppress the urge for micturition
- क्षीण– Those who are very weak
- अभिक्षत्–Those who are subjected to injuries

मूत्रवहस्त्रोतस् as per सुश्रुत,

मूत्रवहेद्वेतयोर्मूलंबस्तिमेढ्रंच।।

According to susrutha the moolas of moothravaha srothas are Basthi and medra

विद्दलक्षण of मूत्रवहस्त्रोतस् (सुश्रुत)

"तत्रविद्दस्यान्अनद्दबस्तितामूत्रनिरोधः स्तब्धमेढ्रताच।।"

If any injury happen to moothravahasrothas leads to,

- अनद्दबस्तित– Distention of the bladder
- मूत्रनिरोध– Obstruction to the flow of urine
- स्तब्धमेढ्रता– Stretching of penis

Anatomical Elaboration of Kidney

The paired kidneys are reddish, bean shaped organs located just above the waist between the peritoneum and posterior wall of the abdomen, and it is a retroperitonial organ.

EXTERNAL ANATOMY OF THE KIDNEY

Length=10-12 cm Thickness=3 cm

Width=5-7 cm Mass=135-150g

There is a deppression in the medial side of the kidney known as renal hylum (Through which the blood vessels and ureter emerges out).

Kidney is having 3 layers.

1. Renal capsule 2. Adipose tissue 3. Renal fascia.

Internal Anatomy of the Kidney

The frontal portion of the kidney consists of 2 regeons,

1. Renal cortex 2. Renal medullae

Renal medullae consists cone shaped ranal pyramids.

Physiological Aspects of the Kidney

The ranal system or urinary system is the one having maximum capacity of excretory function and so it plays the major roll in.

Homeostasis

Renal system includes,

- A pair of kidneys
- Ureters
- Bladder
- Urethra

Nephron

Nephron is the structural and the functional aspect of the kidney it is having 2 parts.

- Renal corpuscle
- Renal tubule

Renal corpuscle- It is the first part of the nephron.

Filteration of the blood or the first phase of the urine formation is taking place here.

It is having 5 parts.

1. Efferent arteriole
2. Afferent arteriole
3. Glomerulus
4. Visceral layer
5. Parietal layer

Based on the position of the ranal corpuscle the nephron divided into 2.

- Cortical nephron
- Juxta medullary nephron

Cortical nephron- Renal corpuscle lies on outer cortex near the periphery.

Juxta medullary nephron - Renal corpuscle lies on the inner cortex near to the medullae.

Structure of the Renal Corpuscle

It is having 2 parts, • Glomerulus • Bowman's capsule

Glomerulus-Tuft of cappoillaries enclosed by the Bowman's capsule, connected to the afferent and efferent arterioles.

Bowman's capsule-Capsular structure encloses the Glomerulus

It has 2 layers,

- Inner visceral
- Outer parietal

Tubular portion of the nephron consists of 3 parts,

- PCT (Proximal convoluted tubule)
- Loop of Henle (Hairpin bend)
- DCT (Distal convoluted tubule)

Loop of Henle-

Descending limb

Ascending limb

Descending limb- Thick and thin

Ascending limb- Thick and thin

Juxtaglomerular Apparatus

Juxta glomerular apparatus is a specialised organ situated near to the glomerulus of the each nephron. It consists of-

- Macular densa
- Extra glomerular messengial cells
- Juxta glomerular cells

Function- Secretion of the hormonal susbstances

Regulation of the glomerular blood flow.

Functions of the Kidney

1. Roll in homeostasis

- Excretion of the waste products
- Maintanace of the water balance
- Maintanace of the electrolyte balance
- Maintanace of acid-base balance

2. Haemopoetic function.
3. Endocrine function
   - Erythropoetin • Calcitriol
   - Thrombopoetin • Prostaglandins
   - Rennin
4. Regulation of the blood pressure.
5. Regulation of the blood calcium level.

Urine Formation

According to Ayurveda

मूत्राशयमलाधारप्राणायातनमुत्तमं। पक्वाशयगतस्तत्रनाड्योमूत्रवहासिषु।।
तर्पयन्तिसदामूत्रंसरितः सागरंयथा। सूक्ष्मत्वान्उपलभ्यन्तेमुखन्यासांसहस्त्रशः।।
नाडिभिः उपनीतस्यमुखस्यामाशयान्तरात्। जाग्रतः स्वपतैश्चैवसनिस्यन्तनेनपूर्यते।।
आमुखात्सलिलेन्यस्तः पार्श्वेभ्यः पूर्वन्तेनवः। घटोयथातथाविद्दिबस्तिमूत्रेणपूर्यते।। (सु.नि.३/२९)

Vessels arising from large intestine, called moothravahini nadi, fill the urinary bladder like rivers feed water to ocean, oceans on earth. They are so minute and not to be perceived. These vessels arise from amasaya and convey fluid to bladder day and night. Thousands of these vessel provides water. which exudes from vessels to inside bladder.

According to Modern Science

Urine formation is a blood cleansing function.

Normally about 26 % of cardiac output enters the kidneys to get rid of unwanted substances, kidneys excretes the unwanted substances along with water as urine.

About 1-1.5 litres of urine is formed everyday.

Urine formation consists of 3 stages.

- Glomerular filteration
- Tubular re- absorption
- Tubular secretion

1. Glomerular Filteration

The process by which blood passes through glomerular capillaries is filtered through filteration membrane.

Process of glomerular filteration :

Blood passes through glomerular capillaries

↓

Plasma filtered into the Bowman's capsule

↓

All the substances of the plasma filtered exept plasma protein

↓

This filtered fluid is **glomerular filterate.**

Ultra filteration : The glomerular filteration is also called as Ultra filteration.

Glomerular filteration rate : Total quantity of filtrate formed in all the nephrons of the each kidney in the given unit of the time.

Normal GFR =125ml/min

2.Tubular re-absorption

In this process by which water and other substances transported from renal tubules to the blood.

While the filterate is passing through the tubular portion of the nephron both quantitative and qualitative changes of the blood will be occur.

↓

Large quantity if water (99%) and electrolytes absorbed by tubular epithelial cells.

↓

The re absorbed substances will go to the renal medullae.

↓

From there it will move to the blood in peritubular capillaries

- Since the substances are taken back into the bllod from the glomerular filterate, the entire process is known as TUBULAR RE-ABSORPTION.
- Tubular re-absorption by PCT = About 7/8 of the filtrate is reabsorbed by PCT.

The substances which are re absorbed by the PCT are- Glucose, Amino acids, Na, K, Ca, Bicarbonates, Chlorides, Phosphates, Uric acid and water.

- Substances are absorbed from the Loop of Henle -Na, Chloride.
- The substancesare absorbed from the DCT- Na, Ca, Bicarbonate, water.
- Collecting duct- Na, HCO, K

3. Tubular secretion

The process by which the substances are transported from blood into renal tubules.

In addition to re absorption from the renal tubule some substances are also secreted into the lumen of the renal tubules.

The substance swhich are usually secreted out are,

- Para amino hippuric acid
- Diodrast
- Hydroxy indole acetic acid
- Amino derivatives
- Pencilline.

Substance sescreted in different segments of the renal tubule

- Active escretion of potassium by sodium-potassium pump in PCT, DCT, Collecting duct.
- Ammonia is secreted in the PCT
- Hydrogen ions by PCT and DCT

Ureter/ गाविनि

According to atharvaveda

"यथात्रेषुगविन्योर्यत्वस्तावधिसंश्रितं।।"

The fluid which is conveyed by 2 vessels called gavini , from intestine to urinary bladder and remains there is called mutra.

According to Modern

Each of the 2 ureters transports the urine from the renal pelvis of 2 kidneys towards the urinary bladder.

Length = 25-30 cm

Ureters are thick walled narrow tubes that vary in diameter from 1 mm to 10 mm.

It is a retroperitoneal organ.

It is connected to urinary bladder posteriorly.

Peristaltic contractions of the muscular walls of the ureter push urine towards the urinary bladder, but hydrostatic pressure and gravity also contribute.

Urinary Bladder/बस्ति

- मूत्राशयोबस्तिर्नाम। (सु.शा.)

Organ which stores urine is called basthi.

According to Modern

The urinary bladder is a hollow, distensible muscular organ situated in the pelvic cavity posterior to the pubic symphysis.

In males it is directly anterior to the rectum and in females it is anterior to thevagina and inferior to the uterus. And it is smaller in females.

Capacity =700-800 ml

Anatomy and physiology of the urinary bladder

In the floor of the urinary bladder there is a small triangular area called as TRIGONE.

posterior to this there will be two ureteral openings

In the Anterior corner there is oppenig to the urethra.

There are 3 coatings for urinary bladder.

1. Deep layer mucosa 2. Transitionalepithelium

3. Lamina propria

Rugae are also present. (To permit expansion of the urinary bladder)

Around the opening of the urethra the circular fibres form an

- Internal urethral sphincter
- External urethral sphincter (inferior to the above one.)

These structures helps for the elimination of urine to out.

Function of the Urinary Bladder

Collection and storage of the urine.

Urethra/मूत्रप्रसेक

मूत्रप्रसेकोनाममूत्रम्येनबस्तिमुखाश्रयेणास्रोतसाक्षरति। (डल्हण com. on सु.चि.)

From the moothravaha srothas urine is expelled from opening of the urinary bladder, the channel through ahich this is done is named as moothrapraseka/urethra.

According to Modern

It is small tube leading from the internal urethral orifice in the floor o the bladder to the exterior of the body.

In both males and females it is the terminal part of the urinary system, and passageway for the discharge of the urine, in males discharge of semen as well. Length = 4 cm.

Micturition

Micturition is the process of voiding of the urine.

Filling of the urinary bladder.

↓

Stimulation of the stretch receptors

↓

Afferent impulses via pelvic nerve

↓

Sacral segment of the spinal cord

↓

Efferent impulses via pelvic nerve

↓

Contraction of the detrusor muscles and relaxation of internal sphincter

↓

Flow of urine into urethra and stimulation of stretch receptors

↓

Afferent impulses via pelvic nerve

↓

Inhibition of the pudental nerve

↓

Relaxation of the external sphincter

↓

Voiding of the urine

Clinical Conditions Related to the Urinary System

1. Renal Calculi

- Its due to the accumulation of the mineral salts.
- It can lodge anywhere along the course of urinary tract
- Accumulation of uric acid, phosphate and calcium oxalate as salts
- Occurs crystal formation

2. Hydronephrosis

- It is the swelling of the kidney
- Due to the accumulation of the water inside the kidney
- Distention and dilation of pelvis and calyxes caused by obstruction of free flow of urinefrom the kidney.

3. Glomerulo Nephritis

- Damage to the glomerulus
- Cause-streptococcal infection of throat leads to this.
- It can be due to hereditary, or due to high BP. .

4. Pylonephritis

- It is the kidney inflammation or infection
- Inflammation of the pelvis and calyx
- Cause-E.choli (bacteria)

5. Renal Failure

- Occurs when excretory function of the kidney failures
- Cause-Diabetes mellitus and also due to hypertension

6. Nephrotic Syndrome

- Occurs when the filters in the kidney leaks the protein

13. Pureeshavaha Srotas :-

विण्मूत्रमाहरमलः।। (सु.सू. ४६/२३)

The pureesha and mootra are formed by the food, after the digestion of food, the saara bhaga gets absorbed and the remaining undigested and indigestible food becomes solid and that is called as pureesha.

पक्वाशयंतुप्राप्तस्यशोष्यमाणस्यवह्निना। परिपिण्डितपक्वस्यवायुःस्यातकटुभावतः।।(च.वि.२५/२०)

With the help of vaayu, the remaining undigested and indigestible food comes to pakvasaya (large intestine) in the pakvasaya that part become solid by the effort of agni and vayu, this paka is called as paripinditapakwa.

Function of Pureesha

पुरीषमुपस्तंम्भवाय्वाग्निधारणञ्च।। (सु.सू. २५/८)

Pureesha is called as upasthambha, that which bears the body, along with this function pureesha also bears the vayu, which is very essential for life. Susruta also says avashtambha pureeshasya, means its maintains the bearing capacity of body.

The pureesha vega means desires/urge to pass the faecal matter, is one of the adhaaraneeya vega, one should not suppress this vega, it suppressed it leads to abdominal pain, head ache, constipation, pain in the calf muscle etc.

पुरिषवह स्रोतो मूल

- **पुरीषवहानं स्रोतसां पक्वाशयो मुलं स्थुलं गुदं च** (च./वि)

  पक्वाशयं : Large intestine

  स्थुल गुद : Anus/rectum

- **पुरीषवहेद्वेतयोमूलंपक्वाशयंगुदंच।** (सु.शा. ९)

  Moola of pureesha vaha srotas is

1. Pakwashaya : Production of पुरीष
2. Guda : Exeretion of पुरीष

वाग्भट : स्थूलान्त्र instead of **स्थूल गुद**

दुष्टिकरण

संधारणादत्यशनादजीर्णध्यशनात्तथ। वर्चोवाहिनीदूष्यन्तिदुर्बलाग्नेःकृशास्यच।। (ch.vi.5)

The dushti janaka factors of pureeshavaha srothas are,

संधारणा : Holding the urge of vegas

अत्यशन : Over eating

अजीर्ण : Due to indigestion

अध्यशन : Eating before the digestion of previous food

दुर्बलाग्ने : Due to weak digestive power

दुष्टिलक्षण

प्रदुष्टानांतुखलुएषामिदंविशेषविज्ञानंभवतितथ्यताकृच्छ्रेणाल्पाल्पसशब्दशूलमतिद्रवमति-ग्रथित्मतिबहुचोपविशनंदृष्ट्वापुरीषवहान्यस्यस्रोतांसिप्रदुष्टानितिविद्द्यात।। (च.वि./५)

When the moolas are undergoing dushti it leads to,

कृच्छ्रेण : With difficult

अल्पाल्पं : Less quantity

सशाब्दं : Bowels passing with sounds

शूलं : Bowels passing with pain

अतिद्रव : Very watery stool

अतिग्रथित : Hard stool

अतिबहु : Large quantity of stool will be expelled out

विद्धलक्षणा

तत्रविद्धस्यानाहोदुर्गन्धितामन्थितान्त्रताच।। (सु.शा.)

आनाह : Distention with obstruction ग्रथितान्त्रता : Fecoliths

दुर्गन्धता : Foul smelling stool

चिकित्सा

स्वेदाभ्यंगावगाहखवर्तयोबस्तिकर्मच।। (च.सू. ७/९)

स्वेद : Sadation अवगाह : Bath to the body

अभ्यङ्ग : Oil anointing बस्तिकर्म : Inserting wicks in to the rectum

Large Intestine

The large intestine is also know as colon . it extends from ileocecal valve up to anus . it consists

- Cecum with appendix
- Ascending colon
- Transverse colon
- Descending colon
- Sigmoid colon/pelvic colon
- Rectum
- Anal canal

Functions of Large Intestine

1. Absorptive Function

Large intestine plays an important role in the absorption of various substances such as,

- Water
- Electrolytes
- Alcohol
- Drugs like anesthetic agents, sedatives & steroids

- Organic substances like glucose

2. Formation of Feces

After the absorption of nutrients, water & other substance, the unwanted substance in the large intestine from feces this is excreted out.

3. Excretory Function

Large intestine excretes heavy metals like mercury, lead, bismuth, & arsenic through feces.

4. Secretory Function

Large intestine secretes mucin and inorganic substance like chlorides and bicarbonates.

5. Synthetic Function

The bacterial flora of large intestine synthesizes folic acids, vitamin B_{12} and vitamin K by this function large intestine contributes in erythropayotic activity and blood clotting mechanism.

Formation of Pureesha

- By the time chyme has remained in the large intestine 3-10 hrs it has become solid/semisolid because of water absorption and is now called feces.
- Chemically, feces consists of water, inorganic salts, epithelial cells from the mucosa of the gastro intestinal tract, bacteria, products of bacterial decomposition, unabsorbed digested materials and indigestible parts of food.

Although 90% of water absorption occurs in the small intestine, the large intestine absorbs enough to make it an important organ in maintaining the body's water balance, of the 0.5-1L of water that enters the large intestine, all but about 100-200ml is normally absorbed via osmosis. the large intestine also absorbs ions including sodium, chloride, and some vitamins.

Faeces

The feces are derived;

I. Mainly from the intestinal secretions and

II. Partly from the ingested food

(feces in starving animals are decreased in bulk differ comparatively little in composition from those of normally fed animals).

If vegetables and coarsely ground cereals are excluded fro diet, the feces have fairly constant composition.

Composition of Feces on an Average Diet

- Water : 75% of total fecal matter weight
- Solids : 25% of total fecal matter weight Percent of total soli

Cellulose and other indigested fibers. -30%

Ash ie inorganic material mostly compounds of ca, iron mg -15%

Fat and fat derivatives (fatty acids, neutral fats, cholic acid etc. -5%

Desqumated mucosal cells, mucus and small amounts o digestive enzymes (most which are dead).

Pathological Significance

- Increased bulk of this undigested residue stimulate intestina peristalsis which in turn increase the passage of food through the intestine.
- Similarly, increased cellulose content of food increase the bulk of the feces ie. Feces contain more water, solids, and more o ingested food. Thus high fiber diet plays an important role i prevention and treatment of constipation.
- High fiber diet helps reducing the sudden increase in blood glucose level after a meal.
- Reduce blood- cholesterol level by binding it with bile salts.

Dietary fibers

It helps to control all metabolic disorders associated with over nutrition such as obesity, atherosclerosis, hyper cholesterolaemia and diabetes mellitus.

In humans there is no appreciable digestion of dietary fibers

Eg: cellulose, hemi cellulose and lignin etc. due to absence of certain micro organisms in GIT which breaks down these substance.

Defecation

Voiding of feces is known as defecation.

Feces is formed in the large intestine and stored in sigmoid colon by the influence of an appropriate stimulus , it is expelled out through the anus. This is prevented by tonic constriction of anal sphincters in the absence of the stimulus.

Defecation Reflex

The mass movement drives the feces in to sigmoid/pelvic colon. In the sigmoid colon the feces is stored. The desire for defecation occurs when some feces enters rectum due to the mass movement . usually the desire for defecation is elicited by an increase in the intra rectal pressure to about 20-25cm water.

The usual stimulus for defecation is intake of liquid like coffee/ tea/water but it differs from person to person.

Pathway for Defecation Reflex

The afferent impulses travel in pelvic nerves and induce reflex parasympathetic discharge over the pelvic splanchic nerve to cause:

a) Inhibition (relaxation) of internal anal sphincter and,

b) Inhibition of discharge in somatic pudendal nerve this relaxes the external anal sphincter.

Voluntary Defecation

- Before the pressure that relaxes the external anal sphincter is reached, voluntary defecation can be initiated by voluntarily relaxing the external anal sphincter and contracting the abdominal muscles. thus aiding the reflex emptying of the distended rectum.
- Defecation reflex therefore a spinal reflex that can be voluntarily facilitated by relaxing the external anal sphincter and contracting the abdominal muscle.

(Here excitatory sympathetic innervations of internal anal sphincter are not involved, only the sacral segment of the spinal cord is concerned.)

In infants, defecation is a simple spinal reflex.

Anal sphincters

1) Internal 2) External

Internal/Involuntary Anal Sphincter

It consists of thickening of circular smooth muscle at pelvic-rectal flexure and is inverted by,

a) Parasympathetic pelvic splanchnic nerve are inhibitory.

b) Sympathetic nerves are excitatory. This sphincter relaxes when the rectum is distended.

External/voluntary Anal Sphincter

- It consists of somatic skeletal muscle it is inverted by pudendal nerve and is maintained in a state of tonic contraction. mild to moderate distention of rectum increases the force of its contraction, where as moderately severe distention of rectum relaxes it.
- The first urge to defecation occurs when rectal pressure increase to about 20mmHg by stimulation of receptors in the rectal wall. when the rectal pressure increase to about 55mmHg, both the anal sphincters relaxand the contents of the rectum are expelled.

14. Swedavaha Srotas :-

Acc to charakacharya it is the 13th srothas.

As we all know sweda is considered as one of the waste product of the body.

It is formed because of the meda which is undergoing paka inside the body.

It is important to eliminate the sweda thus formed out of the body.

Mula

स्वेद वहानां स्रोतसां मेदो मूलं लोमकूपश्च

Adipose tissue and hair follicle are the sites of origin of sweda vaha srothas.

Dushti Lakshana

प्रदुष्टानां तु खल्वेषामिदं विषेश विज्ञानं भवति तध्यथा अस्वेदनमतिस्वेदनं। पारुष्यमतिश्लक्ष्णतामनास्य परिदाहं लोमहर्षश्च च दृष्ट्वा स्वेदवहन्यस्य स्रोतांसि प्रदुष्टानिति विध्यात्। (Cha vim)

The symptoms manifested due to vitiation of channels of sweda is-

Asweda	: No perspiration
Atisweda	: Excessive perspiration
Parushya	: Roughness of the skin
Athislakshna	: Excessive smoothness
Angasya paridaham	: Burning sensation of body part
Lomaharsha	: Falling of hair

Dushti Karana

व्यायामादतिसंतापाच्छीतोष्णाक्रम सेवनात्। स्वेदवाहिनि दुष्यन्ति क्रोधशोकभयेस्तथा।। (च.वि.)

Sweda vaha sroths get vitiated due to following causes-

Ati vyayama: Excessive exercise

Ati athapa sevana: Excessive exposure to sun

Thiksna ushna anna sevanath: Intake of corrosive, hot stems.

Skin

We all know that skin is one of the important sensory organ.It is responsible for both protection and sensory perception.Skin is considered as the largest organ of our body. Also it plays an important role in the metabolic function of our body. Skin has got three layers. They are as follows epidermis, dermis and hypodermis. These three layers are formed one after other respectively. These layers have their own function to provide on the basis of both sensory and protection.

Epidermis

It is the outer most layer of the skin. It is avascular and it is nourished by diffusion from the dermis it contain four principle layer of cells. They are as follows keratinocyte, melanocyte, langerhans cell and merkel cell.

Epidermis layer of the skin is divided in to five sub layers they are-

- Stratum corneum
- Stratum lucidum
- Stratum granulosum
- Stratum spinosum
- Stratum basale

Dermis

It is the layer between the epidermis and the subcutaneous tissue that consists of- connective tissue and cushions the body from stress and strain. It consist of two layers the superficial area adjacent to the epidermis is called the papillary region and deeper thicker area is called the reticular dermis.

It is composed of three types of cell they are - matrix, elastin and glycosaminoglycans. The sebaceous gland and sweat gland present in the reticular layer of the dermis.

Accesory Structures of Skin

The accessory structures of skin are the structures that develop from the epidermis of an embryo. They are- Skin gland, Hair and nail. Hair and nail protects the body and sweat gland helps in regulate body temperature.

Skin Glands: There are three types of skin gland they are sweat gland sebaceous gland and ceraminous gland.Sebaceous gland secretes sebum secretary protein. It is present in the dermis, usually opens in to the neck of hair follicle.

Sweat Glands: They are small tubular structures of the skin that produce sweat. They are of two types they are- eccrine and apocrine sweat gland.

Eccrine gland	Appocrine gland
Present throughout skin	Present in axilla, pubic region
It regulates body temperature	Stimulates during emotional stress and sex
Onset is soon after birth	
Function throughout life	Non functional till puberty
Functions under nervous control	Functions under hormonal control

Adipose Tissue

Derived from fibroblast that are specialized for storage of triglycerides as large centrally located drop let fills up with a single large triglyceride drop let and cytoplasm and the nucleolus are pushed to periphery. As a result of this the weight of the adipose tissue increases and a new blood vessel. Thus an observe person has many blood vessels then does a normal person.

It is the thick layer under the skin, around the kidneys and in the buttocks.

More generally it is found at the same locations throughout body areolar connective tissue.

Functions of Adipose Tissue

- It act as an insulating layer and help to reduce heat loss through skin.
- It also has protective functions, providing mechanical protection and support around some major organ.

- It is also meant for energy storage

Mechanism of Body Temperature Regulation

When body temperature increases, blood temperature also increases.

↓

Thermo receptors stimulates in the brain

↓

The heat center bring the temperature back to normal by 2 mechanism

↓

1. Promotion of heat loss, 2. Prevention of heat production

↓

It stimulates the sweat gland and increases the elimination of heat

15. Manovaha Srotas :-

The Manovaha Srotas has not been separately mentioned in Charaka Samhita. It is however stated that the entire sentient body represents the abode of the manas and therefore all srotases of the body can be considered as the manovaha srotases.The manovaha srotases transport either the manas or the information related to it.

- शिरस्ताल्वन्तरगतम् सर्वेइन्द्रियपरम् मनः। (Bhe. chi. 8/2)

Since Bhela has clearly stated the location of the manas is the Shiras, hence it is to be assumed that these xÎÎiÂiìxìcarry the information related to manas.

स्थान of Manas

- Acc. To chakrapani - हृदयाश्रितत्वान्मनस: Manas is located in the हृदय.
- Acc. To Bhela - शिरस्ताल्वन्तरगतम् सर्वेइन्द्रियपरम् मन:। (Bhe.chi.8/2) It is located in the shiras.
- Acc. To Bhela - चित्तम् हृदयसम्श्रितम् Location of manas is in हृदय.
- The स्थान of manas as हृदय. requires further explanation.
- A close study of functions of manas clearly indicates that manas is located in the shiras only, but not in हृदय, and that it may function as हृदय in receiving information and transmitting that information to respective areas.
- The reference of Manovaha Srotas is found in the description of psychological disorders in the Ayurvedic Classics.

- The Manovaha Srotas has been described as follows in the Madhukosha commentary of Madhava Nidana.

मनोवहानि स्रोतांसि यद्यपि पृथक्प्रोक्तानिदा धमन्यो मनोवहा अभिधीयन्ते।

उन्मर्गमागता विमार्गमागता मनोवह धमनीरनुप्राप्ताम्।।

Madhukosha stated that although the separate anatomical description of manovaha srotas is not found, dasha dhamanis might be considered as channels of manas.

i.e. Dasha dhamani which are rooted to the ṛËSrī may be considered as manovaha srotas.

मनोवहानाम् पुर्णत्वात् दोषैरति बलैस्त्रिभिः। स्रोतसाम् दारुणान् कालेपयति दारुणे।। (च.चि.५/४१)

When the manovaha srotas are filled with the exceedingly aggravated tri doshas, one sees terrific dreams in ominous situations.

- **Ref. in Charaka chikitsa sthana**

तैरल्पसत्वस्य मलाः प्रदुष्टा बुद्धेर्निवासम् हृदयम् प्रदूष्य।
स्रोतांस्यधिष्ठाय मनोवाहिनि प्रमोहयन्त्याशु नरस्य चेतः।। (च.चि. ९/५)

In the samprapti of unmada, charaka has mentioned about the manovaha srotas.

प्रकुपित वातादि दोष (अल्पसत्व व्यक्ति)

dushta doshas goes to hrdaya and travel up and gets

↓

lodged in manovaha srotas.

↓

Then it goes and affects the manas.

↓

Ultimately causes unmada.

मनोवहा स्रोतो दुष्टि कारण

- Affliction of the mind by the predominance of rajas and tamas
- Intake of unwholesome and unclean diet, viruddha aahara, food touched by the unclean hands of the person suffering from contagious disease.
- Neglecting the prescribed dietic rules.
- When the manas is afflicted with emotional stress.

मनोवहा स्रोतो दुष्टि lakshana

- There will not be proper perception of knowledge.
- The person may suffer from dhi, dhriti, and smriti bhrama.
- The vitiation of manovaha srotases causes unmada and apasmara.
- The person sees terrific dreams.
- The vitiation of manovaha srotases may also cause disease related to pranavaha, annavaha, udakavaha etc. srotases. (psychosomatic diseases).

What is Mind?

- A mind is the complex of knowing or perceiving faculties that enables consciousness, thinking, reasoning, perception, and judgment. Some people think that the mind is the brain or some other part or function of the body, but it is not true. The brain is a physical object that can be seen with the eyes and that can be photographed or operated by surgery. While the mind on the other hand is not a physical object, it cannot be seen with the eyes, nor it can be photographed or repaired by surgery. Therefore the brain is not the mind but simply a part of the body. There is nothing within the body that can be identified as being our mind because our body and mind are different entities. E.g:- Sometimes when our body is relaxed, our mind can be very busy thinking about different topics. This indicates that our body & mind are not same entity.

In the Buddhist Scriptures, our body is compared to a guest house, and our mind as a guest who is dwelling in it.When we die our mind leaves our body and goes to the next life, just like a guest leaving a guest house and going somewhere else.So in simple words "Mind is a formless continuum that functions to perceive and understand objects.

Psychology

- "The scientific study of behavioral and mental processes is known as psychology.
- Here, behavior consist of wide range of observable actions, from simple movements like blinking, pointing, nodding etc. to a

very complex activities like eating, writing, talking, driving etc. While the mental processes includes perception, thoughts, memories, expectations, desires and feelings etc.

Relation Between Physical & Mental Health

- Mental health and physical health are fundamentally linked. People living with a mental illness are at greater risk of experiencing a wide range of physical health problems. The reverse relationship is also true: people living with chronic physical health conditions may experience depression, anxiety etc.

Why do mental and physical health conditions co-exist?

Both mind and body are affected by biological and emotional changes, as well as by social factors such as income and housing. These three pathways of biology, illness experience, and the social determinants of health can increase the likelihood of someone living with a mental illness or chronic physical condition developing a co-existing condition.

Relation of doshas of mind and body

Acc. To sharangdhar Samhita.

- Satwa Pitta
- Rajas Vata
- Tamas Kapha

The Subconscious Mind

Think of the subconscious mind as the storage room of everything that is currently not in your conscious mind. The subconscious mind stores all of your previous life experiences, your beliefs, your memories, your skills, all situations you've been through and all images you've ever seen.

Conclusion

By this we can conclude that the दश धमनी which are located in हृदय can be considered as Manovaha Srotas. By the above explainations we can conclude that the mind and the brain are different entity.And by the Explanations given by the Acharya's like Charapani, bhela, shela etc. we can conclude that the sthana of manas is shiras or hrdaya, but a close study of functions of manas says that shiras is the sthana of manas, hence we can conclude that the

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Chapter- 2

VYADHI VIGYANA

1. Definition, synonyms and classification of Vyadhi & Vyadhi Ghatak

Introduction

इहखलु पुरुषेणनुपहतसत्वबुद्दिपौरुषपराक्रमेण हितमिह चामुष्मिंश्च लोके समनुपश्यता तिस्र एषणाः पर्येष्टिव्या भवन्ति। तद्यता-प्राणैषणा धनैषणा परलोकैषणेति। (च.सू. ११/१३)

Dharma, Artha, Kaama, Moksha are chaturvidha purushaarthas.

For attainment of above one should have desire towards three aspects viz Praana Dhana and Paraloka.

There are lot many interruptions on the path of fulfillment of above.

Disease or vyadhi manifestation is one such interruption.

To get through this there evolved Trisutra Ayurveda consisting of Hetu, Linga & Aushadha. Knowledge of hetu and linga comprises Roga Nidana or diagnosis of disease which helps Aushadha.

Definition of Vyadhi

Introduction

The different types of causitive agents enters the body and interact with dosha, dhatu, Mala. As result the body elements will undergo vriddi, kshaya, prakopa, pradosha etc type of dushti and leads to disease or Vyadhi which is expressed in the form of pain or suffering (symptoms)

विकारो धातुवैषम्यं साम्यं प्रकृतिरुच्यते। (Ca.Su. 1/4)

The stage of dis equilibrium of doshas and dhatus is called as Vikara which is abnormal, the stage of equilibrium is Prakruthi which is physiological.

तद् दुःखसंयोगो व्याधय उच्यते। (Ca.Su. 1/23)

The one which brings all the miseries is called as Vyadhi.

रोगस्तु दोषवैषम्यं दोषसाम्यमारोगता (A.Hu.Su. 1/20)

The abnormal fluctuation of dosha beyond the physiological limitation is called as roga, the normal state of dosha is arogya.

दोषदूष्यसम्मूर्च्छना जनितो व्याधि (One Ni.)

The interaction of abnormal dosha with abnormal dushya the resultant is called as Vyadhi.

तथाविध दोषदूष्यसम्मूर्च्छनावस्थाविशेषो ज्वरादि रुपो व्याधि (Ma.N.)

The nidana will produce various types of dushti of doshas. Doshas inturn exploit dushyas. Both dosha & dushya combine together and affect any organ of the body & dostroy its function. As a result various features or symptoms like jwara will manifest. Such entity is called as vyadhi.

विविधं दुःखमादधाति इति व्याधि।

The one which gives different types of pain & suffering is called as vyadhi.

Synonyms of Vyadhi

तत्र व्याधिरामयो गद आतङ्को यक्ष्मा ज्वरो विकारो रोग इत्यर्थान्तरम् (Ca.Ni.1/5)

आमय	: All diseases (Nija) are originated due to Ama.
गद	: Accumulation of toxins/poisons make disease.
आतङ्क	: That which produces fear of death, pain & suffering.
यक्ष्मा	: It may be very complex due to presents or association of too many symptoms.
ज्वर	: The first disease to evolve in mankind.
विकार	: Anything that brings abnormal
रोग	: That which gives pain

रोगः पाप्मा ज्वरो व्याधिर्विकारो दुःखमामयः।
यक्ष्मा आतङ्कगदाबाध शब्दः पर्यायवाचिनः।। (A.huNid.1/1)

पाप्म	: Resultant of पापकर्म or bad deeds done in past or present life.
दुःख	: The one which gives sorrow.
आबाध	: That which gives trouble.

Sushrutha has classified chiefly vyadhi in 3 broad classifications.

1. Adhyatmika : derived from Adi + Atmika that means disease originating from atma gunas (shareera, dosha, dhatu, mala, avayava, manas)

- Adibala pravrutta
- Janmabala pravrutta
- Doshabala pravrutta

2. Adiboutika : derived from bhoutika which means from physical injuries of external cause.

- Shastra krit
- Vyaala krit

3. Adidaivika : derived from those force which is beyond human influence or control.

- Kala bala pravrutta
- Daivabala pravrutta
- Swabhavabala pravrutta

Adibala Pavrutta Vyadhi:

तत्र आदिबलप्रवृत्ता ये शुक्रशोणितदोषान्वयाः।
कुष्टार्शः प्रभृतयः तेऽपि द्विविधाः मातृजाः पितृजाः। (सु.सू. २४/५)

If both shukra and sonitha becomes nasta there is no praja but any part of either shukra or shonitha (beeja bhaaga) is diseased or roga grastha then there will be praja but with some pathological manifestations.

Which particular avayava of beeja bhaaga is abnormal the respective anga developing from that beeja bhaagavayava becomes diseased.

स्त्रीपुंसयोः कुष्ठ दोषाद दुष्टशोणितशुक्रयोः।
यदपत्यं तयोर्जातं ज्ञेयं तदपि कुष्ठितम्। (सु.नि. ५/२८)

Among stree or purusha in their shonita or shukra respectively if there is dushti that can produce kushta then the progeny also gets kushta. Therefore it is of two types.

1. Matruja - inherited from female or mother to progeny
2. Pitruja - inerited from male or father to progeny.

Janmabala Pravrutta Vyadhi:

जन्मबलप्रवृता ये मातुरपचारात् पङ्गु जात्यन्ध बधिर मूक मिन्मिन वामन प्रभृतयो जायन्ते।
तेऽपि द्विविधा रसकृत दौहृदापचारकृताश्च।

This disease will be present in the individual right from birth. It is caused due to garbhakaleena apachara by garbhini stree. i.e. mithyaahara and vihaara that cause garbha vyapath leading to inadequate nourishment and abnormal growth of the foetus giving rise to life long disease and disability. This may bring up with

congenital anomalies. Some of the manifestations in janmabala pravrutta vyadhi are pangu (locomotor dysfunction), badira (deafness), mooka (dumb), minmina (stammering) vaamana (dwarf structure). As the name suggests it is inborn disorder that can be caused due to Teretogens. Teratogens can lead to agenesis, aplasia, hypoplasia to the foetus during intra uterine life.

It is again sub devided into

1. Rasakruta - The foetus gets oxygen and nourishment from Rasa of Matru (mother) through garbha nadi. If the rasa supplied by mother is less or inadequate and contaminated or poor in quality then the growing foetus suffers from growth retatdation.

मातुस्तु खलु रसवहायां नाड्यां गर्भनाभिनाडी प्रतिबद्धा सास्य मातुराहार रसवीर्यमभिवहति।
तेनोपस्नेहनास्यामिवृद्धिर्भवति असङ्घाताङ्गप्रत्यङ्गप्रविभागमानिषेकात् प्रभृति।
सर्वशरीरावयवानुसारिणीनां रसवहानां (तिर्यग्गतानण्) धमनीनामुपस्नेहो जीवयति। (सु.शा.३/३९)

2. Douhrudapacharaja - The condition during pregnancy where there are two functioning hearts (that is - one functioning heart of mother and one functioning heart of foetus) is called as douhrudavastha. During this stage various longings are expressed from mother. These longings represent the likes of foetus or nutritional need and demand from the body. Such longings are to be fulfilled by mother. If not it causes douhrudapachara or douhrudavamana leading to foetal distress and disease.

Dosabala Pravrutha Vyadhi

दोषबलप्रवृत्ता ये आतंकसमुत्पन्ना । मित्थ्याहाराचारकृताश्च तेऽपि द्विविधा।।
आमाशयसमुत्थाः पक्वाशयसमुत्थाश्च । पुनश्च द्विविधाः सारीरमानसाश्च।। (सु.सू. २४/५)

This is a variety of disease due to doshavaishamya caused by consumption of mithya ahara and vihara by an indivisual. This is acquired disease which is not based on heredity of regiemen of mother during pregnancy. It is again subdivided into

1. Amashaya samutta : Disease originating from amashaya (includes respiratiory, cardiovascular and upper GIT structures) eg; kasa, chardi, hridroga
2. Pakwashaya samutta : disease originating from pakwashaya (includes lower GIT, excretory, reproductive and nervous system.) eg; atisara, mutrakruchra, pakshagata.

Again Subdivided into

1. Shareerika - Somatic or physical disorders

2. Manasika- psychological disorder (eg unmade, apasmara, vishada)

All these are adhyatmika vikaras

Adiboutika

Sanghatabala Pravrutta Vikara

भूतेष्वधिकृत्य यत्प्रवर्तते तद् आदिभौतिकम्

Human being, animal, bird or such physical agents producing dhukka is adiboutika. The external factors causing injury to the body. It is also called as sanghatabala pravrutta vyaadhi i.e. due to injury. The injury may be due to agni, shastra from any prahara or attack and accident. It is again devided into shastrakrut and vyala krut.

आगन्तुरोगाः ये भूतविषवायु अग्निसम्प्रहारादिसम्भवाः नृणामागन्तवो रोगाः।

- Shastra krit - injury due to sharp instruments.
- Vyaala krit - injury due to bite of animals.

Adidaivika

This is becauses of those forces which are beyond human access, influence or control. The disease caused due to bad deeds done in present or past life are giving its effect back in the form of disease or suffering. It also due to effect of curse or wrath of Gods, elders, teachers or divine people. Adidaivika vikaras can also be produces as result of invasion of evil spirits.

निर्दिष्टं दैवशब्देन कर्मयत् पौर्वदैहिकम्।
हेतुस्तदपि कालेन रोगाणामुपलभ्यते। (च.शा. १/११६)
दैव प्राक्तनं कर्माधिकृत्य वर्तते इत्याधिदैवं तत्र भूतं इत्याधिदैविकमङ्ग। (डल्हण)
दैवबलप्रवृत्ता ये देवद्रोहादभिश्प्तका अथर्वणकृता उपसर्गजाश्च। (सु.सू. २४/७)
त्रयो मलाः भूताभिषङ्गात् कुप्यन्ति।

This can be classified into :

1. Vidyudadashanikruta- hit from thunder blot, electric injury
2. Pishachadi kruta- Invasion of evil spirits inside the body.

तेऽपि द्विविधा विद्युदशनिकृताः पिशाचादिकृताश्च। पुनश्च द्विविधा संसर्गजा आकस्मिकाश्च।

Again it is of two varieties :

1. Samsargaja- communicable disease from person to person transmitting from sexual contact, direct transmission, physical contact, inhalation, sharing utensils, cloths, jewels and cosmatics.

प्रसङ्गात् गात्रसंस्पर्शात् श्वासात्सहभोजनात्। सहशय्यांऽऽसनच्चैव वस्त्रमाल्यानुलेपनात्।
कुष्ठं जरश्च शोषश्च नेत्राभिष्यन्दव् एव च। औपसर्गिकरोगाश्च सङ्क्रामन्ति नरन्नरम्।।(सु.नि.५/३३-३४)

2. Aaakasmika - All of sudden, accidental, unexpected and unexplainable

आकस्मिकाः संसर्गजव्यतिरिक्त आधिदैविका। अपरिदृश्यमानहेतुतया आकस्मिका भज्यन्ते। (सु.सू. २४/७)

Kaala bala pravrutta

The disease caused due to climatic variations, weather impact, natural calamities due to excessive or inadequate seasonal manifestations.

कालर्थकर्मणां योगो हीनमिथ्यातिमात्रकः। सम्यग्योगश्च विज्ञेयो रोगारोग्यैककारणम्।।

It is due to hinayoga (inadequate manifestation of seasonal feature eg: less or no rainfall during rainy season), mithya yoga (abnormal seasonal qualities eg- rainfall in winter season), atiyoga (excessive seasonal manifestation eg ; too heavy rain fall in rainy season or too intence and sharp dry sun in summer).

कालबलप्रवृत्ता ये शीतोष्णवातवर्षातपप्रभृतिनिमित्ताः। (सु.सू. २४/७)
शीतोष्णवर्षलक्षणाः पुनर्हेमन्तग्रीष्मवर्षाः संवत्सराः। (च.सू. ११/४२)

This undue variation of sheeta, ushna climate during summer, rainy, winter season, movement of wind and intensity of sun gives kala bala pravrutta roga. It can cause Janapadodwamsa.

Swabavabala pravrutta

This is a variety of disease caused due to very human nature. There are few natural qualities which tend to cause disease without much outer influence. These disease are un avoidable.

स्वभावबलप्रवृत्ताः क्षुत्पिपासाजरामृत्युनिद्राप्रभृतयः। (सु.सू. २४/८)
तेऽपिद्विविधाःकाल्कृता अकालकृताश्च।

Those natural qualities or components are-

1. Kshut - hunger (if this kshut vega is not answeres or kshut is unmanifested it causes disease). It is due to kshut vega person eats food and hence gets nourished. If njo hunger then poor nourishment deplets the body.
2. Pipasa - thirst (to hydrate the body and maintain water balance and homeostasis). Absence of pipasa may give dehydration and death.

3. Jara - Oldage or aging process. Degeneration of tissues, organs, damage to vital physiology takes the patient to death.
4. Mruthyu - Death

तत्र परिरक्षणकृताः कालकृताः अपरिरक्षणकृताः अकालकृताः। (सु.सू. २४/८)

it is of two types again 1. Kalaja- according to time
2. Akalaja- untimely or prematurely (eg premature againg, death at young age before completion of life span).

क्षुत्- कार्श्यदौर्बल्यवैवर्ण्यमङ्गमर्दोऽरुचिभ्रम।
पिपास- कण्ठस्यशोषोबादियश्रमः सादो हदि व्यथा।
निद्रा- जृम्भाऽङ्गमर्दस्तन्द्रा च शिरोरोगाऽक्षिगौरवम् निद्राविधाअणात्तत्र..... (च.सू. ७)

Santarpanajanya Vikara

The diseases caused due to over nourishment comes under ati santarpanajanya vikaras. Body needs energy for functioning or working. The energy is provided by carbohydrates, protein mainly through dietary supplement. Adequate food supply and apt breakdown of calories keeps the body sound. But when food supply becomes too much and no breakdown of ingested carbohydrates and proteins takes place then storage in the formof fat is the ultimate end. Fat storage continues and gives rise to various ailments called as santarpanotta vikaras.

Nidana of Santarpana janya vikaras:

संतर्पयति यः स्निग्धैर्मधुरैर्गुरुपिच्छिलैः।
नवान्नैर्नवमद्यैश्च मांसैश्चानूपवारिजैः।।
गौरसैर्गौडिकैश्चानैः पैष्टिकैश्चातिमात्रशः।
चेष्टाद्वेषी दिवस्वप्नशय्यासनसुखे रतः।। (च.सु. २३/३,४)

Consumption of excessive oily, sweet, heavy, slimy foods. Freshly harvested rice, freach alcohol beverages, meat of marshy and aquatic in origin. Milk and milk products, Sugarcane products, baked foods. All these have high carbohudrates, proteins and fat in excess. They are high calorie food stuffs. If these calories are not burnt by metabolism and physical stress then they are stored as fats.

The sedentary life will not allow burning of calories. Less physical movements, day sleeping, always sitting or sleeping leads accumulation of fat. This will result in atisantarpana. The diseases causes due to atisantarpana are as follows:

रोगस्तस्योपजायन्ते सन्तर्पणनिमित्तजाः। प्रमेहपिडकाकोटकण्डूपाण्डूवामयज्वराः।।
कुष्ठान्यामप्रदोषाश्च मूत्रकृच्छ्रमरोचकः। तन्द्रा क्लैब्यमतिस्थौल्यमालस्यं गुरुगात्रत।।
इन्द्रियस्रोतसाम् लेपः बुद्देर्मोहः प्रमीलकः। शोफश्चैवंविधाश्चान्ये शीघ्रमप्रतिकुर्वत।।(च.सु.२३/२६)

Prameha and prameha pidakas, various kinds of skin lesions, and rashes, pandu, jwara, kushta and other ailments of ama. Diseases of urinary system, loss of taste, lazyness, infertility, obesity, lethargy, heaviness of body. Unclear indriyas due to accumulation of mala in their patway, diminished alertness and diminished higher mental functions. Sluggish circulation and altered cutaneous sensorium. Oedema of various types.

Apatarpanotta Vikara

Disease caused due to under nourishment or poor nourishment. It can be caused due to inadequate dietary suppliments, chronic disease, usage of strong medecines.

वक्ष्यन्ते सौषधाश्चोर्ध्वमपतर्पणजा गदा। देहाग्निबलवर्णौजःशुक्रमांसपरिक्षयः।।
ज्वर कासानिबन्धश्च पार्श्वशूलमरोचक। श्रोत्रदौर्बल्यमुन्मादः प्रलपो हृदय व्यथाः।।
विन्मूत्रसंगः शूलं जंघोरुत्रिकसम्श्रयम्। पर्वास्थिसन्धिभेदश्च ये चान्ये वातजा गदाः।।
उर्ध्ववातादयः सर्वे जायन्ते ते अपतर्पणात्।। (च.सु. २३/२८-२९)

Any thing that cause kshaya of body, agni comes under apatarpana. Decreasing of bala due to dhatu kshaya and ojokshaya. The patient becomes lean and debilitated. Due to poor bala the immunity may decrease. Frequently patient becomes prone for jwara, kasa, parshwashoola, arocaka. Dourbalya of all the indriyas due to dhatu kshaya. This decreases satwabala of patient and cause unmada, pralapa and hrudaya vyatha. Less or dry stools and poor excretion. Pain in low back, thighs,pelvic region. Degeneration of asti causes pain in parva, sandhi and all bones. Patient is prone for any vata vikaras as Vata dosha dominates in क्षय (kshaya).

Samanya and Nanatmaja Vikara

The disease produced by dushti of one single dosha only. That dushta dosha will not combine with other dosha to produce disease or symptom. Such diseases are named as Nanatmaja roga.

It is derive from

Na + anatmaja

Atmaja gunas are Vata, pitta, kapha, manasika doshas. Anatmaja means devoid of atmaja. Therefore with combination of Na it again means absence of Anatmaja, which again is inferred as

Na+Anatmaja = Atmaja. From this equation Nanatmaja is evolved. Thus nanatmaja means kevala eka doshaja, originated from one singu dosha.

There are 80 vataja nanatmaja vikaras, 40 pittaja nanatmaja vikaras, 20 kaphaja nanatmaja vikaras.

तत्र सामान्यजा ये पृथक् समस्तैर्वा दोषैः क्रियन्ते, नानात्मजा विशेषजा ये नियतेनैव दोषेण क्रियते। (रत्नप्रभा टीक)
सामान्यजा इति वातादिभिः प्रत्यैकम् मिलितैश्च ये जन्यते।
नानात्मजा इति ये वातादिभिर्दोषान्तरासंपृक्तैर्जन्यन्ते।। (चक्रपाणि च.सु. २०/११)

On the contrary there are diseases called Samanya vikaras which is produced by combination, mutual association of two or three doshas by permutation and combination (samsarga-dwidoshaja, sannipataja - tridoshaja). Thus one can say that samanya vikaras are due to association of two or three doshas together, but nanatmaja vikaras are produced by single and independent dosha (eka doshaja).

2. Criteria for nomenclature of Diseases in Ayurveda (Vyadhinamakarana)

The disease are known through their clinical features. But there are multiple clinical features in a disease. Therefore there is a need of naming a disease to identify. Vyadhi Namakaran Siddhanta means naming the vyadhi on certain criteria.

- त एवापरिसंख्येया भिद्यमाना भवन्ति हि। रुजावर्णसमुत्थानस्थानसंस्थाननामभिः।।
(च.सू. १८/४२)

The disease is named with certain paremeters or criteria E.g. Based on Prakriti, adhistana or samsthana, laxana, samuthana or hetu, varna, ruja, adrishya, aakriti etc.

- विकाराः पुनप्रिसंख्येयाः, प्रकृत्यधिष्ठानलिङ्गयतनविकल्प विशेषापरि-संख्येयत्वात्।।(च.सू.२०/३)

Based on Prakriti

It is the nearest cause for the Vyadhi (sannikrishta karana).

- On the basis of vyadhi prakriti - 1. Vataja vyadhi

Ex.
- Vata rakta,
- Grudrasi
- Vata kantaka,
- Apabahuka,
- Pakshagata,
- Ardita etc.

On the basis of vyadhi prakriti - 2.Pittaja vyadhi

Ex. • Pittaja jwara, • Pittaja grahani,
• Pittaja arsha, Pittaja apasmara etc
• On the basis of vyadhi prakriti - kapha.

Ex. • Dhamani pratichaya, • Sthoulya,
• Granthi, • Kaphaja unmada,
• Kaphaja arsha etc. Based on Adhistana

2. The Naming can be done According to Sthana or Site of Manifestation

- Grahani
- Charmakila
- Manyastamba
- Hanugraha
- Hridroga
- Shira shoola
- Urusthambha

3. Based on Lakshana/linga or Clinical Features

Based on prathyatmaka lakshana naming can be done

- Athisara----गुदेन बहुद्रवसरणमतिसार

It means the sarana which take place in the adhik pramana. The characterized features of athisara.

- Chardi--- छादयति मुखम् अर्दयति चाङगनीति छर्दि:

Chardi that which comes out through mouth and which causes discomfort to sharira.

- Kasa---- "कासनं कास इति वा, भिन्नस्वरः कासति शुष्कमेव"

One which makes the sound of the bhina kansya is called kasa, because of its featurers it is called kasa.

- Hikka--- "हिगिति कृत्वा कायति शब्दायते इति हिक्का"

One which makes the sound like hikka.

- Alasaka---- आमाशयेऽलसीभूतस्तेन् सोऽलसकः स्मृतः

Stasis in amashaya which neither moves upwards or downwards

- Mutrakruchra----मूत्रस्य कृच्छेण महता दुःखेन प्रवृतिःमूत्रकृच्छम।

Passing of urine with great difficulty.

4. Based on Samuttana or Cause of the Particular Disease

A very particular causes leads to that particular disease only.

Ex.

- Krimija hrid roga is chiefly causes due to krimi.

- Saahas janya rajayakshma is caused due to excessive physical exertion or sahasa.
- Madhyaj trishna caused due to excessive and improper intake of madhya.
- Mritika janya pandu caused typically due to mrudbhakshana.

5. Based on Varna

Namakarana of vyadhi on the basis of varna that is color imparted on any of body parts like twak, nakha, nayana, mutra or pureesha.

Ex. • Haridra Meha • Manjistha meha

- Pandu----पाण्डुना वक्ष्यमाणहरितादिवर्णेभ्यः प्रधानेन वर्णेनोपलक्षितो रोगः पण्डुरोगः

Pandu roga indicates varna pradhana roga like harithadi varna

6. Based on Ruja or Pain

Variety of disease depending on severity, intensity, nature of pain.

- Arsha----अरिवत् प्राणान् शृणाति हिनस्तीत्यर्थः

That which tortures body and mind just like enemies

- Sula----शङकुस्फोटनवत्तस्य यस्मात्तीव्रा हि वेदना।

Pain experienced as if being pierced with of a cone or exploding nature.

- Visuchika----सूचीभिरैव गात्राणि भिनत्तीति विसूचिका

Pricking type of pain experienced all over body

7. Based on Sadrusha or Simulation

Naming the vyadhi on the basis of resembalance of structure with any other material.

- Manjistha Meha- color of urine resembles to the color of Manjishta.
- Kroshtuka shirsha---क्रोष्टुकशीर्षवत शृगालमस्तकवत् स्थूलः

The part of affected limb resembles similar to head of the fox.

- Gridhrsi---गृधवत चलति इति गृध्रसी

The gait of the affected person resembles to the gait of the vulture.

8. Based on Akruti or Shape

- Namakarana of vyadhi on the basis of aakriti.

Ex.
- Masurika--- मसूरिकाकृति संताना: पिडका:स्युः मसूरिका:
  Shape of the pidakas will be like masura dala
- Dhanu sthambha---धनुस्तुल्यं नमेद्यस्तु स धनुःस्तम्भसंज्ञकः
  Body will bend like a bow in dhanu sthambha

3. Bija, Bija Bhaga and Bija Bhaga Avayava Dushti

यस्य यस्य अङ्गावयवास्य बीजे बीजभग उपतप्तो भवति।
तस्य तस्याङ्गवयवस्य विकृतिरुपजायते नोपजायते चतुपातापीतात्। (च.शा. ३/१८)

Which ever part in beja or beejabhaga gets abnormal, the structures originated from that abnormal beeja or beeja bhaga or beeja bhaga arayava will be in dusta valtha. If both shukra & shonita becomes nasta there is no praja.

- If both shukra and sonitha becomes nasta there is no praja but any part of either shukra or shonitha (beeja bhaaga) is diseased or roga grastha then there will be praja but with some pathological manifestations.
- If particular avayava of beeja bhaaga is abnormal the respective anga developing from that beeja bhaagavayava becomes diseased.

Interpretation

Hereditary disease or disease caused due to abnormal chromosomal structures or numbers may come under this group. Abnormalities of structure of chromosomes like deletion, translocation, inversion, ring formation.

It is an incurable but preventable disease

जातप्रमेही मधुमेहिनो वा । न साध्यः उक्तः स हि बीजदोषात्।
ये चऽपि केचित् कुलजा विकारा भवन्ति ताश्च प्रवदन्त्यसाध्यात्। (Cha. chi. 6/57)

Disease of beeja dushti is never curable. In jatapramehi there is beeja dushti. Such diseases run within the family.

4. Basic knowledge of Hereditary, Congenital, Acquired, Multifactorial, Traumatic and Environmental disorders

The word hereditary refers to transmission of genetic characters from parents to offsprings. Cells or organism acquire the characteristics of its parent cell or organism. It also transmits traits from parents and their ancestors. Many elements can associate transmission example-physical characters, mental attributes, behavioral patterns and also illness. It depends upon the segregation and recombination of genes during meiosis and fertilization which results in genesis of new individual similar to others of its kind but exhibiting certain varieties resulting from particular mix of genes and its interaction with the environment.

Genes are made from long molecules called DNA which is copied and inherited across generations. DNA is made up of simple units that line up in particular order of letters on page carries information. The language used by DNA is called genetic code which allows the genetic machinery to read the information in the genes in triplet sets of codons. This information is the instructions for constructing and operating a living organism.

This gene may possess error. Thus genetic disease is caused by single error in a single gene in our DNA. The effects of the disease depend upon what that gene was supposed to do. Everyone has two copies of each gene. One copy on each of the chromosome pair and some disease require both copies to be damaged(recessive gene) and some need only one gene copy damaged (dominant gene). In recessive disease genetic error usually needs to be inherited from both parents to get the disease and in dominant disease error need only be inherited from one parent in order to get the disease. The word carrier refers to an error in genes is present but the individual does not possess any symptoms.

Classification

1. X-linked disease : These are due to errors in genes in the X chromosomes.
2. Autosomal disaease : Caused by errors in genes on the non sex chromosomes (that is other 22 chromosomes)

4. Major Classes of Genetic Disease with Different Inherent Patterns.

1. Autosomal recessive genetic disease: disease occurs when both copies of a gene on chromosome 1-22 is inherited from both parents eg: cystic fibrosis, phenyl ketonuria, sickle cell anaemia, albinism, taysachs galactosemia.
2. Autosomal dominant genetic disease: disease occurs when a single damaged copy of a gene on chromosome 1-22 inherited from either parent. The bad copy dominate the other good copy eg- huntongton's disease, achondroplasia (dwarfism).
3. X-linked recessive genetic disease : one gene error on X chromosomes cause disease n men(who have only one copy of X) eg- hemophilia, duchenne muscular atropy.
4. X-linked dominant genetic disease: A single gene error on the X chromosome cause disease in both men and women. Men have only 1 copy of X and in woman are XX. The bad copy dominates the good copy. X linked dominant are much rarer than X linked recessive gene.

Conclusion

Sometimes even an autosomal dominant disease can arise surprisely when neither parent has the disease. This can be due to mutation or germinal mosaicism.

All types of genetic disease occur at birth. It will be inherited from parents through DNA, cannot be caught later on in life. Genetic tests can determine whether or not a person has the disease, even as early as in fetus by antenatal testing for genetic disease.

Sickle cell anaemia, Down's syndrome, color blindness such 4,100 genetic diseases are identified. This has no treatment. Some are less burdensome. It is always associated with medical maladies and short life span.

Congenital Diseases

It is a condition tat exists right from birth and often before birth. The defect may develop during first month of life (neonatal disease). Of these disease those charecterised by structural deformities are called as congenital anomalies and involve defects in or damage to developing fetus. A congenital disorder may be the result of genetic anomalies, uterine environment, errors of morpogenesis, infection or inherited metabolic disorder and rare disease a chromosomal abnormality. The outcome of disease may depend upon complex interaction between prenatal deficit and post natal environment. Eg; mucopoly saccharoidosis, down syndrome, phenyl ketonutria, osteogenesis imperfecta, hyperammonia.

Multifactorial Disease

They are caused not by a single gene mutation, but by a combination of genetic and environmental factor working together in wage. They involve variations in multiple genes often coupled with environmental causes. Eg; Alzheimers disease, obesity, hypertension, Diabetes mellitus, infertility, ischemic heart disease, irritable bowel syndrome.

Acquired Disorder

These are the disease incurred as result of factors acting from or originating outside the organism, not inherited. That develops after birth or develops in response to an antigen. Pertaining to characteristic, condition or disease originating after birth not caused by hereditary or developmental factors but caused by reaction due to environmental influences like food, air, human resources etc outside the organism.

Traumatic disease

These are the disease caused due to external factors that cause injury or trauma to the organism. Trauma can be due to physical agents like attack by sharp or blunt instrument, road traffic accidents, thermal or electric injury, chemical injury. A serious injury or shock to the body, as from violence or an accident. It may also cause post traumatic stress or neurosis. The range of disease may vary from disease to deformity or disability.

Environmental Disease

Environmental pathology encompass all such disease caused by progressive deterioration in the environment, most of which are man made. The causes may be population explosion, urbanization, deforestation, accumulation of waste and unsatisfactory disposal of radioactive waste, industrial effluents, automobile exhausts domestic wastes. Among them most affecting environmental cause to human health are use of tobacco, alcohol consumption and intoxicant drugs.

1. Environmental pollution : Air pollution due to various gases like carbon monoxide, sulphur dioxide, carbon dioxide, nitrous

oxide, coal and so on produce asbestose lung, silicosis, pneumoconiosis, cancer of lung. This depends upon duration of exposure, dose of inhalation, and particle size (1-5mm) gases like carbon monoxide are produced by incomplete combustion of carbon. Its sources are automobile exaust, burning fossil, fuel industry, home and tobacco smoke. It causes accidental death due to systemic oxygen deprivation of tissues, it mixes with hemoglobin producing carboxyhemoglobin which leads to hypoxia, oedema and petechial hemorrhage.

2. Tobacco consumption: This causes coronary heart disease, cancer of oral cavity, aero digestive system, and lung. It also causes COPD, Peptic ulcer, vascular disease, cancer of pancreas, urinary bladder and kidney. In pregnant this leads to low birth weight of fetus, perinatal mortality and intellectual deterioration of new born in non pregnant woman it causes premenopausal and post menopausal syndrome.
3. Alcoholism: chronic alcoholism is defined as regular imbibing of an amount of ethyl alcohol that is sufficient to harm an individual physically, psychologically and socially. Acute alcoholism has impact on following organs.
   a. CNS: depression, disordered cortical function, motor ataxia, behavioral changes. These changes occur when blood alcohol levels not more than 100mg/dl. If it raches 100-200mg/dl the impacts are depression of cortical center, lack of coordination, impaired judgment, and drowsiness. Blood alcohol level at 300mg/dl causes stupor and coma. Blood alcohol at 400mg/dl can cause anesthesia, depression of medullary center and death due to respiratory arrest.
   b. Stomach : Vomiting, acute gastritis and peptic ulceration
   c. Liver : cirrhosis, fatty liver.
   d. Pancreas: chronic calcifying pancreatitis, acute pancreatitis.
   e. GIT : Gastriris, Peptic ulceration, esophageal varesis, massive GI bleeding.
   f. CVS : Cardiomegaly, beer drinkers myocardiosis, wit dialated cardiomyopathy.
   g. Endocrine system: testicular atropy, feminization, loss of libido and potency, gyenecomastia and decreased testicular levels.
   h. Hemopoietic system : Megaloblastic anemia, decreased efficiency of immune system.
4. Drug abuse: narcotics, sedatives, tranquilizers or barbiturates, psychedelics (enjoyable - perception giving) inhalants are few variants of drugs which are addictive. They cause effective adverse action on psychosomatic system of abuser. It causes focal glomerulonephritis, talc granuloma formation in lung. They are CNS depressants in long run causing significant degenerative changes in brain and nervous system. Dependence or withdrawal causes agitation, restlessness, tremors, insomnia and behavioral changes. Overdose will cause irreversible damage and also may turn fatal.
5. Environmental chemicals: Insecticides, pesticides, fertilizers of various forms are used for agricultural purpose and lots of synthetic food colors, flavors and preservatives are used in food industry. The accumulation of these chemicals in body can cause severe damage to consumer. Organo phosphorous compounds, voletile organic solvents, metals, aromatic hydrocarbons, cyanide, environmental dust all these can harm any system of our body to severe extend. Skin ailments, respiratory distress, premature aging, musculo skeletal weakness, sensory defecits, birth defects, mental retardation, physical deformity are various presentation of usage of such elements.
6. Thermal and electrical injury : Temperature below 35 degree centigrade is considered as hypothermia and above 41 degree Celsius as hyperthermia. Hazards due to hypothermia are focal injury as in frost bite, chill blaints, claudication, systemic injury and death. Hyper thermia can manifest in the form of thermal burns, heat exhaustion, heat stroke. Severe sweating, cramps, shock are some of its presentations.
7. Radiation injury : radiation injury can happen due to exposure to radiation by ultra violet rays, X rays, electromagnetic and infrared radiation. Among them hazards by X-ray and ultra violet rays are more significant. Acute cell killing, malignant transformation of cell, genetic damage by mutation which passes genetic defects to next generation or next progeny of

cell. Ionizing radiation is widely used in diagnostic purpose as well as for radiotherapy of malignant tumors. Radiation induced cell death is mediated by radiolysis of water in the cell with generation of toxic hydroxyl radicals. During radiotherapy, some normal cells coming in the field of radiations are also damaged. In general radiation induced tissue injury predominantly affects endothelial cells of small arteries and arterioles causing necrosis and ischemia. Some of its hazards are seen as follows.

a. Skin : Radiation dermatitis, cutaneous carcinoma,
b. Lungs : interstitial pulmonary fibrosis
c. Heart : myocardial fibrosis, constrictive pericarditis
d. Kidney : Radiation nephritis
e. GIT: Strictures of small bowel and esophagus
f. Gonads : testicular atropy in males and destruction of ovaries in females.
g. Hemopoietic system : Bone marrow depression
h. Eye : cataract

Ultra violet rays : acute skin injury as sunburns, cutaneous cancer, solar keratosis. Non iodizing radiations like electromagnetic radiation produced by microwave ovens, radio, diathermy, ultra sound do not produce tissue damage.

5. Introduction to ICD Classification of Diseases of WHO and DSM classification

"A classification of diseases can be defined as a system of categories to which morbid entities are assigned according to established criteria. The purpose of the ICD is to permit the systematic recording, analysis, interpretation and comparison of mortality and morbidity data collected in different countries or areas and at different times. The ICD is used to translate diagnoses of diseases and other health problems from words into an alphanumeric code, which permits easy storage, retrieval and analysis of the data".

ICD-10 represents International Statistical Classification of Diseases and Related Health Problems.

It can, therefore, be used to classify data recorded under headings such as "diagnosis", "reason for admission", "conditions treated" and "reason for consultation", which appear on a wide variety of health records from which statistics and other health-situation information are derived.

Complexity and Importance

- International Classification of Disease has multi-dimensional purpose and usage. This has made the classification most complex.
- It has become the international standard diagnostic classification for all general epidemiological and many health management purposes.
- ICD helps in analysis of the general health situation of population groups and the monitoring of the incidence and prevalence of diseases and other health problems recorded on many types of health and vital records.

Development of ICD - 10

- Work on the Tenth Revision of the ICD started in September 1983 when a Preparatory Meeting on ICD-10 was convened in Geneva. The programme of work was guided by regular meetings of Heads of WHO Collaborating Centres for Classification of Diseases. Policy guidance was provided by a number of special meetings including those of the Expert Committee on the International Classification of Diseases
- Meeting for Tenth Revision was held in 1984 and 1987.

Arrangement of Volumes of ICD-10

- Volume 1: Main classifications
- Volume 2: Instruction/Guidance to users
- Volume 3: Alphabetical Index
- ICD-10 has 21 chapters against 17 Chapters in ICD-9

Chapters of ICD-10

- Chapters I to XVII : Diseases and other morbid conditions

- Chapter XVIII: Symptoms, signs and abnormal clinical and laboratory findings, not elsewhere classified.
- Chapter XIX: Injuries, poisoning and certain other external causes.
- Chapter XX: External causes of morbidity and mortality
- Chapter XXI: Factors influencing health status and contact with health services.

Structure and Principles of ICD

- Originally conceived by William Farr
- The Classification is grouped as below:
- Epidemic diseases
- Constitutional or general diseases
- Local diseases arranged by site
- Developmental diseases
- Injuries.

The Arrangement of Chapters

1. Certain infectious and parasitic diseases
2. Neoplasms
3. Diseases of the blood and blood-forming organs and certain disorders involving the immune mechanism
4. Endocrine, nutritional and metabolic diseases
5. Mental and behavioral disorders
6. Diseases of the nervous system
7. Diseases of the eye and adnexa
8. Diseases of the ear and mastoid process
9. Diseases of the circulatory system
10. Diseases of the respiratory system
11. Diseases of the digestive system
12. Diseases of the skin and subcutaneous tissue
13. Diseases of the musculoskeletal system and connective tissue
14. Diseases of the genitourinary system
15. Pregnancy, childbirth and the puerperium

16. Certain conditions originating in the perinatal period
17. Congenital malformations, deformations and chromosomal abnormalities
18. Symptoms, signs and abnormal clinical and laboratory findings, not elsewhere classified
19. Injury, poisoning and certain other consequences of external causes
20. External causes of morbidity and mortality
21. Factors influencing health status and contact with health services
22. Codes for special purposes

WHO: Help-Line

There are nine WHO Collaborating Centres for Classification of Diseases, who assist countries with problems encountered in the development and use of health-related classifications and, in particular, in the use of the ICD.

Ref: Australia, England and USA for English knowing countries. Besides, there are at France, Russia, China, Venezuela Sweden, Brazil

Problem Encountered

- Size of the Classification
- No formal training
- No user-friendly software to guide
- No abridged Classification of Diseases based on Indian condition & requirement
- No Online Help-line system

Diagnostic and Statistical Manual of Mental Disorders

DSM-V Diagnostic and Statistical Manual of Mental Disorders is the planned fifth edition of the American Psychiatric Association's (APA)

In 1999, a DSM-5 Research Planning Conference; sponsored jointly by APA and the National Institute of Mental Health (NIMH), was held to set the research priorities. There were six workgroups, each focusing on a broad topic:

- Nomenclature,

- Neuroscience and Genetics,
- Developmental Issues and Diagnosis,
- Personality and Relational Disorders,
- Mental Disorders and Disability, and
- Cross-Cultural Issues.

First Draft Diagnostic Criteria of Psychotic Disorders

1. The recommendation of new categories for learning disorders and a single diagnostic category, "autism spectrum disorders" that will incorporate the current diagnoses of autistic disorders, Asperger syndrome, childhood disintegrative disorder and pervasive developmental disorder (not otherwise specified). Work group members have also recommended that the diagnostic term "mental retardation" be changed to "intellectual disability," bringing the DSM criteria into alignment with terminology used by other disciplines.
2. Eliminating the current categories substance abuse and dependence, replacing them with the new category "addiction and related disorders." This will include substance use disorders, with each drug identified in its own category. Eliminating the category of dependence will better differentiate between the compulsive drug-seeking behavior of addiction and normal responses of tolerance and withdrawal that some patients experience when using prescribed medications that affect the central nervous system.
3. Creating a new category of "behavioral addictions," in which gambling will be the sole disorder. Internet addiction was considered for this category, but work group members decided there was insufficient research data to do so, so they recommended it be included in the manual's appendix instead, with a goal of encouraging additional study.
4. New suicide scales for adults and adolescents to help clinicians identify those individuals most at risk, with a goal of enhancing interventions across a broad range of mental disorders; the scales include research-based criteria such as impulsive behavior and heavy drinking in teens.
5. Consideration of a new "risk syndromes" category, with information to help clinicians identify earlier stages of some serious mental disorders, such as neurocognitive disorder (dementia) and psychosis.

6. A proposed new diagnostic category, temper dysregulation with dysphoria (TDD), within the Mood Disorders section of the manual. The new criteria are based on a decade of research on severe mood dysregulation, and may help clinicians better differentiate children with these symptoms from those with bipolar disorder or oppositional defiant disorder.
7. New recognition of binge eating disorder and improved criteria for anorexia nervosa and bulimia nervosa, as well as recommended changes in the definitions of some eating disorders now described as beginning in infancy and childhood to emphasize that they may also develop in older individuals.

Dimensional Assessments

In addition to proposed changes to specific diagnostic criteria, the APA is proposing that "dimensional assessments" be added to diagnostic evaluations of mental disorders. These would permit clinicians to evaluate the severity of symptoms, as well as take into account "crosscutting" symptoms.

Careful Consideration of Gender, Race and Ethnicity

The process for developing the proposed diagnostic criteria for DSM-5 has included careful consideration of how gender, race and ethnicity may affect the diagnosis of mental illness.

Proposed changes to DSM-IV diagnoses

Asperger Syndrome

Asperger syndrome will be eliminated as a separate disorder, and merged under autism spectrum disorders (ASD). Under the new classification, clinicians would rate the severity of clinical presentation of ASD as severe, moderate or mild.

Attention Deficit Hyperactivity Disorder

There has been a proposal to increase the diagnostic criteria for the age when symptoms became present. The proposal would change the diagnostic criteria from symptoms being present before seven years of age to symptoms being present before twelve years of age. The new diagnostic criteria would be Several noticeable inattentive or hyperactive-impulsive symptoms were present by age 12

Bipolar Disorder

There have been proposals to include further and more accurate sub-typing for bipolar disorder. There have been proposals for more

stringent criteria for the diagnosis of bipolar disorder in children with a new diagnosis temper dysregulation disorder with dysphoria proposed.

Depression

While currently grief is only considered a sign of depression if two months have elapsed since the death of a loved one, the new version would allow for diagnosis within the first few weeks.

Dissociative Identity Disorder

Proposed changes to the controversial dissociative identity disorder diagnosis include adding a new diagnostic criterion: "C. Causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Gender Identity Disorder

Gender Identity Disorder (GID) will be renamed "Gender Dysphoria" in the DSM 5. Along with these changes comes the creation of a separate Gender Dysphoria in Children as well as one for Adults and Adolescents.

Hypersexual Disorder

Hypersexual Disorder is proposed as a new category to be added. The diagnosis would apply when a person experiences several of the indicated symptoms (extreme amounts of time spent in the sexual activity, using the sexual activity in response to low mood or stress, failed attempts to reduce the behaviors, etc. Moreover, it would apply only when the problem lasted six months or more, when person experienced significant distress or impairment in major life areas because of it, and when the problem was not directly caused by a medication or drugs, as well as other criteria. The label "hypersexual disorder" was reportedly chosen because it did not imply any specific theory for what causes hypersexuality, which remains unknown.

Oppositional Defiant Disorder

It is proposed that the eight symptoms of Oppositional Defiant Disorder should be divided into the following categories: Angry/ Irritable Mood; Defiant/Headstrong Behavior; and Vindictiveness. However, just as in the DSM-IV-TR, four of these symptoms need to be present to meet diagnostic criteria. The minimum four symptoms can come from all (or even just one or two) of the three

categories. It is proposed that a section be added to the diagnostic criteria for Oppositional Defiant Disorder stating that for children under 5 years of age, oppositional behavior "must occur on most days for a period of at least six months". For children 5 years or older, oppositional behavior "must occur at least once per week for at least six months".

Personality Disorders

Major changes have been proposed in the assessment and diagnosis of personality disorders. These include a revamped definition of personality disorder and a dimensional rather than a categorical approach based on the severity of dysfunctional personality trait domains (negative emotionality, introversion, antagonism, disinhibition, compulsivity, and schizotypy). In addition, patients would be assessed on how much they match each of six prototypic personality disorder.

Pica

It is proposed that Pica is reclassified from the "Disorders Usually First Diagnosed in Infancy, Childhood, or Adolescence" classification to the "Eating Disorders" classification. It is proposed that the wording of "non-food substances" be added alongside the current DSM-IV-TR wording of "non-nutritive substances". "Non-food" was added to further clarify that items consumed are not just merely lacking nutrients

Post Traumatic Stress Disorder

Various criteria changes are proposed.

Schizophrenia

The following Schizophrenia subtypes are proposed for removal from DSM-5. :

- 295.30 Schizophrenia - Paranoid Type
- 295.10 Schizophrenia - Disorganized Type
- 295.20 Schizophrenia - Catatonic Type
- 295.90 Schizophrenia - Undifferentiated Type
- 295.60 Schizophrenia - Residual Type

Somatoform Disorder

Additional proposed somatoform disorders are:

- Abridged somatization disorder - at least 4 unexplained somatic complaints in men and 6 in women.

- Multisomatoform disorder - at least 3 unexplained somatic complaints from the PRIME-MD scale for at least 2 years of active symptoms.

These disorders have been proposed because the recognized somatoform disorders are either too restrictive or too broad. In a study of 119 primary care patients.

Proposed New Diagnoses

The proposed DSM-5 new diagnoses include the following:

- Complex post-traumatic stress disorder
- Depressive personality disorder
- Compulsive hoarding
- Olfactory Reference Syndrome
- Negativistic (passive-aggressive) personality disorder
- Relational disorder
- Skin Picking Disorder
- Sluggish cognitive tempo
- Binge Eating

6. Samanyaja and Nanatmaja Vikara, Nidanarthakara Vyadhi, Hetu Sankara, Lingasankara, Vyadhisankara, Vyadhi Awastha

तत्र विकाराः सामान्यजाः नानात्मजाश्च तत्र सामान्यजाः पूर्वमष्टोदरीये व्याख्याताः नानात्मजांस्त्विहाध्यायेऽनुव्याख्यास्यामः तद्यथा–अशीतिर्वातविकाराः चत्वारिंशत् पित्तविकाराः विंशतिः श्लेष्मविकाराः १० (Ca. su. 20)

Samanya Vikaras

These are causes due to single dosha or by combination of two or three doshas depending on etiological factors and their interaction with dhatus.

Classification of disease having 8 types :

इह खल्वष्टावुदराणि अष्टौ मूत्राघाताः अष्टौ क्षीरदोषाः अष्टौ रेतोदोषाः

1. Eight types of udara- Vataja, pittaja, Kaphaja, sannipataja, Yakrut and pleehodara (together), baddagudodara, chidrodara, Jalodara.

अष्टावुदराणीति वातपित्तकफसन्निपातप्लीहबद्धच्छिद्रदकोदराणि

2. Eight type of mootraaghata- Vataja, Pittaja, Kaphaja, Sannipataja, Ashmarija, Sharkaraja, Shukraja, Shonotaja.

त्राघाता इति वातपित्तकफसन्निपाताश्मरीशर्कराशुक्रशोणितजाः अष्टौ

3. Eight types of ksheera doshas- Vaivatnya, Vaigandhya, Vairasya, paicchilya, Phenasanghata, Roukshya, gourava, Bahu sneha.

दोषा इति वैवर्ण्यं वैगन्ध्यं वैरस्यं पैच्छिल्यं फेनसङ्घातो रौक्ष्यं गौरवमतिस्नेहश्च अष्टौ रेतोदोषा इति तनु शुष्कं फेनिलमश्वेतं पूत्यतिपिच्छलमन्यधातूपहितमवसादि च।।१।।

4. Eight types of retadosha- Tanu, Shushka, Phenila, Ashweta, Pooti, Piccila, Anyadhatu upahita, Avasaadi.

Classification Based on Seven Types

1. Seven types of Kushtas- Kapala, mandala, Audumbara, Rushyajivha, Pundarika, kakanaka, Sidhma.

सप्त कुष्ठानीति कपालोदुम्बरमण्डलर्ष्यजिह्वपुण्डरीकसिध्यकाकणानि

2. Seven types of Pidakas- Sharavcika, Kacchapika, Jalini, Sarshapi, Alaji, Vinata, Vidhradhi.

पिडका इति शरावि का कच्छपिका जालिनी सर्षप्यलजी विनता विद्रधी च।

3. Seven types of Visarpa- Vataja, Pittaja, Kaphaja, Sannipataja, Agni, Kardama, Granti.

सप्त विसर्पा इति वातपित्तकफाग्निकर्दमकग्रन्थिसन्निपाताख्याः

Classification of disease having six types

1. Six types of Atisaara- Vataja, Pittaja,Kaphaja, Sannipataja, Bhayaja, Shokaja.

षडतीसारा इति वातपित्तकफसन्निपातभयशोकजाः षडुदावर्ता इति वातमूत्रपुरीषशुक्रच्छर्दिक्षवथुजाः।।३।।

2. Six types of Udavarta- Vataja, Mootraja, Pureeshaja, Shukraja, Chardi, Kshavathuja.

Classification of disease having 5 types

1. Five types of gulma : Vataja, Pittaja, Kapaja, Sannipataja, Raktaja.

पञ्च गुल्मा इति वातपित्तकफसन्निपातशोणितजाः।

2. Five types of Pliharoga - Vataja, Pittaja, Kapaja, Sannipataja, Raktaja.

वातपित्तकफसन्निपातशोणितजाः पञ्च प्लीहदोषा इति

3. Five types of Kasa- Vataja, Pittaja, Kaphaja, Kshataja, Kshayaja

पञ्च कासा इति वातपित्तकफक्षतक्षयजाः।

4. Five types of Shwasa- Maha shwasa, urdwa shwasa, china shwasa, tamaka shwasa, kshudra shwasa.

महोर्ध्वच्छिन्नतमकक्षुद्राः पञ्च

5. Five types of hicca- Mahahicca, gambheera hicca, Vyapeta hicca, Kshudra Hicca, Annaja Hicca.

पञ्च हिक्का इति महती गम्भीरा व्यपेता क्षुद्राऽन्नजा च।

6. Five types of trushna - Vataja, Pittaja, Amaja, kshayaja, upasargaja

पञ्च तृष्णा इति वातपित्तामक्षयोपसंसर्गात्मिकाः।

7. Five types of chardi- Vataja, pittaja, Kapaja, Sannipataja, Dwishtarthasamyogaja

पञ्च छर्दय इति द्विष्टार्थ-संयो गजा वातपित्तकफसन्निपातोद्रेकोत्थाश्च

8. Five types of Arocaka- Vataja Pittaja, kaphaja, sannipataja, Dveshaja.

पञ्च भक्तस्यानशनस्थानामीति वातपित्तकफसन्निपातद्वेषाः

9. Five types of shiroroga - Vataja, Pittaja, kaphaja, Sannipataja, Krimija.

पञ्च शिरोरोगा इति पूर्वेद्दिशमभिसमस्य वातपित्तकफसन्निपातक्रिमिजाः

10. Five types of hridroga - Vataja, Pittaja, kaphaja, Sannipataja, Krimija.

पञ्च हृद्रोगा इति शिरोरोगैर्व्याख्याताः।

11. Five types of Pandu- Vataja, Pittaja, kaphaja, Sannipataja, Mrudbhakshanaja.

पञ्च पाण्ड रोगा इति वातपित्तकफसन्निपातमृद्भक्षणजाः

12. Five types of Unmada- Vataja, Pittaja, Kapaja, Sannipataja, Agantuja.

पञ्चोन्मादा इति वातपित्तकफ सन्निपातागन्तुनिमित्ताः

Classification of Diseases of Four Types

1. Four types of Apasmara- Vataja, Pittaja, Kaphaja, Sannipataja

चत्वारोऽपस्मारा इति वातपित्तकफसन्निपातनिमित्ताः चत्वारोऽक्षिरोगाश्चत्वारः कर्णरोगाश्चत्वारः प्रतिश्यायाश्चत्वारो मुखरोगाश्चत्वारो ग्रहणीदोषाश्चत्वारो मदाश्चत्वारो मूर्च्छाया इत्यपस्मारैर्व्याख्याताः चत्वारः शोषा इति साहससन्धारणक्षयविषमाशनजाः चत्वारि क्लैव्यानीति बीजोपघाताद्ध्वजभङ्गाज्जरायाः शुक्रक्षयाश्च।।५।।

2. Four types of netra roga- Vataja, Pittaja, Kaphaja, Sannipataja
3. Four types of karna roga- Vataja , Pittaja,Kaphaja, Sannipataja

4. Four types of prathishyaya- Vataja, Pittaja, Kaphaja, Sannipataja.
5. Four types of mukha roga- Vataja, Pittaja, Kaphaja, Sannipataja
6. Four types of madaroga - Vataja , Pittaja, Kaphaja, Sannipataja
7. Four types of grahani- Vataja , Pittaja, Kaphaja, Sannipataja
8. Four types of murcha- Vataja , Pittaja, Kaphaja, Sannipataja
9. Four types of Shosha- Sahasa, Vegadharana, Kshaya, Vishamashana.
10. Four types of Klabya- bijopaghata, dvajabhangaja, jaraja, shukrakshaya

Classification of Diseases of Three Types

त्रयः शोथा इति वातपित्तश्लेष्मनिमित्ताः त्रीणि किलासानीति रक्ताम्रशुक्लानि त्रिविधं लोहितपित्तमिति ऊर्ध्वभागमधोभागमुभयभागं च।।६।।

1. Three Shothas- Vataja , Pittaja, Kaphaja
2. Three types of Khilasa- Rakta, Tamra, Shukla
3. Thre types of Rakta pitta- Urdhwaga, Adhoga, ubhayaga

Classification of disease of two types.

द्वौ ज्वराविति उष्णभिप्रायः शीतसमुत्थश्च शीताभिप्रायश्चोष्णसमुत्थः द्वौ व्रणाविति निजश्चागन्तुश्च द्वावायामाविति बाह्यश्चाभ्यन्तरश्च द्वे गृध्रस्याविति वाताद्वातकफाश्च द्वे कामले इति कोष्ठाश्रया शाखाश्रया च द्विविधमाममिति अलसको विसूचिका च द्विविधं वातरक्तमिति गम्भीरमुत्तानं च द्विविधान्यर्शासीति शुष्काणयार्द्राणि च।।७।।(Ca.su.19)

1. Two types of jwara- Ushnabhipraya- Sheetabhipraya, sheetabhipraya- Ushna samutta.
2. Two types of vrunas- Nija, Agantu.
3. Two types of Ayaama- Antarayama, bahirayama.
4. Two types of Grudrasi- vataja, vatasleshmaja.
5. Two Ama- Alasaka, Vishuchika.
6. Two vatarakta- Uttana, Gambeera.
7. Two arshas- Sushka, Ardra.

Classification of disease of one type

एक ऊरुस्तम्भ इत्यामत्रिदोषसमुत्थः एकः संन्यास इति त्रिदोषात्मको मनः-शरीराधिष्ठानः एको महागद इति अतत्त्वाभिनिवेशः।।८।।

1. One Urusthambha- causes by Ama with tridosha
2. One Sanyasa- causes by tridosha in manas and sharira
3. One mahagada- Atatwabhinivesha

Classification of disease of twenty types

1. Twenty krimi-

विंशतिः कृमिजातय इति शूका पिपीलिकाश्चेति द्विविधा बहिर्मलजाः

Raktaja krimi-Keshada, lomada, romadweepi, sourasa, audumbara, jantumar.

शादा लोमादा लोमद्वीपाः सौरसा औदुम्बरा जन्तुमातरश्चेति षट् शोणितजाः

Sleshmaja krimi- Antrada, udaraveshta, hrudayada, curu, darbapushpa, sougandhika, Mahaguda.

अन्त्रादा उदरावेष्टा हृदयादाक्षुरवो दर्भपुष्पाः सौगन्धिका महागुदाश्चेति सप्त

Pureeshaja krimi- Kakeruka, makeruka, leliha, sasulaka, sausurada.

ककेरुका मकेरुका लेलिहाः सशूलकाः सौसुरादाश्चेति

and 2 types of bahya krime → शूक & लिक्षा → 20 types

2. Twenty types of prameha-

Ten types of kaphaja meha- udakameha, ikshubalika meha, sandrameha, sandraprasada meha, shukla meha, shukrameha, sitameha, sanaimeha, sikata meha, alala meha.

विंशतिः प्रमेहा इत्यदकमेहइक्षुबालिकारसमेहश्च सान्द्रमेहश्च सान्द्र प्रसादमेहश्च शुक्लमेहश्च शुक्रमेहश्च शीतमेहश्च शनैर्मेहश्च सिकतामेहश्च

Six types of pittaja meha- neelameha, rakta meha, manjista meha, kaalameha, haridra meha.

लवणमेहश्च रक्तमेहश्च मञ्जिष्ठामेहश्च हरिद्रामेहश्चेति षट् पित्तनिमित्ताः

Four types of Vataja meha- Vasameha, Majja meha, hastimeha, madhumeha.

वसा मेहश्च मज्जामेहश्च हस्तिमेहश्च मधुमेहश्चेति चत्वारो वातनिमित्ताः

3. Twenty types of yoni rogas;

तिः प्रमेहाः विंशतियोनिव्यापद इति वातिकी पैत्तिकी श्लैष्मिकी सान्निपातिकी चेति चतस्रो दोषजाः दोषदूष्यसंसर्गप्रकृतिनिर्देशैरवशिष्टाः षोडश निर्दिश्यन्ते तद्यथा रक्तयोनिश्चारजस्का चावरणा वातिचरणा च प्राक्चरणा चोपप्लुता च परिप्लुता चोदावर्तिनी च कर्णिनी च पुत्रघ्नी चान्तर्मुखी च सूचीमुखी च शुष्का च वामिनी च षण्ढयोनिश्च महायोनिश्चेति विंशतियोनिव्यापदो भवन्ति।।९।।

Vataja, pittaja, Kaphaja, Raktayoni, Arajaska, Acarana, Aticarana, Prakcharana, Upapluta, Paripluta, Udavartini, Karani, Putraghni, antarmukhi, suci mukhi, suska, Vamini, Shanda yoni, mahayoni.

Nanatmaja Rogas

1. Vataja Nanatmaja Roga:

तत्रादौ वातविकाराननुव्याख्यास्यामः

तद्यथा- - -नखभेदश्च विपादिका च पादशूलं च पादभ्रंशश्च पादसुप्तता च वातखुड्डता च गुल्फग्रहश्च पिण्डिकोद्वेष्टनं च गृध्रसी च जानुभेदश्च जानुविश्लेषश्च ऊरुस्तम्भश्च ऊरुसादश्च पाङ्गुल्यं च गुदभ्रंशश्च गुदार्तिश्च वृषणाक्षेपश्च शेफस्तम्भश्च वङ्क्षणानाहश्च श्रोणिभेदश्च विड्भेदश्च उदावर्तश्च खञ्जत्वं च कुब्जत्वं च वामनत्वं च त्रिकग्रहश्च पृष्ठग्रहश्च पार्श्वमर्दश्च उदरावेष्टश्च हृन्माहेश्च हृद्ग्रवश्च वक्षउद्धर्षश्च वक्षउपरोधश्च वक्षस्तोदश्च बाहुशोषश्च ग्रीवास्तम्भश्च मन्यास्तम्भश्च कण्ठोद्ध्वंसश्च हनुभेदश्च ओष्ठभेदश्च अक्षिभेदश्च दन्तभेदश्च दन्तशैथिल्यं च मूकत्वं च वाक्सङ्गश्च कषायास्यता च मुखशोषश्च अरसज्ञता च प्राणनाशश्च कर्णशूलं च अशब्दश्रवणं च उच्चैःश्रुतिश्च बाधिर्यं च वर्त्मस्तम्भश्च वर्त्मसङ्कोचश्च तिमिरं च अक्षिशूलं च अक्षिव्युदासश्च भ्रूव्युदासश्च शङ्खभेदश्च ललाटभेदश्च शिरोरुक् च केशभूमिस्फुटनं च अर्दितं च एकाङ्गरोगश्च सर्वाङ्गरोगश्च पक्षवधश्च आक्षेपकश्च दण्डकश्च तमश्च भ्रमश्च वेपथुश्च जृम्भा च हिक्का च विषादश्च अतिप्रलापश्च रौक्ष्यं च पारुष्यं च श्यावारुणावभासता च अस्वप्नश्च अनवस्थितचित्तत्वं च इत्यशीतिर्वातविकारा वातविकाराणामपरिसंख्येयानामाविष्कृततमा व्याख्याताः।।९१।। (Ca. su. 20)

Nakhabeda, vipadika, vatasula, paadabramsha, paadasuptata, vatakhuddata, vatagulpha, pindikodweshta, grudrasi, janubedha, jaanivishlesha, urusthambha, urusaada, pangulya, gudabramsha, gudarti, vrishannkshepa, shephasthamba, vanshanamaha, shronibedha, udaavarta, kanjatwa, vidbedha, vaamanatwa, trikagraha, katigraha, prusta graham, parshwashula, udaraveshta, hriddrava, hrinmoksha, vaksha toda, vakshaudgarsha, vakshoparodha, bahososha, greeva sthambha, manya sthambha, kantodhwamsa, hanubedha, ostabedha, akshibedha, talu bedha, danta bedha, danta shaitilya, mukatwa, gadgadatwa, vaakgraha, kashayasyata, mukhasosha, arasajnata, graananaasa, karnasula, ashabdata, ucchaishravana, badiryata, vartmasthambha, vartmasan-cocha, timira, astisula, akshisula, akshivyudasa, bruvyu-dasha, shankhabedha, lalatabedha, siroruk, kesabhumi sputana, ardita, ekangaroga/pakshavada, sarvangaroga, akshepa, dandaka/danda-patanaka, tama, brahma, shrama, vepatu, jrumbha, hikka, vishaada, pralapa, glaani, roukshya, parushya, shyavarunabhasata, aswapna, anavastita chittatwa.

2. Pittaja Nanatmaja Vikara

पित्तविकाराश्चत्वारिंशतमत ऊर्ध्वमनुव्याख्यास्यामः- -ओषश्च प्लोषश्च दाहश्च दवथुश्च धूमकश्च अम्लकश्च विदाहश्च अन्तर्दाहश्च अंसदाहश्च ऊष्माधिक्यं च अतिस्वेदश्च अङ्गस्वेदश्च अङ्गगन्धश्च

अङ्गावदरणं च शोणितक्लेदश्च मांसक्लेदश्च त्वग्दाहश्च मांसदाहश्च त्वगवदरणं च चर्मदलनं च रक्तकोठश्च रक्तविस्फोटश्च रक्तपित्तं च रक्तमण्डलानि च हरितत्वं च हारिद्रत्वं च नीलिका च कक्षा च कामला च तिक्तास्यता च लोहितगन्धास्य ता च पूतिमुखता च तृष्णाधिक्यं च अतृप्तिश्च आस्यविपाकश्च गलपाकश्च अक्षिपाकश्च गुदपाकश्च मेढ्रपाकश्च जीवादानं च तमः प्रवेशश्च हरितहारिद्रनेत्रमूत्रवर्चस्त्वं च इति चत्वारिंशत्पित्तविकाराः पित्तविकाराणामपरिसंख्येयानामाविष्कृततमा व्याख्याताः।।१४।। (Ca. su 20/14)

Osha, plosha, daha, dhava, davathu, dhumaka, dhumodgaara, malaka, vidaha, antardaha, amsadaha, ushmaadikya, atisweda angagandha, angaavadarana, shonitakleda, maamsakleda, twakdaha, twagavadarana, charmadala, raktakota, raktavispota, raktapitta, raktamandala, haritatwam, haaridratvam, neelika, kakshaa, kamala, tiktasyata, lohitagandhasyata, putimukha, trishnadhikya, atrupti, aasyavipaaka, galapaka, akshipaaka, medrapaka, jeevadaana, tamapravesha, harita haridra netra, mutra and varca

3. Kaphaja Nanatmaja Vikara

श्लेष्मविकारांश्च विंशतिमत ऊर्ध्वं व्याख्यास्यामः तद्यथा---तृप्तिश्च तन्द्रा च निद्राधिक्यं च स्तैमित्यं च गुरुगात्रता च आलस्यं च मुखमाधुर्यं च मुखस्रावश्च श्लेष्मोद्गिरणं च मलस्याधिक्यं च बलासकश्च अपक्तिश्च हृदयोपलेपश्च कण्ठोपलेपश्च धमनीप्रतिचयश्च गलगण्डश्च अतिस्थौल्यं च शीताग्निता च उद्दर्दश्च श्वेतावभासता च श्वेतमूरनेत्रवर्चस्त्वं च इति विंशतिः श्लेष्मविकाराः श्लेष्मविकाराणमपरिसंख्येयानामाविष्कृततमा व्याख्याता भवन्ति।।१७।।

Tripti, tandra, nidradhikya, sthaimitya, gurugaatrata, aalasya, mukhamadurya, sleshmodgirana, maladhikya, balasaka, apakti, hridayopalepa, kantopalepa, dhamanipratichaya, galaganda, atisthoulya, seetagnita, udarda, shwetavabhasata, shwetamutra-netra-varca.

Nija and Agantu Roga

चत्वारो रोगा भवन्ति--आगन्तुवातपित्तश्लेष्मनिमित्ताः तेषां चतुर्णामपि रोगाणां रोगत्वमेकविधं भवति रुक्सामान्यता द्विविधा पुनः प्रकृतिरेषाम् आगन्तुनिजविभागात् द्विविधं चैषामधिष्ठानं मनःशरीरविशेषात् विकाराः पुनरपरिसंख्येयाः प्रकृत्यधिष्ठानलिङ्गायतनविकल्पविशेषपरि-संख्येयत्वात्।।३।। (Ca. su. 20/3)

Every classification of disease has a purpose. Disease may be of one type because in every disease pain, or suffering is common. Based on prakruti it may be of two types that is.

1. Nija- Causes due to dosha dushti like vata, pitta, kapha. The dushti may be in the form of vriddi prakopa, leena, sthambhita and so on. Dosha dushti is out come of varios ahara and viharaja nidanas with respect to prakruti. Disequilibrium of dosha and association with respective dhatus and malas in particular ashayas give particular niji vikara.
2. Agantu- it is causes due to external agents. Physical trauma by any sharp instruments, contact with poisons, bite of poisonous animals, invasion of evil spirits. Dosha dushti takes place after trauma. In nija vikaras dosha dushti takes place first and then disease manifestation.

Disease becomes innumerable due to variation or taratama in vriddi kshaya of dosha, involment of dhatu, mala. Variation again comes when multiple ashayas, multiple srotas with various dushti prakaras takes place.

Nidanarthakara Rogas

This is the state of progression of pathology where one disease becomes the cause of onset of another disease. In the initial stage the disease will manifest and later on the same disease act as causative factor for the other disease. The disease manifested first will continue to exist even after the manifestation of secondary disease.

निदार्थकरो रोगो रोगस्याप्युपजायते। तद्यथा ज्वरसन्तापाद्रक्तपित्तमुदीर्यते।
रक्तपित्तज्ज्वरस्ताभ्यां शोषश्चाप्युपजायते। प्लीहाभिवृद्द्यजठरं जठराच्छोथ एवच।
अर्शोभ्यो जाठरं दुःखं गुल्मश्चाप्युपजायते। दिवास्वप्नादिदोषैश्च प्रतिश्यायादयो कासः कासात् सञ्जायते क्षयः।
क्षयोरोगस्य हेतुत्वे शोषस्याप्युपजायते।। (च.नि. ८/१६)

Some of the examples are : jwara when increases excessively in tapa it causes raktapitta. On the contrary Raktapitta cause jwara. Both jwara and raktapitta can cause sosha. Pleehodara or any other variety of udara will lead to jalodara and jalodara causes Sopha. On the other hand Arshas causes both gulma and udara (jatara). From divaswapna etc nidanas Pratishyaya can manifest which in turn lead to kasa. Kasa causes Kshaya. And Kshaya cause Sosha.

ते पूर्व केवला रोगाः पश्चाद्देत्वर्तकारिणः। उभयार्थकरा दृष्टास्तथैवैकार्थकारिणः।(च.नि.८/२०)
कश्चिद्हि रोगो रोगस्य हेतुर्भूत्वा प्रशाम्यति। न प्रशाम्यति चाप्ययो हेतुत्वं कुरुतेऽपिच।

These disease at first manifest as single disease but in later progression the same disease causes manifestation of another disease. It is again subdivided into two types;

1. Ekarthakari- the primary disesease will discontinue when it

causes the manifestation of next disease ex: jwara causes raktapitta. After raktapitta is manifested jwara disappears

2. Ubhayarthakari- The primary disease willalso continue after manifestation of another disease. Eg; Jwara causes raktapitta. After manifestation of raktapitta jwara continues to manifest.

Linga Samkara

Lingasamkara is combination of multiple symptom in one single disease. It can also be called as syndrome or symptom complex. Eg : Trirupa, Shad rupa, Ekadasharupa of Rajayakshma.

In this disease multiple systems are involved therefore the disease presents with symptom complex. Respiratory system symptoms like kasa, swarabedha. GIT system complaints like chardi, aruchi, atisara. Musculoskeletal system symptoms like amsa shoola, parvabedha. Hematological symptoms like raktapitta. All these are manifested in single patient from single disease. The number of symptoms may vary from patient to patient on the basis of roga bala and rogi bala.

Hetu, Linga, Vyadhi, Samkara

एको हेतुरनेकस्य तथैकस्यैव एवे हि। व्याधेरेकस्य चानेको बहूनां बहवो अपि च।।
ज्वरभ्रमप्रलापाध्या दृश्यन्ते रूक्षहेतुजाः। रूक्षेणैकेन चाप्येको ज्वर एवोपजायते।।
हेतुभिर्बहूभिश्चैको ज्वरो रूक्षादिभिर्भवेत्। रूक्षादिभिर्ज्वराध्याश्च व्याधयः संभवन्ति हि।।
लिंगं चैकमनेकस्य तथैवैकस्य लक्ष्यते। बहून्येकस्य च व्याधेर्बहूनां स्युर्बहूनि च।।
विषमारम्भमूलानां लिंगमेकं ज्वरो मतः। ज्वरस्यैकस्य चाप्यैकः संतापो लिंगमुच्यते।।
विषमारम्भमूलैश्च ज्वर एको निरुच्यते। लिंगैरेतैर्ज्वरश्वासहिक्काध्याः सन्ति चामयाः।।(च.नि.८/२४-२९)

The functional diversity of nidana or their productivity of disease can occur in 4 ways.

- Single causative factor can cause many disease. Eg : Ruksha guna can produce Jwara, Brama, Pralapa.
- Single cause can produce only one disease eg; Ruksha guna can produce jwara alone.
- Many causative factors can produce one disease- for example ruksha guna, bhotabhishanga, abhigata, vishada can produce jwara.
- Many causative factors produce many disease eg; ahitahara, ativyayama, chinta can cause Jwara, Atisaara, Pandu.

- Similarly one single symptom may be seen in one disease eg: संताप is chief symptoms of jwara.
- Multiple symptom can be seen in one disease eg: Aruchi, angasaada, dourbalya, santapa all can be seen in jwara.
- One common symptom can be seen in many disease ey: ज्वर is seen in Rajayakshma, pittaja vrina, Pittaja pandu, Raktapitta, Vidradhi.
- Many disease may have many manifestations ey: Jwara, shwasa, hicca are seen in Rajayakshma, Raktapitta, Udara, Kumbha-kamala.

Vyadhi Samkara

It is the complex condition where there is combination of many disease or overlapping of multiple disease in a single patient. Two basic causes are attributed to this complex combination. Basically it is due to improper management of existing disease which results in origin of another another disease. This makes treatment more difficult to cure. For example in Ama atisara if sthambana chikitsa is given then the ajeerna anna stays inside the body and later produces anaha, adhmana, shula, grahani roga. The disease has turned from atisara to various other GIT disease of severe form. The combination of atisaara with anaha, adhmana, shula, grahani all in a single patient together is vyadhi samkara. Instead in amatisaara anulomana should be given to eliminate ajeerna anna and ama. If done so there is no turning of disease into complex form. Rather it cures the disease and patient becomes free of symptoms.

एवं कृच्छ्रतमा नृणां दृष्यन्ते व्यधि सङ्करा। प्रयोगापरिशुद्दत्वात्तथा चान्योन्यसंभवात्।।
(च.नि.८/२०-२२)

प्रयोगः शमयेध्व्याधिं यो अन्यमन्यमुदीरयेत्। नासौ विशुद्धः शुद्धस्तु शमयेध्यो न कोपयेन्।।
(च.नि. ८/२२,२३)

Thus the combination of multiple disease together with its symptom is called as Vyadhi samkara.

Vyadhi Avastha

In the body during pathological state when दोष-दूष्य समुर्च्छन take place, it leads to formation of disease. It follows a path consisting of onset duration and progress or retrogression.

As the nidana enter the body it influences doshas and then dushyas. Based on the intensity of nidana and the nature of disease there may be various presentation in every disease. In the period of duration of disease differ rent stages appear due to several factors and they are called the stages of disease or व्याधि अवस्था.

व्याधि अवस्था denotes the changes that occur from time to time in a disease. These changes depend on two opposite force acting on the body.

1. The factors that are responsible for increasing the disease pathology.
2. The forces like व्याधिक्षमत्व and appropriate fulfillment of पादचतुष्य which are helpful in managing and curing disease.

Therefore during approach to the patient, for the diagnosis and treatment the various stages of the disease and its form has to be understood. Therefore a good physician should observe for दूष्यु देश, बल, काल, अनलं, प्रकृति, वयः, सत्व, सात्म्य, आहार and the व्यधि अवस्थ while determining the दोषs and mode of treatment to be adopted.

The factors like दूष्यु देश, बल, काल, अनलं, प्रकृति, वयः, सत्व, सात्म्य, आहार may favour either of these sides. When they are favourable for व्यधि the pathology that will progress and when they are helpfull for treatment disease will subside.

दूष्यं देशं बलं कालमनलं प्रकृतिं वयः । सत्वं सात्म्यं तथाहारमवस्थाश्च पृथग्विधाः ।
सूक्ष्मसूक्ष्माः समीक्ष्यैषां दोषौषधनिरूपणे । यो वर्तते चिकित्सायां न स स्खलति जातुजित् ।
(अ.ह.सू./१२/६६-६७)

Various Vyadhi Avasthas

1. Uttana and Ghambheera Avastha

This refers to the stage of disease that signifies at which dhatu level the disease stays. Does the disease stay in superficial dhatu level or deeper level.

* **उत्तान अवस्थः** Only the superficial dhatus are involved in the samprapti. That is the disease that lies between Rasa, Raktha, Maamsa, Meda dhatus are considered as uttana dhatus. The diseases in this level is called as Uttana dhatu avastha.
* **गम्भीरावस्थाः गम्भईरानुगता इति गम्भीरं मज्जाधातुगत इत्यर्थः ।** (च.सू.२८/७)

Deeper धातु s like meda, Asthi, Majja, Shukra are involved in the samprapti. Susruta said that this stage is called अवगाढ अवस्था. In this stage Massive destruction of deeper dhatus take place.

Eg: उत्तान एवं गम्भीर वातरक्त

When the pradhaana vyadhi limits at twak, it is called as uttaana vatarakta. In this vivarnataa of twak, as well as daha, peeda of twacha is seen. It is twak maamsa asrita.

When the vikruti of vaatarakta spread from mamsa ,meda and deeper dhatus as well as sandhi, it is called as Gambheera vatakta. In this shwayathu, sthabdha, daha, toda spurna, paka are seen.

2. Nava Avastha and Jeerna Avastha

नव or तरुणावस्थाः Newly manifested disease is called as nava. It may varies from disease to disease. In jwara 7 days from the time of manifestation is called as nava avastha.

जीर्णावस्थाः दौर्बल्यात् देहधातूनां ज्वरो जीर्णानुवर्तते । (च.चि. ३/२९१)

जीर्णावस्था is a chronic stage when weakness develop in the Dhatus. Deficient dhatus fail to perform their normal protective function leading to chronicity of the disease.

3. Dosha Paka and Dhatu pakavastha

दोषपाक अवस्था

It is a stage favorable to treatment of disease because aama separated from doshas.

दोषप्रकृति वैकृत्यं लघुता ज्वर देहयोः । इन्द्रियाणां च वैमल्यं दोषाणां पाक लक्षणम् ।।
(मा.नि. २/६६)

This condition is stated to be nirama stage of dosha as a result, diseases either cures completely or symptoms start diminishing gradually or agitated doshas starts coming towards koshta. It is essential stage of recovery of disease.

During dosha paka, certain symptoms will manifest

* The symptoms of the doshas involving in the development of disease start diminishing.
* In case of fever the increased temperature in affected person starts to subside
* As the Ama starts separating from Doshas, dhatus and srotases, the bodywill be relieved from heaviness. Laghuta or sense of relaxation will be produced.
* In the Dushta state of doshas and ama state, the quality of sense organ in response to sensation remain depressed, but in the stage of dosha paka, this depression is removed .The sense organ tends to respond to the sensation and clarity of sense perciption is improved.

धातुपाक

धातुपाकाद्धन्ति, मलपाकाद्विमुञ्चतीति व्यवस्थितविकल्पः धातुमल पाकविकल्पे च दैवमेव हेतुः। उत्तरोत्तरोगवृद्धिबलहानिभ्यां शुक्रादिधातुसहितमूत्रादीनां च धातुपाको ज्ञेयः, अन्यथा तु मलपाकः; यदुक्तं निद्रानाशो हृदिस्तम्भो विष्टम्भो गौरवारुचि। अरतिर्बलहानिश्च धातूनां पाकलक्षणम् ।।

धातुपाक & दोषपाक are the two different process responsible for the prognosis of diseases

धातुपाक means worsening of the disease condition due to its advance stage along with loss of streangth (बलहानि) and elimination of शुक्रादि धातुs via मुत्रादि मलाs

लक्षण

- निद्रानाश-loss of sleep
- हृदिस्तम्भ-unusual feeling in chest such as pressure, heaviness, constriction etc.
- विष्टम्भ-constipation
- गौरव-heaviness

- अरुचि-anorexia
- अरति-restlessness
- बलहानि-loss of streangth

Dhatupaka causes damage to cell and tissues. This stage is considered to be serious stage in progress of pathogenesis of diseases. Dhatus does not perform their normal function as a result dushti in disease. The physician should attempt to create doshapaka and prevent dhatupaka as much as possible.

Advanced devices of modern technology are very helpful in tracing the symptoms of धातुपाक for example, the presence of albumin in urine suggest मांसधातुपाक,

High level of SGPT in blood is suggestive of धातुपाक of liver tissue. Ketone bodies in urine suggests धातुपाक of मेधोधातु

4. Ama and Niramavastha

Presence of Ama with dosha or in the disease is called as amavastha, dissociation of ama from the same is called as niramavastha. General features of presence of ama in the body that are suggestive of amavastha are:

स्रोतोरोध बलभ्रंश गौखानिलमूढताः । आलस्यपक्तिनिष्ठीवमलसंगारुचिक्लमाः ।
लिङ्गं मलानां सामनं, निरामाणां विपर्ययः । (अ.ह.सू. १३/२३-२४)

* स्रोतोरोध- blockage in srotases
* बलभ्रंश-loss of streangth
* गौरव-heaviness of the body
* अनिलमूढता-blockage in functions of vaata
* आलस्य-laziness
* अपक्ति-indigestion
* निष्ठीव-spitting continiously
* मलसंगा-constipation
* अरुचि-loss of appettite
* क्लमा-weakness

निरामावस्थ

The symptoms are opposite to Amavastha. .ie.lightness of the body, प्रसन्नता of इन्द्रिया, vatanulomana, proper taste perception etc.

सामव्याधि

आमेन तेन संपृक्ता दोषा दूष्याश्च दूषिता। सामा इत्युपदिश्यन्ते येच रोगास्तुदुद्भवाः।। (अ.ह.सू.)

Apakwa annarasa or apakwa rasadhatu when mixed with dosha and dooshya, it is said to be saama the disease arising due to this is called saamavyadhi.

निराम व्याधि

Lightness of body, प्रसन्नत of इन्द्रिया, वातनुलोमन, proper ruchi & appetite starts to manifest in a disease, such a disease is said to be निराम व्यधि।

5. Antarvegi and Bahirvegi Avastha

अन्तर्वेगी The disease runs within internal enviroments of the body symptoms are vaguely expressed.

अन्तर्दाहो अधिकस्तृष्णा प्रलापः श्वसनं भ्रमः। सन्ध्यस्थिशूलमस्वेदो दोषवर्चोविनिग्रः।। (च.चि. ९/३९)

* अन्तदार्ह-internal burning sensation
* अधिकस्तृष्णा-exessive thirst
* प्रलापः-delirium
* श्वसनं-dyspnoea
* भ्रमः-gidiness
* सन्ध्यस्थिशूल-pain in bones and joints
* अस्वेद-absence of sweating
* दोषवर्चोविनिग्रः-difficulty in the passage of urine and feaces

Symptoms of pitta and vaata are predominant in this condition.

बहिर्वेगी

सन्तापो अभ्यधिको बाह्यतृष्णादीनं च मार्दवम्। बहिर्वेगस्य लिङ्गानि सुखसाध्यत्वमेव च।।

In this condition,burning sensation occures mainly on skin and not inside the body. All other symptomslike thirst,delirium etc. get diminished and disease become sukha saadhya the disease out busts the symptoms and gets manifested fully with all its intensity.

6. Aashukari and Chirakari

आशुकारी: – The condition of suddden onset and short duration of disease. The disease of Acute onset is called as ashukari.

Eg: visoochikaa, pitta raktaja pravaahika.

चिरकारी: The disease which are chronic in nature are called as chirakari.

चिरस्थित इति देहे विरकालवस्थेन कृतमूलत्वात् कष्टतमो असाध्यः। (च.सू. २८/७)

* Persistence of symptoms for longer period.
* That is why our achaaryas said that if disease become one year old is incurable.
* कृछ्रसाध्य
* **Eg:** kushta, jeernajwara, pakshaagadha, prameha

वृद्धिस्थानक्षयावस्थां रोगणामुपलक्षयेत्। ससूक्ष्मामपि च प्राज्ञे देहग्निबलचेतसाम्। व्याध्यवस्थाविशेषान् हि ज्ञात्वा ज्ञात्वा विचक्षण।

If treatment given after a comprehensive understanding of stage of doshas, condition of body and agni, the mental states and व्याधि अवस्था, vaidya will attain four fold success. The treatment must change according to व्याधि अवस्था . for example in vrina the surgical intervention should not be given in Amavastha. Aharuvadi upanaha is applied to the vrina and awaited for niramata or pakwavastha to undergo bhedana or visravana.

भैषज्यमनवस्थायं पथ्यमाप्यवचारितम्। गुणं न किंचित् कुरुते दोषयेव तु कल्पते।। (काश्यप)

Kashyapa says that if medicine and pathya are admininistered during improper avasthaas, then they produce greater form of dushti of doshas. Hense value and efficasy of treatment depend on a comprehensive and minute knowledge of various avasthaas

A proper knowledge of avasthaas are helpful in preventing the development of more severe stage of disease.

A knowledge of avasthaas also helps in determining the साध्यासाध्यता of the disease.

Treatment becomes successful only after the proper assessment of disease based on अवस्थाs

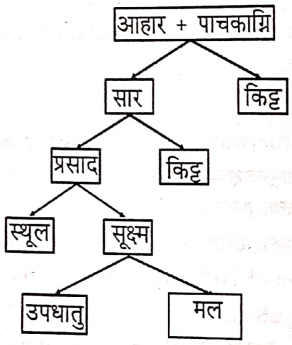
Treatment may change according to व्याधि अवस्थाs otherwise will leads to complication.

व्याधि अवस्था plays an inevitable role in diagnosis, treatment, as well as prognosis of the disease.

7. Dhatu, Updhatu, Mala and Indriya Pradoshaj Vikara

उपधातुः धातुभ्यश्च उपजयन्ते तस्मात् उपधातवः।

upadhatus are formed from dhatus.



Formation of Upadhatu

रस	स्तन्य	आर्तव
रक्त	कण्डरा	सिरा
मांस	वसा	षट्त्वचा
मेद	स्नायु	

कर्म धारण

- Sthanya provides nourishment and health to the infant
- आर्तव-capable of producing garbha.
- कण्डरा-helps in flexion and extension of the body.
- सिरा-contraction and expansion of bodily organs and vessels to supply essentials.
- वसा-provides lubrication maintain vitality, gives strength.
- त्वचा-covering and protection of body.
- स्नायु-it binds mamsa, asti and medas.

उपधातुप्रदोष विकार

स्नायौ सिराकण्डराभ्यो दुष्टाः क्लिक्ष्नन्ति मानवम्। स्तम्भसंकोचखल्लीभिर्ग्रन्थिस्फुरणसुप्तिभिः।।
(च.सू. २८/२१)

Dushita dosha move to the sthana of upadhatus and causes vikara like- sthamba, (stiffness), sankocha (spasm), khalli (contractures), granthi, (cysts and nodules) sphurana (pulsation or fasiculations) and supti (numbness or parasthesia).

उपधातु प्रदोषज विकार

स्नायौ सिराकण्डराभ्यो दुष्टाः क्लिक्ष्नन्ति मानवम्। स्तम्भसंकोचरवल्लीभिर्ग्रन्थिस्फुरणसुप्तिभिः।।
(Ca.Su. 28/21)

Dushita Doshas will move to sthana of upadhatus that is in snayu, sira, kandara (nerves, tendons ligaments) and cause sthambha (stiffness), sankoch (spasm) khalli (contractures), granthi (cysts and nodules) sphurana (pulsation and fasciculations) and supti (numbness of parasthesia).

Indriya pradoshaja Vikara:

इन्द्रियाणि समाश्रित्य प्रकुप्यन्ति यदा मलाः। उपघातोपतापाभ्यां योजयन्तीन्द्रियाणि।।
(Ca.Su. 28/2)

Dushita doshas will move to wards the sthanas of indriya and cause disease of sense organs in following ways:

1. इन्द्रिय उपघान-विनाश-Destruction
2. इन्द्रिय उताप-विकलाता/कष्ठ-Deformity
3. इन्द्रियाणीति-शरीरावयव गत व्याधि- Diseased sense organ

8. Concept of Ashta Mahagada

Maha roga or Mahagada are such disease which possess qualities like Durvijneya, duschikitsa, dustara, aristakaaraka, daaruna, ghora likewise. That means diseases having multiple symptoms, varied presentation, and association of complications often confuse physician. Thus disease becomes complex to understand and identify, due to complexity treatment and prognosis becomes poor.

The disease having above features may be seen in arsha, gulma, grahani, vatavyadhi, raktapitta. All such diseases can be clubbed under one umbrella and can be called as MAHAGADA.

महागद इति मारणात्मकत्वादसाध्यावाच्च महत्वमेषामिति (दल) सु.सू. ३३/४)
महरोगाः सुदुस्तराः (अ.ह.नि. ८/३०)
महारोगाः सुदुस्तरत्वादेव तेषां महारोगत्वम्, (भेल.इ. ५/६)
महान् घोरानिष्टकारकः रोगः यद्वा महाजन्मान्तरीणमुक्तविशिष्टातिशयपातकेन जनितः रोगः पापरोगः महरोगेण वा भितप्तः.....

Those disease which are severe, aggressive, complicated and fatal are considered as Mahagada. Mahagada is outcome of papa karma done in past or present life. It is unfortunate to the parient to suffer from pathetic illness.

There are 8 varieties of mahagada and thus named as Astamahagada

वातव्याधिः प्रमेहश्च कुष्टमर्शोभगन्दरम्। अश्मरी मूढगर्भश्च तथेवोदरमष्टमम्।।
अष्टावेते प्रकृत्यैव दुश्चिकित्स्या महगदाः।। (सु.सु. ३३/४)

Neurological disease (vatavyadhi), prameha (urinary disease) kushta (skin diseases). Arsha (piles), bhagandara (fistula), ashmari (caliculi), moodha garbha (dead foetus), udara (ascitis). These 8 disease give hard suffering to patient and these are difficult to cure.

वातव्याध्याश्मरीकुष्टमेहोदरभगन्दराः। अर्शांसि ग्रहणीत्यष्टो महारोगाः सुदुस्तराः।। (अ.ह.नि. ७/३०)

Neurological disease (vatavyadhi), ashmari (caliculi), kushta (skin diseases) prameha (urinary disease) udara (ascitis). bhagandara (fistula) Arsha (piles) grahani (digestive disorders) are called as mahagada which are severe and difficult to cure.

अपस्मारः क्षयः कुष्ट रक्तपित्तमथोदरम्। गुल्मश्चमधुमेहश्च दीर्घरोगा भवन्ति ते।। (भे.इ. ५/६)

Epileptic disorder (apasmara) kshaya (depleting disorder), kusta (skin disease), rakta pitta (bleeding disorder, udara (ascitis) gulma (moveable mass) madhumeha (urinary disorder) and all chronic disease are mahagadas.

The upadravas of mahagadas make disease more worse :

प्राणमांसक्षयः शोषक्षुण्णा च्छर्दिज्वरस्तथा। अतिसारश्च मूर्च्छाच हिक्का श्वासस्तथैव च।।
एतैरुपद्रवैर्जुष्टान् सर्वानेव विवर्जयेत्। (सु.सू. ३३/५)

Rogas are associated with decrease of vital signs, muscle bulk, undergo degeneration and depletion, vomiting, fever and loose stools. Diminishion of mental awakeness or fainting, respiratory distress all are considered as complications that take the patient to fatality.

●

9. Introduction to Ashta Nindita

Introduction

- External appearance is gift of god. This gift is influenced by पूर्वजन्मकृत पाप कर्म, बीज, दोष, मातृज अहार विहार during गर्भिणी अवस्था.
- "Guilty should be punished" so the disease or health which we have imbibed is based on deeds done on the past, if any sympathy or concern is shown to the culprit, that person is also considered as criminal, so allow the culprit to receive the punishment which paves the way for karma kshaya.
- Nindita's are those who were not allowed to participate in auspicious ceremony, public gathering or any appearances in front of any community.

The **word nindita means**

- ✓ Blamed
- ✓ Censured
- ✓ Abused
- ✓ Defamed
- ✓ Despire
- ✓ Revile
- ✓ Ridicule
- ✓ Strong disapproval

The **word purusha means**

- Men, mankind
- A member or representative of generation

Asto**nindita Purushas are**

इह खलु शरीरमधिकृत्याष्टौ पुरुषा निन्दित भवन्ति तद्यथा---अतिदीर्घश्च अतिह्रस्वश्च अतिलोमा च अलोमा च अतिकृष्णाश्च अतिगौरश्च अतिस्थूलश्च अतिकृशश्चेति।। (Cha.Su. 21/3)

1. अति दीर्घ - Excessively tall
2. अति ह्रस्व - Excessively short
3. अति लोम - excessive hair in the body
4. अलोम - absence of body hair
5. अतिकृष्ण - excessively dark in skin
6. अति गौर - excessively fair
7. अति स्थूल - excessively stout
8. अतिकृश - excessively lean

Effects of Atideerga (Gigantic Structure)

- ✓ Excessive oxygen demand due to tall structure
- ✓ Excessive nutritional demand
- ✓ Overload to cardiac function
- ✓ Psychological depression
- ✓ Circulatory failure.
- ✓ Early degeneration.

Effects of Atihruswa/Too Short Structure

- Growth retardation
- Inadequate growth and development of organs
- Poor health ststus.
- Psychological depression
- Short life span

Effects of Atiloma (Chromosomal or Hormonal cause)/ Excessive Hair

- Cosmetic disfiguration leading to psychological upset
- In the condition of Hirsutism due to the hormonal imbalance the fertility decreases.

Effects of Aloma/Seanty hair or absolutely no hair

- Alopecia universallis
- Congenital hypotrichosis
- Kleinfelter syndrome

Absence of body hair or minimal growth of body hair can also be seen in kleinfelter syndrome. Is caused by extra x-chromosome present in the male karyotype and occurs in about 1 in every 400 men. This is the most common sex chromosome syndrome marked by primary testicular failure affected persons have small firm testis, gynecomastia, minimal or complete absence of body hair and infertility.

Effects of Ati Gaura (Vitiligo/albinism)/Excessively fair skin

- The actual skin color of different humans is affected by many substance the single most important substance determining human skin color is the pigment melanin.

- Melanin is produced within the skin cells called melanocytes. And it is the main determinant of the skin color of darker skinned humans.
- Persons with the light skin is determined mainly by the bluish white connective tissue under the dermis and by the hemoglobin circulating in the veins of the dermis.
- The red color underlying the skin becomes more visible especially in the face when the person does physical exercise or stimulation of the nervous system (anger & fear) the arterioles dilate.
- Albinism– Genetic, non pathological, partial or total absence of melanin pigment in skin, hair and eyes. It is often accompained by-

  Astigmatism

  Photophobia

  Nystagmus

Effects of Atikrushna (hyper pigmentation)/Excessively dark skin

- Increased skin pigmentation or darkening of the whole body skin, directly due to an increased amount of melanin pigment in the skin.
- Generalized skin discoloration caused by either an increase in the production of melanin by the normal number of melanocytes or an increase in the number of melanocytes.
- Etiology– Excessive exposure to UV light(esp., sunlight)

  Genetic illness in some families

  Chronic renal failure

  Chronic pruritis
- They may also suffer from mental depression, social stigma

Effects of Atisthoulya (Morbid obesity)

- It can be defined as an excess of adipose tissue that imparts health risk; a body weight of 20% excess over ideal body weight for age, sex and height is considered a health risk.

- Can be calculated by BMI=kg/m2
  The body mass index (BMI), or Quetelet index, is a measure for human body shape based on an individual's mass and height. In adults, damaging effects of excess weight are seen when the body mass index exceeds 25 kg/m2.
- Obesity is defined as having a BMI of >30 kg/m2

Causes of Obesity are as Follows:

- Causing over eating.
- Inactivity and sedentary life style.
- Diets largely derived from carbohydrates and fats than protein rich diet.
- Genetic predisposition.

Consequences of Obesity

- Hypertension
- Atherosclerosis
- Coronary Artery Disease and Stroke
- Cholelithiasis
- Osteoarthritis
- Fatty liver
- Diabetes mellitus

Effects of Ati Krusha

- Poor physical strature, poor strength and endurence
- Poor or under nourished and under weight
- Poor physical stamina, some times poor immunity
  Early exhaustion and poor life expectancy.

10. Definition and classification of Vyadhikshamatva

Definition

व्यधिक्षमत्वं नाम व्यधिबलविरोधित्वं व्यद्युत्पादकप्रतिबन्धकत्वमिति। (च.सु. २६/८१ (चक्रपाणि)

It is the power of the body which prevents the development of disease or resists the onset of developed disease is called as vyadhikshamatwa. This resistive power of disease by the body differs from person to person.

It depends upon following factors :

1. Bala : The natural inherent strength, power of the body which is responsible for health.

बलाधिष्ठानमारोग्यम् ..... (च.चि. ३/१४१)

Bala depends upon the healthy status of dhatus. The factors which influence bala are:

बलबुद्दिकरस्त्वमे भावा भवन्ति/तथ्यथा बलवत्पुरुषो देश जन्म, बलवत्पुरुषो काल च सुखच्च कालयोगः बीजक्षेत्रगुनसम्पच्च आहरसम्पच्च शरीरसम्पच्च सत्म्यसम्पच्च सत्वसम्पच्च स्वभवसम्सिद्दिस्च यौवनं च, कर्म च, सहर्षस्चेति।

2. **Prakruti :** The primary constitution of an individual. Vata prakruti person is said to be sada atura due to poor vyadhi kshamatwa, kapha prakruti with good bala and least number of disease.
3. **Desha :** People who dwell in jangala desha are said to be suffering from less disease
4. **Kala :** Visarga kala and yuva avastha is said to have maximum vyadhi kshamatwa.
5. **Ojus:** Dalhana opines that the abyantara bala is derived from oja. As bala is derived from excellency of healthy dhatus ojus also contributes bala and finally enhances general health and also vyadhikshamatwa.

अत्राभ्यन्तरप्राणो बलं तच्चौजोजनितम्। (सु.सू. १७/१३)

Classification

त्रिविधं बलमिति - सहजं कालजं युक्तिकृतं च।

(a) Sahaja : **सहजं वत् शरीरसत्वयोः**

It is the inherent bala attained due to excellence of components of shareera. It is formed from the time of formation of garbha based on excellence of shukra and arthava.

(b) Kaalaja : **प्राकृतं कालकृतमृतुविभागजं वयः कृतं च।**

The strength attained by the individual due to the impact of seasonal variation and aging phenomenon is kaalaja bala which is temporary. The best bala is seen during visargakaala and youvanaavastha and bala deteriorates naturally during adaanakaala and vruddavastha. The age of person बाल, मध्यम, वृद्ध and diurnal changes infuence dosa and that will again influence बल and Health.

(c) Yukti kruta : **युक्तिकृतं पुनस्तद्याहारचेष्टायोगजं।**

It is an acquired bala gained by planned implimentation of combination of diet, medication and other regiemen by the patient as planned by the physician.

Methods of Improving Vyadhi Kshamatwa

- Garbadaana poorvaka shodana to couple
- Maasaanumaasika garbhini paricharya by mother
- Shodasha sishu samskaara
- Nitya shadrasopeta aahara
- Nitya sevana of shaali, mudga, maasha, maamsarasa, draksha, amalaki, pippali, saindhava, grita, ksheera, dadhi, sura etc
- Niyamita vyayama, non supression of adharaneeya vegas,
- Following dinacharya, ritucharya, sadvritta
- Avoiding janapadodwamsa & paryavaranapradoshana
- Periodic shodhana and Nitya rasayana.

11. Ojas- types of Ojo Visrimas- Vyapad & Kshaya & It's Disease

Introduction

The excellence of all dhatu or the best of all body constituents are segregated to produce a unique element called ojus. The word 'ओज' is said to arise from "ऊर्जतेवाउब्जतेवाइति।" can be said as the one which provides bala.

"ऊर्जबलप्राणयोः"

It means as the one responsible for 'बल' & 'प्राण'

Utpatti of Ojus

- तत्ररसादीनांशुक्रान्तानांधातुनांयत् परंतेजस्तत्खल्वोजस्तेवबलमित्युच्यतेस्वशास्त्रसिद्धान्तात्।। (सु.सू. १५/१९)

Ojus is the pure essence of dhatus from rasa to shukra which is said to be the 'BALA'.

- ओजस्तुतेजोधातुनांशुक्रतानांपरस्मृतम्।। (अ.ह.सू. ११/१७)
- Ojus is the pure essence of all the seven dhatus.
- सर्वधातुनां स्नेहमोजः क्षीरेघृतमिव तदेव बलमिति।। (भा.प्र. ३/१८१)

Like ghrita is sneha of milk, in the same way ojus is of all the seven dhatus and the same ojus is the 'BALA' of the body.

- यथा कृत्स्न क्षीर स्नेहाः तथैव ओजो अपि कृत्स्न धातुस्नेहः इत्यर्थः।। (डल्हण on सु.सू. १५/१९)

Ojus is that entity which is present in dhatus just like the sneha is present in milk.

Nourishment of Ojus

- प्राणिनां पुनर्मूलं आहार बेल वर्णौजसाम्।। (सु.सू. १/२८)

i.e. The food is the principle factor which nourishes strength, varna & OJUS.

- यदन्ने देह धात्वोजो बल वर्णादिपोषकम्। (च.चि. १५/५)

Food provides nourishment to dehadhatus, OJUS, strength & complexion.

Gunas of Ojus

- हृदितिष्ठति यत् शुद्धरक्तमीषत् पीतकम्। ओजःशरीरेसंख्यातं। (च.सू. १७/७४)

The one which dwells in the heart and is pure (shudha), slightly reddish yellow in colour is known as OJUS of the body.

- सर्पिवर्णंमधुरसंलाजगन्धिप्रजायते। (च.सू. १७/७५)

The ojus in the body is of the colour of Ghruta, in taste it is like that of honey and in smell it is like that of laja.

- गुरुशीतंमदुश्लक्ष्णंबलंमधुरंस्थिरम्। प्रसन्नंपिच्छिलंस्निग्धमोजोदशगुण स्मृतम्।। (च.चि.२४/३१)

The ten attributes of ojus are Guru (heaviness), seeta (cold), mrudu (soft), shlakshana (smoothness), bahala (dense), madhura (sweet), sthira (stable), prasanna (clear), pichilla (slimy) & snigdha (unctious).

Importance of Ojus

- दशैव आयतनान्याहुःप्राणाःयेषुप्रतिष्ठिताः।शंखमर्मत्रयंकण्ठोरक्तंशुक्रंओजसीगुदम्।।(सु.सू.१५/२१)

There are ten life sustaining vital components viz. two shankha, trimarmas, hrudaya, shira, basti, kantha, rakta, shukra, OJUS & guda.

- स्निग्धं सोमात्मकं शुद्धमीष्ल्लोहितपीतकम्। (अ.ह.सू. ११/३८)

Ojus is snigdha, shita, shudha (pure) & appears to be slightly reddish yellow in colour.

ओजःसोमात्मकं स्निग्धं शुक्लं शीतं स्थिरं सरम्। विविक्तं मृदु मृत्स्नं च प्राणायतनमुत्तमम्।। (सु.सू. १५/२१)

Ojus is somatmakam (cooling substance), snigdha, shukla,

seeta, sthira, sara (mobile), vivikta (pure), mrudu, mrutsanna (pichila or slimy) and is the site of vitality.

देहः सावयवस्तेनव्याप्तोभवतिदेहिनाम्। तद्भावाच्चशीर्यन्तेशरीराणिशरीरिणाम्।। (सु.सू.१५/२२)

The whole body is permeated with ojus and loss or diminution leads to wasting, decay & destruction.

Classification of Ojus

- द्विविधमोजो दर्श्यति परम् अपरंच। (चक्र. on च.सू. ३०/७)

There are two types of ojus:

1. Para Ojus 2. Apara Ojus

Quantity and Location of Ojus

1. Para Ojus

- प्राणाश्रयस्यौजसो ऽष्टौबिन्दवो हृदयाश्रिता। (चक्र. on च.सू. ३०/७)

Even minute kshaya of which can cause death , which is in the ashtabindu pramana and is located in the hrudaya is the Para Ojus.

2. Apara Ojus

- तत्र अर्धाञ्जलि प्रमाणमपरं यदुक्तं-"तावदेव परिमाणं श्लैष्मिकस्यौजसः"

(चक्र on च.सू. ३०/७)

Apara Ojus is in the ardhanjali pramana.

- तत्र अर्धाञ्जलि प्रमाणमपरं यदुक्तं–"तावदेव परिमाणं श्लैष्मिकस्यौजसः"।

(चक्र. on च.सू. ३०/७)

The ardhanjali pramana Ojus or the Apara Ojus is located in the heart and the vessels attached to it.

Functions of Ojus

- ओजस्तुतेजोधातुनांशुक्रान्तानांपरंस्मृतम्। हृदयस्थमपि व्यापिदेहस्थितिनिबन्धनम्।।

(अ.हृ. ११/३,७)

The essence of all the dhatus ending in shukra dhatu is termed as Ojus. Though it is located inhrudaya it spreads all over the body & maintains it.

Ojo kshaya Hetu

In AshtangaSamgraha the etiological factors are mentioned:

- क्रोधः क्षीयतेकोपक्षुद्व्यायामशोक श्रमादिभिः। (अ.सं.सू. १९/३८)

Anger, hunger, worry, grief, over exertion causes Ojo Kshaya.

According to Acharya Charaka:

- वातश्लेष्मक्षयेपित्तंदेहौजः स्रंसयच्चरेत्। ग्लानिमिन्द्रियदौर्बल्यंतृष्णांमूर्च्छांक्रिया क्षयम्।।

(च.सू. १७/६०)

When vata &kapha are in a state of diminution, the pitta while eliminating ojus causes depression, weakness of senses, thirst, fainting & loss of action.

Ojo Kshaya Lakshana

According toAshtanga samgraha:

- विभेति दुर्बलो ऽभीक्षणं ध्यायति व्यतितेन्द्रियः। दुश्छायोदुर्मनारुक्षोभवेत्क्षामश्चतत्क्षये।।

(अ.सं.सू. १९/३८-३९)

1) Fear
2) Constant weakness
3) Worry
4) Affliction or discomfort of sense organs
5) Loss of lustre
6) Cheerlessness
7) Roughness
8) Emaciation

Ojo Visramsa Lakshana

- सन्धिविश्लेषो गात्राणां सदनं दोष च्यवनं क्रिया सन्निरोधश्च विस्रंसे। (सु.सू. १५/२४)

Dislocation of joints, feeling tired, displacement of dosha's from its own sthana, restriction for shareerika manasika & vachika kriya's.

Ojo Vyapat

- व्यापत् अन्यथापत्तिः सा दुष्ट दोष दूष्य संसर्गात्। (Dalhana on सू.सू. १५/२४)

Vyapat means ojus gets vitiated by dushta dosha & dushya.

Lakshanas of Vyapat:

- स्तब्ध गुरुगात्रता वाताशोफो वर्णभेदो ग्लानिस्तन्द्रा निद्रा च व्यापन्ने। (सू.सू. १५/२४)

1) Stiffness & heaviness in body
2) Swelling due to vata
3) Discolouration or loss of complexion
4) Exhaustion

Summary of the Functions of Ojus:

- At the time of conception, it is the essence of shukra & shonita.
- In the second stage, i.e. the kalala avastha it is the essence of the rasa.
- In the third stage when there is formation of various organs ojus is present in its own form & manifests its own actions.

Desa Saatmya

Desa means place of residence of asn individual

1. Deha (Body) 2. Bhumi (Land)

1. **Deha satmya:** Foods that are conducive to particular body is Deha satyma.
   Example- Ghee, milk and meat are satmya to some people.
   Rice, peya, yusa and wheat are satmya for other people.
2. **Bhumi satmya:** Foods that are conducive to particular land is Bhumi satyma.
   Example- Jangle desha- Ghee, milk

Roga Satmya

Foods that are conducive to particular disease is roga satyma.

Example- Milk is satmya in tumors, Honey is satmya in urinary disorders.

Oka Satmya

The continuous use of even asatmya will not cause disease due to habituation and this is known as oka satmya. Example-curd in night, ice creams in winter season.

Rutu Satyma

Food that are conducive to particular season is rtu satyma.

Example- Graisma rtu- Food and drinks should be liquid, sweet, cold.

Varsa rtu- old rice, wheat , Barley; Vasanta rtu- wheat, barley, Rice, yava

Importance in Roganidana

- It is the trial and error method for planning different therapeutic procedures.
- Upasaya stage signifies the curability or incurability of the disease based on observation of 18 subtype of upasaya.
- Knowledge of satmya helps to prevent disease and also to control disease
- Effect on body: Satmya always make good effect on the body in normal equilibrium stage of the dosha dhatu and mala, and helps to cure the disease in the abnormal stage.

Asaatmya Definition

विपरीतोऽनुपशयोव्याध्यस्वात्म्याभिसंज्ञितम्। (अ.ह.नि. १/७)

Opposite to the description of there upasaya is called anupasaya, which is not conductive to the body.

विपरीतोऽनुपशयइतिऔषधादीनांदुःखकरउपयोगोऽनुपशयइत्यर्थः। (मा.नि. १/९)

Medicines, food and regimen which are not conductive to the body and develop displeasure to the body is called Anupasaya. It is also called Asatmya.

Synonyms: Asatmya, Apathya, Dukha, Ahita, Viruddha.

Effect on Body: Asatmya make bad effect on the body. It causes bad effect on the dosha dhatu and mala, and causes diseases.

Difference Between Satmya and Asatmya

	Satmya	Asatmya
Desa	Simillar	Dissimillar
Kala	Simillar	Dissimillar
Roga	Good	Bad

Conclusion

Satmya has to be considered as-

- Treatment (chikitsa) and upashaya in diseased person.
- Wholesome for healthy individual.

Asatmya is the opposite of satmya.

●

Chapter- 3

BASIC PATHOLOGY

1. Introduction to Pathology and its Sub-Divisions

It is derived from the word pathos which means suffering and logos means study. Thus pathology is scientific study of structure and function of body in the diseased. It consists of the abnormalities that occur in normal anatomy including histology and physiology owing to disease.

Pathophysiology. Study of disordered function or breakdown of homeostasis in diseased. Pathology explains Etiology (why) pathogenesis (how) and functional implications of the lesion felt by the patients (symptoms) and those discovered by the physician (through physical signs). All these will help a clinician to arrive at a particular diagnosis, prediction of future (prognosis) and the probable treatment. Hence the method of prevention and avoiding its spread and complications.

History of Pathology

The earliest concept of disease understood by the patient and the healer was the religious belief that disease was the outcome of "curse from god"or the belief in magic that the affliction had supernatural origin from"evil eye" of spirits that leads to disease. The link between medicine and religion became so firmly established throughout the world that different socities had their gods and goddess of healing.

For example-Mythological Greeks had "APOLLO" as the principal God of healing.

Dhanwantri as the diety of medicine in India and orthodox Indians belief in Mata Sheetala Devi as the Goddess.

Pre historic period. The period of ancient religious and magical belifs was followed by philosophical and rational approach to disease by observation. Greek philosopher Socretes, Plato and Aristotle introduced philosophical concept for all. Real practice of medicine

began after Hippocrates a clinical genius of 460 - 370 BC. His postulations and oath has created revolutionary landmark in history of medicine. He explained how disequilibrium of 4 humors water, air, fire and earth can cause disease. He explained doctrines of case taking known as Hippocrates aphorisms. They are-

- Observe all objectively
- Study the patient rather than disease
- Evaluate honestly
- Assist nature.

- Cornelius celsus (53 BC) first described signs of inflammation
- Cladius Galen (130-200AD) explained humoral theory as 4 humours which include Blood, Lymph, Black bile, and bile of Liver. Disequilibrium of these cause disease.

From Human Anatomy to era of gross pathology

- Italian painter Leonardo Da Vinci (1452-1519) explained human anatomy.
- Dissection of human body by Vesalius (1514) on executed criminals.
- Antony Van Leuwen Hook (1032-1723) invented microscope and observed male spermatozoa, blood corpuscles, introduced histological staining in 1714 using saffron to examine muscle fiber.
- Marcello Malpighi (1624-1694) used microscope extensively and observed the presence of capillaries, layers of skin, lymphoid tissues in spleen.
- Giovanni B. Morgagni (1682-1771) explained morbid anatomy, clinic pathological methods in study of disease. He introduced the concept of clinic pathologic co relation.
- Sir Percival pott (1714-1788) observed occupational cancer in chimney sweapers.
- William hunter developed first pathology museum.
- Richard Bright (1789-1858) explained nonsuppurative glomerulonephritis which is today known as Bright's disease
- Thomas Addision explained chronic adrenocortical insufficiency termed as Addison's disease.

- Thomas Hodgkin (1798-1886) explained chronic enlargement of lymphnodes often with enlargement of liver and spleen termed as odgkin's disease
- Xavier Bichat (1771-1802) explained general and systemic pathology.
- RT Laennec (1781-1826) French physician described several lung disease like tuberculosis,caseous lesions, military lesions, pleural effusion, bronchiectasis. Also explained chronic sclerotic liver disease. He invented stethoscope

- **Era of technology development and cellular pathology**

Pathology started developing as a diagnostic discipline, in later half period the evolution of cellular pathology which has closely linked to technology advancements in machinery manufacture for cutting thin section of tissue improvement in microscopic and development of chemical industry and dyes for staining.

Rudolf Virchow in Germany is credited with the beginning of microscopic examination of diseased tissue at cellular level and thus began Histopathology as a method of investigation.

Classification of Pathology

Traditionally - (1) General pathology
(2) Systemic pathology

Some subbranches of pathology

1. Histo pathology- It is the classical method of study and includes structural changes observed by naked eyes examination referred to as gross or macroscopic changes.

Three sub division of histo pathology

- Surgical pathology- It is the study of tissues removed from the living body.
- Forensic pathology and autopsy work- It is the study of organ and tissues removed at post-mortem for medicolegal work for determining the underlying sequence and cause of death.
- Cytopathology- It is the study of cells shed off from the lesions and fine needle aspiration cytology of superficial and deep seated lesions for diagnosis.

2. Haemtology- It deals with diseases of blood and management of patient.
   - Laboratory haematology
   - Clinical haematology
3. Chemical pathology- It deals with the analysis of biochemical constituents of blood, urine, semen, csf. and other body fluids.
4. Immunology- It deals with detection of abnormalities in the immune system of the body comprises immunology and immunopathology.
5. Experimental pathology- It deals with production of disease in the experimental animal and its study.
6. Geographic pathology- The study of differences in distribution of frequency and type of disease in population in different part of the world.
7. Medical genetics- It is a branch of human genetics that deals with the relationship between heredity and disease important development in medical genetics.
8. Molecular pathology- The detection and diagnosis of abnormalities at the level of DNA of the cell.

Systemic pathology is deseribed beased in various Systems in the body.

(a) Cardiae System Pathology : Disease of Heart and blood vessels.
(b) Respiratory System Pathology: Disease of Lungs, Nose nasopharynx, throat, Bronehial tree.
(c) Nervous System Pathology: Diseases of Brain and Nerves.
(d) Gastro Intestine System: Disease of Ocsophagus, stomach, Hepatobiliary System, Intestines.
(e) Nephrology : Diseases of Kidneys.
(f) Urogenital System: Diseases of kidney, Ureters, bladder and genital and reproductive Organs.
(g) Gyenecology: Diseases of female reproductive organs.
(h) Musculoskeletal System: Disease of muscles, bones, connective tissues, tendons, ligaments.
(i) Dermatology: disease of skin, hair, nails.
(j) Onehology: The study of fumour or Neoplasia.

2. Introduction to Cell Injury and Cellular adapations

Introduction to Cell

Cell is the basic structural and functional unit of body. Cells are basic units of tissues, which form organs and further systems in the body.

Body cell

Epithelial cell ⟷ Mesenchymal cell

Cell Structure

Cell is enclosed by a cell membrane that encloses nucleus and various subcellular organelles within the cytosol/cytoplasm.

The different cell structures are,

- Cell membrane/plasma membrane
- Nucleus with nuclear membrane enclosing nuclear chromatin and nucleolus
- Cytosol /cytoplasm
- Mitochondria
- Endoplasmic reticulum
- Lysosymes
- Cytoskeleton
- Ribosomes
- Golgi apparatus
- Centriole/centrosome

Injury

Definition

An injury can be defined as a damage inflicted on the body by an external force. It can also be explained as an accident that results in physical damage or hurt.

Synonyms

- Harm
- Hurt
- Trauma
- Wound etc.

Classification of Cell Injury

- By cause
- By location
- By activity

By Cause

- Traumatic injury.
- Injury due to radiation, burn or any other external physical cause.
- Injury from infection.
- Injury due to cancer.
- Injury secondary to other diseases.

By Location

- Sharp injury damaging dermis or skin
- Nerve injury
- Soft tissue injury
- Brain injury
- Spinal cord injury

By Activity

- Sports injury
- Occupational injury

Cell Injury

Definition

- A variety of stress a cell encounters as a result due to changes in its internal as well as external environment.
- In 1859, Virchow first established the cellular theory of disease. In his concept the disease occurs due to the abnormalities at the level of cell.
- Most forms of disease begins with cell injury followed by consequent loss of cellular function

Various cellular responses to cell injury

1. Cellular adaptations
2. Reversible cell injury
3. Irreversible cell injury
4. Sub- cellular changes
5. Intercellular accumulations

Cellular Adaptations

When there is an increased functional demand, the cell may adapt to the changes which are expressed morphologically and revert back to normal after the stress is removed.

Reversible cell injury: When the stress is mild to moderate, the injured cell recovers when stress is removed.

Irreversible cell injury: Persistent cell injury which results in cell death.

Sub- cellular changes: Persistence of the residual effect of reversible cell injury in a cell, as an evidence of cell injury at the sub- cellular level.

Intercellular accumulations: Accumulation of metabolites in the cell.

Pathogenesis

Pathogenesis of Reversible (eg)

1. Low oxygen supply

↓

Aerobic respiration of mitochondria fails

↓

Glycolytic pathway-depletion of glucose & accumulation of lactic acid

↓

Decreases intracellular Ph

↓

Clubbing of nuclear chromatin
2. Reduced protein synthesis

↓

Continued hypoxia

↓

Membrane of ER & golgi apparatus swells

↓

Attachment of ribosome from RER

↓

Ribosomes in the cytoplasm inactivates its functions

Pathogenesis of Irreversible- (eg)

- In sustained ischemia→ excess Ca influx
  activation of phospholipases→ membrane degradation.
- Damage of lysosymal membrane→ release of lysosymal enzymes→ enzymatic digestion of cellular components cell death.

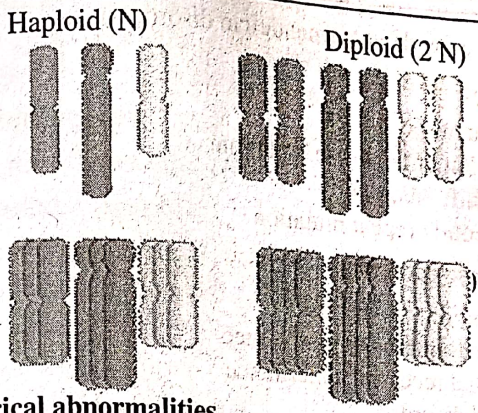
Etiology of Cell Injury

Cells may be broadly injured by two major ways,

- By genetic cause
- By acquired cause

Genetic Cause

1. Developmental defects- Group of abnormalities during fetal life, due to error in morphogenesis, due to some chemicals, drugs, physical agents (teratogens).
   Eg: cleft lip.
2. Cytogenic (karyotypic) defects- Chromosomal abnormalities



- **Numerical abnormalities**

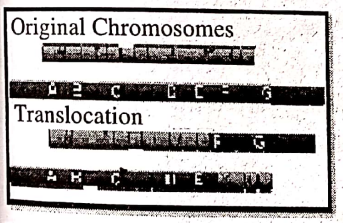
Polyploidy-number of chromosome is multiple of haploid ie 3N-trploid-63 chromosomes & 4N-tetraploid-92 chromosomes, which occurs in megakaryocytes & liver cells.

Aneuploidy- number of chromosome is not an exact multiple of haploid number.

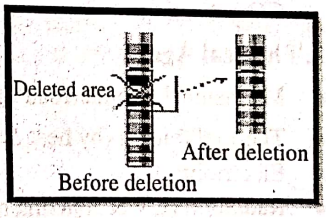
Eg: Down's syndrome-trisomy of 21st chromosome
Klinefelter's syndrome-trisomy of sex chromosome
Turners syndrome-monosomy (45,x0)

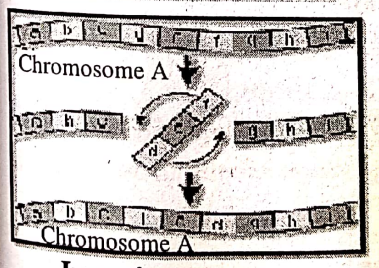
- Structural abnormalities

Translocation

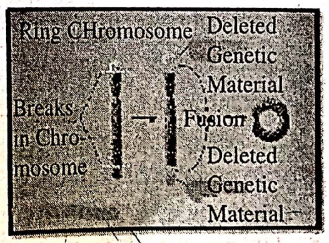


Deletion





Inversion



Ring formation

3. Single gene defects (mendelian disorders)

- Mutations
  - ✓ Point mutation
  - ✓ Stop codon or nonsense mutation
  - ✓ Frame shift mutation
  - ✓ Trinucleotide repeat mutation
- Inheritance pattern
  - ✓ Dominant/recessive gene
  - ✓ Autosomal dominant inheritance
  - ✓ Autosomal recessive inheritance
  - ✓ X-linked disorders

4. Multifactorial inheritance - due to combined effect of genetic composition & environmental factors.

   Eg: type two diabetic mellitus

Aquired Cause

1. Hypoxia

Cells require oxygen to generate energy and perform metabolic functions. The deficiency of oxygen is hypoxia. It occurs due to-

- Ischemia - reduced blood supply to blood cells due to interruption
- Disorders of oxygen carrying capacity of RBC's due to anemia, CO poisoning.

2. Physical Agents

- Mechanical trauma (road accidents)
- Thermal trauma (by heat & cold)
- Electricity
- Radiation (UV & ionizing rays)
- Rapid changes in atmospheric pressure

3. Chemicals & Drugs

- Chemical poisons such as cyanide, arsenic, Hg etc.
- Strong acid & alkalies
- Environmental pollutants
- Insecticides & pesticides
- Oxygen at high concentration

- Hypertonic glucose and salts
- Social agents like alcohol & narcotic drugs
- Therapeutic administration of drugs

4. Microbial agents

Infections by bacteria, virus, fungi, protozoa & other parasites.

5. Immunologic agents

Immunity protects host against various injurious agents but, in some cases it may turn lethal & cause cell injury.

Eg: hypersensitivity reactions, anaphylactic reactions, autoimmune diseases.

6. Nutritional derangements

Deficiency /excess

Eg: rickets, beri beri etc

7. Aging

Cellular aging (impaired ability of cell to undergo replication & repair which leads to cell death.

8. Psychogenic diseases

Due to drug addiction, alcoholism and smoking resulting in various organic diseases.

9. Iatrogenic causes

Owing to physician ie death or diseases due to wrong diagnosis by physician and bad effects of administered therapy.

10. Idiopathic diseases

Of unknown cause
Eg: hypomelanosis

3. Definition and brief description of inflammation- Healing/repair

The word meaning of inflammation is burning. The process inflammation is defined as local response of living mammalian tissue to injury due to any agent. It is a body's defense reaction in order to eliminate or limit the spread of injurious agent followed by removal of necrosed cell and tissue.

Causitiva agent: Infective agents, immunological agents (cell mediated, antigen-antibody), physical agent, inert material (foreign body) which can multiply, release toxin and are antigenic.

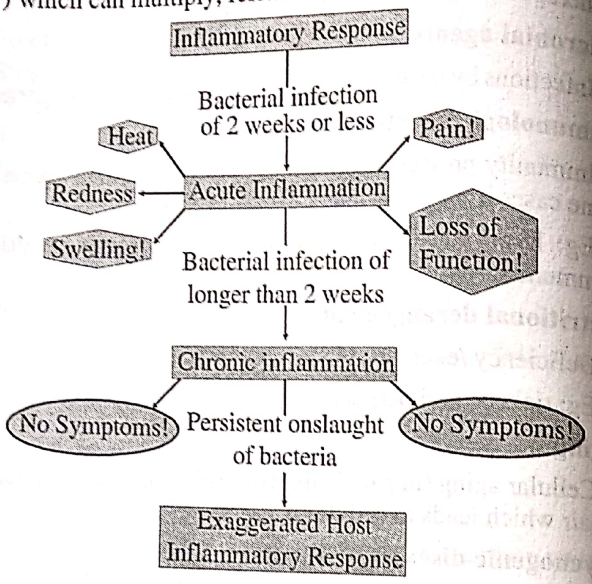
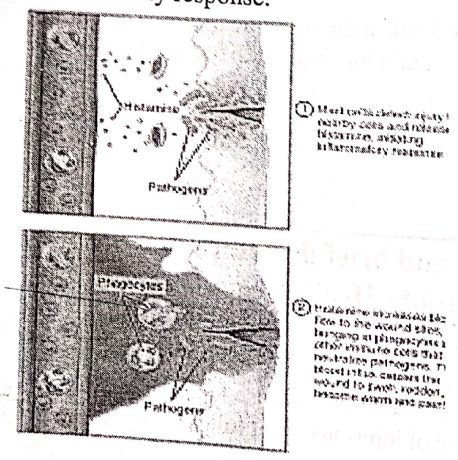
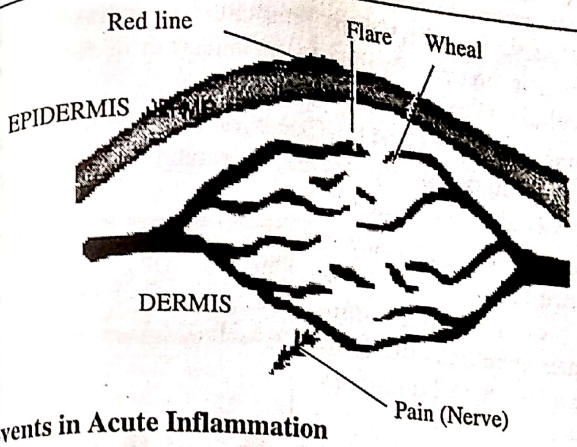


Figure 8.14 Two stages of Inflammation, Acute Inflammation is of short duration, wheres chronic inflammation is a long-lived inflammatory response.





Events in Acute Inflammation

Inflammation is the visible response to an immune reaction and activation of immune response is essential before inflammatory response appears.

Signs: Rubor (redness), tumor (swelling), calor (pain), dolar (fever or rise in temperature), functiono liesia (loss of function pertained to inflamed area).

Acute inflammatory feature: its features are

1. Accumulation of fluid and plasma at the affected site
2. Intravascular activation of platelets.
3. Polymorphonuclear neutrophils form as inflammatory cells

Chronic inflammation: shows presence of chronic inflammatory cells such as lymphocytes, plasma cells, macrophages and granulation tissue formation.

Events in acute inflammation:

1. Vascular Events

A. alteration in microvasculature (arterioles, capillaries, venules) hemodynamic changes like transient vasoconstriction of arterioles lasting for 5 sec - 5min.

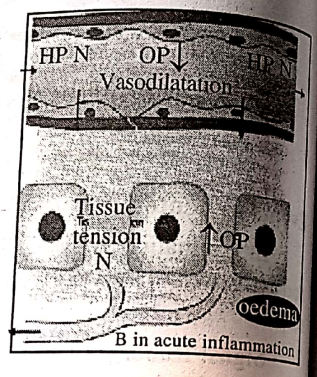
B. Persistent vasodilatation: Within half an hour after injury vasodilatation occurs. This results in increased blood volume leading to redness and warmth at the site.

C. Progressive vasodilatation. This caused increased local hydrostatic pressure leading to transudation of fluid from intracellular to extra cellular space.

D. Slowing/stasis: The blood flow becomes slow so as to facilitate lucocytic migration or peripheral orientation of leucocytes along vascular endothelium.

E. Altered vascular permeability: in inflamed tissue the endothelial lining of microvasculature becomes more leaky that leads to exudative inflammatory oedema

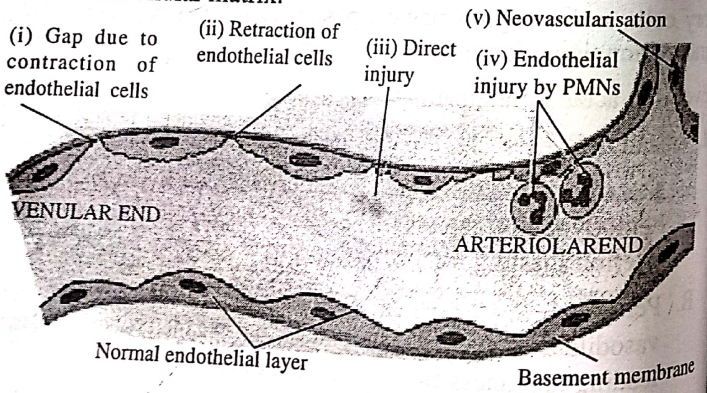
F. Neo vascularisation and leakage from new blood vessel

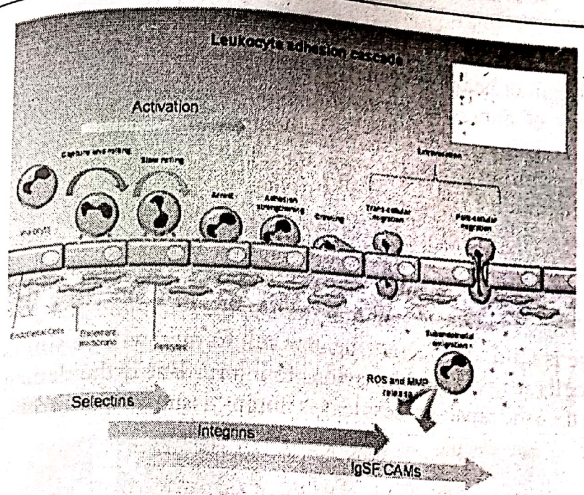


2. Cellular Events

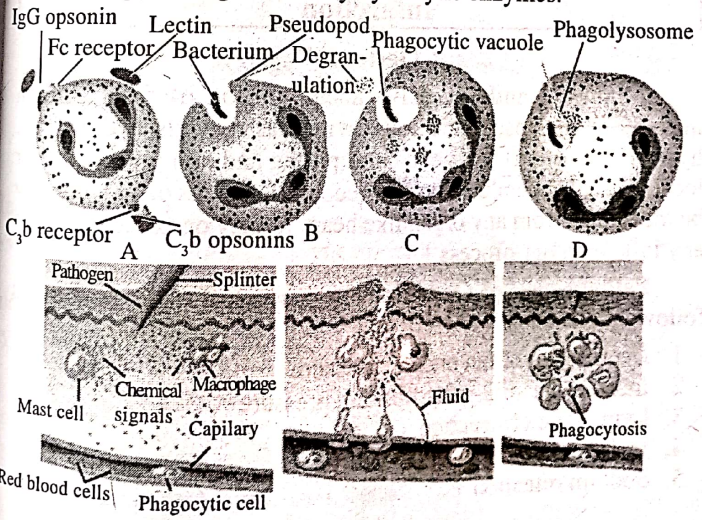
A. Exudation of leucocytes: escape of WBC from lumen of microvasculature to interstitial tissue. This includes Migration, Pavementing, Rolling and Adhesion, Emigration, Chemotaxis

B. Phagocytosis: It is the process of engulfing of solid polymorpho nuclear cells(microphages) and circulatory monocytes, macrophages. They produce protieolytic enzymes like lysosomes, protease, gelatinase, lipase, protiease, elastinase, collagenease and acid hydrolaeses. They degrade collagen and extracellular matrix.



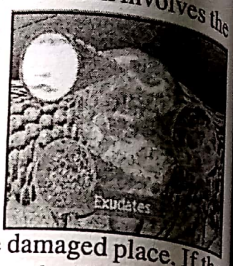


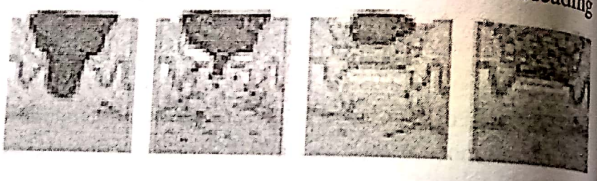
1. The process involves : Recognition and attachment with help of mannose receptors and scavenger receptor, IgG opsonin, lectins.
2. Engulfment with the help of Cytoplasmic pseudopodes due to activation of filaments beneath the cell wall.
3. Killing and degradation by hydrolytic enzymes.



Repair and Healing

Repair or healing is a natural phenomenon which involves the recovery of damaged structure occurred during cell injury. Once the inflammatory response finishes by phagocytosis the repair or regeneration of new cell and tissues in the place of damaged area takes place. Depending on the extend of cell injury the new cells or replacement of new cells takes place. The new cell proliferation starts in the damaged place. If the damage is mild to moderate the new cell formed will be similar to those cells which existed previous to injury. But if the damage is severe then the repair process leaves fibrous tissue formation leading to scar.





4. Definition and brief description of edema-shock-hemorrhage, Thrombosis, embolism, Ischema and Infarction

"Oedema"

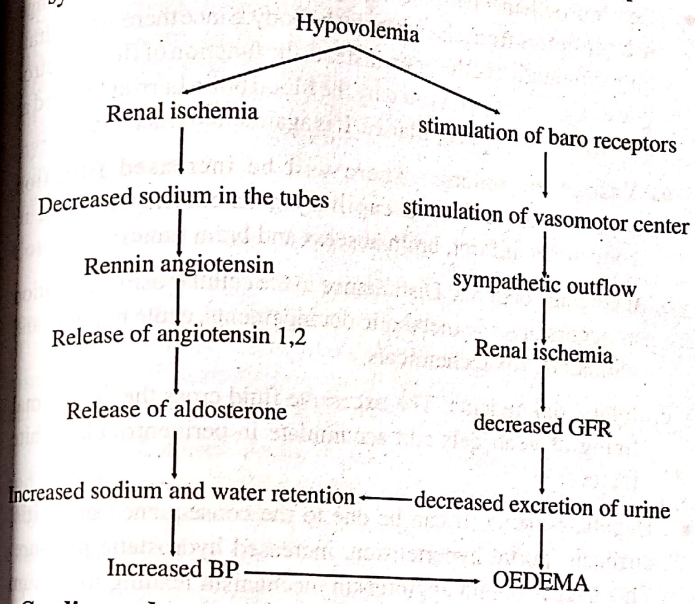
Abnormal and excessive accumulation of free fluid in interstitial tissue space and serous cavities (as in ascitis, hydrothorax, hydro pericardium). This can be of pitting and non pitting type or localized and generalized type of oedema. Contents of oedema may be transudate from any organ like heart, kidney or excudate due to any inflammatory process.

Pathogenesis of Oedema: The process of oedema requires following elements

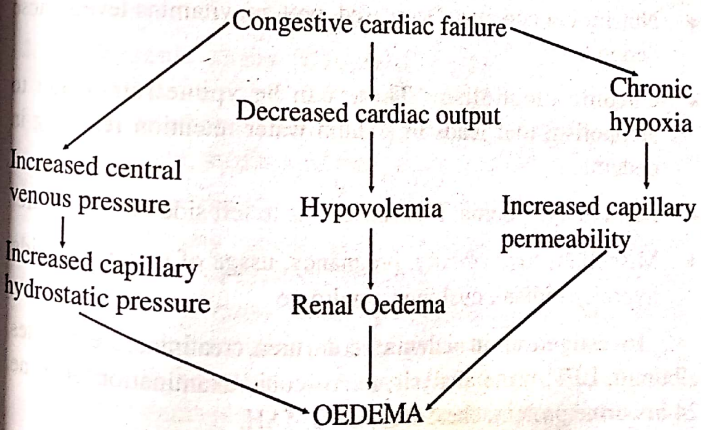
1. Decreased plasma oncotic pressure
2. Increased capillary hydrostatic pressure
3. Lymphatic obstruction
4. Increased capillary permeability
5. Sodium retention

Types of Oedema with Pathogenesis

- Renal oedema: Caused due to glomerulo nephritis, nephritic symdrome, renal failure.



- **Cardiac oedema**



Caused due to congestive cardiac failure and may also progress as renal oedema.

- Cerebral oedema: It the most dangerous oedema as fluid exchange in brain defers from elsewhere in the body. Since there are no draining lymphatics in the brain instead, the function of fluid electrolyte exchange is performed by the blood brain barrier located at endothelial cells of capillaries. It is again subdivided into:

a. Vasogenic oedema: There will be increased filtration pressure or increased capillary permeability as seen in contusions, infarct, brain abscess and brain tumors.

b. Cytogenic oedema: Disturbance in the cellular osmoregulation as occurs in some metabolic derangements, acute hypoxia and with some toxic chemicals.

c. Interstitial oedema: The excessive fluid cross the ependymal lining of ventricals and accumulate in periventricular white matter.

- Hepatic oedema: It can be due to the consequence of ascitis, cirrhosis, portal hypertension, increased hydrostatic pressure. This trigger rennin angiotensin mechanism leading to sodium and water retention that results in oedema.
- Nutritional oedema: Decreased protein, vitamins levels cause oedema
- Chronic alcoholism: There can be yponetremia due to alcoholism that leads to sodium water retention resulting in oedema.
- Pulmonary oedema: It is causes due to left sided heart failure.
- Miscellaneous : obesity, pregnancy, usage of glucocorticoids, hypothyroidism, cushing's syndrome

Investigations in oedema: serum urea, creatinine, electrolytes, albumin, LFT, urine analysis, microscopic examination of urine, 24 hrs urine protein, chest xray, ECG, TSH.

SHOCK

A condition of Acute circulatory failure. It is a clinical syndrome characterized by prolonged hypotension leading to inadequate tissue perfusion. It is a whole body event of complex series of changes which result from acute circulatory failure. All forms of shock result in reduction in effective blood flow (hypoperfusion /under perfusion) of vital organs so that there is reduced delivary of oxygen and nutrients to the tissue and subsequent cell dysfunction.

Compensatory Changes After Shock

Body is generally concerned with maintainance of adequate cerebral and coronary circulation. They are affected by redistribution of the blood in the body as whole by autoregulatory mechanisms.there will be peripheral vaso constriction and selective regional vaso constriction. There will be deviation of blood to vital organs

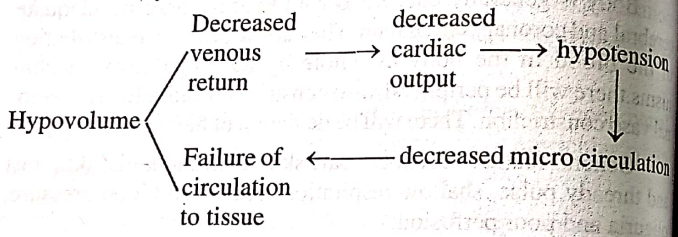
General features of shock: pale skin, cold and moist skin, fast and thready pulse, shallow respiration, decreased blood pressure, oliguria and poor perfusion.

Classification of Shock

1. Hypovolemic shock: Hypovolemia is total decrease in blood or fluid volume. It may be caused due to severe hemorrhage, fluid lossdue to vomiting and diarrhoea burns.
2. Cardiogenic shock: Decreased cardiac out put due to myopathy, myocardial infarction, pulmonary embolism, cardiac arrythemia, mitral regurgitation, cardiac tamponade, pump failure, mediastinal disease.
3. Anaphylactic shock/Neurogenic sock: Due to hypersensitivity reaction on any element manifests as shock. It may due to degranulation of mast cell and release of vaso dialators like histamine, elements causing hypotension as bradykinin contribute in circulatory failure leading to shock. Death occur due to broncho spasm or laryngeal oedema. Neurogenic shock is due to damage to spinal cord with peripheral vasodilation and pooling of blood.

4. Septic shock: Presence of sepsis in the body at any site due to organisms like E coli, pseudomonas, klebshella, strepto coccus aureus will invade into blood stream. These organisms release endogeneous mediators, plasma cells, macrophages, endothelial cells. All these initiate myocardial depressant factors causing organ hypoperfusion. This leads to multi organ failure resulting in acute respiratory distress syndrome. There can be necrosis of liver, pancreas and also haemorrhage or erosion and ulceration of GIT. Associated with ischemia of bowel and hypoperfusion of brain.

Summary of consequences of shock:



HAEMORRHAGE

It is the condition in which there is escape of whole blood. Bleeding may occur externally or into ollow vicera, serous cavity or tissue. The symptoms of hemorrhage depends upon quantity of blood loss and speed. Loss of blood over 30% within few hours is fatal. But loss about 50% of blood over 24 hrs and above is not necessarily fatal.

Early Changes of Hemorrhage: This occurs 48 hrs after hemorrhage. The condition is called as.

1. Vaso Vagal Syncope. There is slightly sweating, pallor, giddiness, decreased blood pressure and decreased pulse, ischemia, emotional stress, anxiety and fear. It is an alarming signal indicating that the safety margin is exceeded.

2. Cardiovascular Cange : Increased brerathing, tacchy cardia due to secretion of aderenalline, selective vasoconstriction to increase blood pressure.

3. Reaction of Blood: Increased platelet count, fibrinogen and ESR.

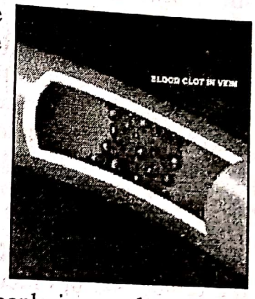
4. Restoration of Blood Volume : There will be withdrawl of fluid and electrolytes from interstitial compartment to plasma compartment. There is decrease in lymphatic flow, water is sequestrated in dense connective tissue is drawn upon. This can be associated with hemo dilution. As blood volume is restored vasoconstriction subcides and capillary pressure is brought back.

Late Changes (After 48hrs)

Regeneration of lost blood: Diluted blood and the resultant anorexia stimulates excess secretion of erythropoietin which stimulates marrow to erythropoietic activity. The hemoglobin production rate increases to 0.04-0.05 gram per day.

THROMBOSIS

Formation of solid plug from the constituents of blood anywhere in the intact cardiovascular tree during life is called as thrombosis.



Causes of Thrombosis

1. Changes in the endothelium: this may be due to physical trauma, burn, freeze, electric injury, chemical damage, bacterial toxins, inflammation of veins, atherosclerosis. Any neoplasia provokes platelet adhering and aggregation. This inturn initiates coagulation of blood and formation of fibrin
2. Alteration in the blood flow: slowing of the stream, turbulence and eddy currents, blood hyper viscosity state.
3. Alterations in the blood constituents

Patterns of Clot Formation

1. Common pattern : Platelet thrombus or white thrombus formed in moving blood stream which is progressive.
2. Uncommon pattern: Erythrocyte agglutination thrombus or hyaline thrombus formen only in capillaries as seen in mismatched transfusion, burns, frostbite and sickle cell disease. Its senn in slowed blood stream. It is followed with platelet coagulation thrombus.

3. Anatomical types:
   a. Occlusive thrombi: Artery or vein which obstruct the lumen of the vessel completely.
   b. Mural thrombi: This can be seen in Aorta or heart. There is no occlusion because of fast blood flow.
   c. Disseminated thrombi occur in micro vasculature.

Formation and growth of thrombus: Sticky endothelium damage invites platelet to adhere. Adherent platelets keeps on growing in the form of arborescent strands and fuse to form glossy clumps. Later it is accompanied by intrinsic thromboplast which clots the plasma trapped between the platelet clumps. The deposition of the fibrin strengthens the thrombus and anchors it to one side of the vessel wall. This small, flat, granular plaque is called as primary platelet thrombi.

Coralline Thrombus : The rough surface of developing platelet thrombus acts further stimulus for platelet adhesion in another fibrin layer and entrapped RBC. This forms platelet plug and then coagulation cascade is triggered. As the bold flow past the platelet thrombi red cells are trapped in mesh and thrombus grows in layers on the direction of blood flow producing the wavy elevated ridges of platelets called lines of Zahn.

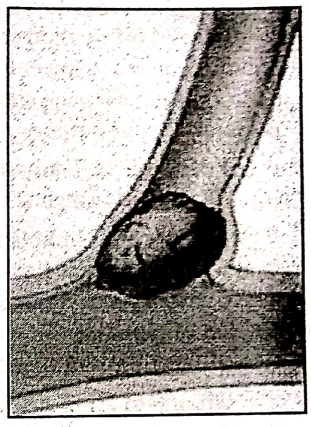
Occluding Thrombus: As the thrombus grows it encroaches the lumen and obstructs the blood stream. Once the vessel is completely occluded, the blood flow ceases and the stagnant column of blood clots without production of any lines of Zahn. This is consecutive clot which is adhered to vessel wall through original thrombus and likely can embolise.

Propagated clot is that which may continue to the point of entry of next tributary in which platelet may arrive and adhere to clot. There can be formation of single long consecutive clot due to stagnant of blood behind the clot which is anchored to the wall only by the initial platelet plug. Endothelium can initiate both thrombogenic and anti thrombogenic stimuli.

Fate of Thrombus:

1. Resolution : Small mural thrombi shrink by retraction and are reabsorbed after being slowly lysed by the fibrinolytic mechanism of blood and lytic enzymes from leucocytes incor-porated in their bodies. Large amount of enzymes, activators of plasminogen present in plasma. Te activated plasmin is proteolytic enzyme and fibrinolytic in action which can explode and destroy itself. Clots intended for temporary haemostasis are destroyed immediately after their utilization is over.
2. Organization : depending upon the size of thrombus ultimately converted into fibrous plaque or fibrous nodule which may show haemosiderin, cholesterol clefts and calcium salts.
3. Embolism : The clot may run along the circulation to other branches and can occlude elsewhere.

EMBOLISM



Embolism is the process consisting of the impaction in some part of the vascular system of any undissolved material brought there by blood current. The impacted abnormal mass is embolus (a plug).

The obstruction of a blood vessel by a foreign substance or a blood clot that travels through the bloodstream, lodging in a blood vessel, plugging the vessel. Foreign substances that can cause embolisms include air bubbles, amniotic fluid, globules of fat, clumps of bacteria, chemicals (such as talc), and drugs. Blood clots are the most common causes of embolisms.

Varieties of Embolism

1. Intra vascular: it can be produced by solid fragments of thrombi (septic/Aseptic) or atheromatous material.
2. Extra vascular: A solid extra vascular embolus may be from normal tissue like marrow fragments, trophoblastic, decidual tissue, pancreatic tissue, cancer cells, clumps of bacteria, parasites, ova, cyst, pigmented granules, foreign bodies like cotton, silk, talc. Extra vascular embolus an be from any liquid element like fat globules, oil, amniotic fluid. Embolus can also be produced from gases like any air or nitrogen.

Localization of emboli: undissolved material carried in direction of flowing stream impacts in the vessel through which it cannot pass further.

1. Emboli originating in systemic veins goes to right heart from there it turns in pulmonary artery. This reaches to lungs to cause pulmonary embolism.
2. Embolus originating in pulmonary veins goes to left heart to reach aorta. From there it can logue anywhere in systemic arteries.
3. Emboli from portal trunk lodge in liver.

Systemic Embolism: Cardiogenic emboli can block brain, kidney, spleen and lower limb. Ulcerated atherosclerotic plaque from carotid bifurcation can reach brain and from abdominal aorta embolus can reach lower limb. Such trash emboli often composd of atheromatus debris and cholesterol crystals are due to complications of major arterial surgery.

Paradoxical Embolism: Venous embolus that gains access to arterial side through a heart wall defect.

Retrograde Embolism: Carriage of an embolism in a direction reversed to the usual flow of lymph or blood. A sharp increase in pressure within cavities as during coughing or vomiting causes reversal of blood flow to enable blood from the lung to reach brain. Also from pelvic and abdominal region to reach spinal cord or brain.

Effects of Embolism: This depends upon nature of embolus, size of vessel, amount of collateral circulation available and nature of organ.

- Aseptic infarct or gangrene
- Septic infarct or mycotic aneurysm
- Metastasis if embolus contains tumor cells.
- Sudden death (if there is large sized artery block)
- Transient ischemic attack.

Pulmonary Embolism

One patient of every 5 with deep vein thrombosis may have pulmonary thrombosis. Large sized embolism leads to sudden death. Medium sized embolism cause pulmonary infarction. Silent micro emboli cause extensive patchy damage in lungs. This leads to chronic cor pulmonale pulmonary hypertension. Its clinical presentations are : dyspnoea, chest discomfort, syncope.

Fat Embolism: fat droplets of more than 8 micro metere can cause embolism. The force of surface tension which gives fat droplets their spherical shape can well counteract the capillary blood pressure. The droplets cannot be deformed or cannot be squeezed out of capillaries. Fat embolism can be caused due to long bone fracture (due to disruption of fatty marrow), severe injury of subcutaneous tissue, deep burns, injection of oily solutions, pelvic injury during child birth, liposuction, fat necrosis from acute pancreatitis, unsuccessful cardio thorasic bypass surgery, fatty liver, ether anaesthesia, gas gangrene. Fat globules enter torn vessels and give symptoms like respiratory distress, cerebral syndrome, petechiae in skin and mucosa.

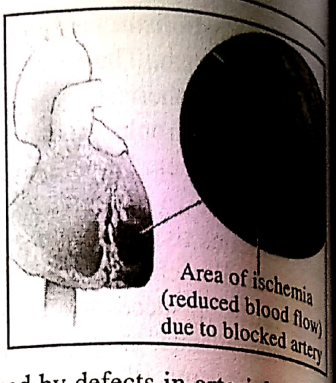
Amniotic Fluid Embolism: Amniotic fluid contains mucin (meconium and foetal components), fat (sebaceous glands) desquamated epithelial cells (skin and lanugo hairs) they enter uterine sinusoids during labour and concealed accidental haemorrhage bypassing between the ruptured membrane wall to reach placental margine and produce pulmonary embolism, obstertetial shock and dessiminated thrombosis.

Gas embolism: The gas embolus may be produced during surgery in neck, chest when large veins open up. It can also occur during cardiac operations, angiographic operations, blood transfusion or intravenous therapy, vaginal douching, induced abortion, insuffulation of fallopian tubes, induction of artificial pneumothorax, pneumoperitonium or any laproscopic procedures. Air enter vein and carried to right heart. Here air and blood churn up into froth. This froth acts as air trap and block the entry of air into pulmonary artery. This leads to massive pulmonary embolism and sudden death. Nearly 100ml-150ml of air is required for this effect. Arterial air embolism leads to blockage of cerebral or coronary artery. In this small amount of air is sufficient to produce death.

'ISCHEMIA'

Inadequate blood supply to a part of the body, even to the point of complete deprivation is ischemia. The inadequacy may be relative, a sudden demand for blood because of increased functional activity. Ischemia primarily affects components of highest oxygen requirement.

Causes: Atherosclerosis, thrombosis, embolism, vasculitis, muscular spasm of vessel wall and external pressure.



Generalized, diffused ischemia is caused by conditions such as hypovolemic shock and cardiac failure as a result of systemic hypotension. Ischemia of an organ or tissue may be caused by defects in arterial supply, the capillaries within that organ or the venous return.

The arterial cause of ischemia: Due to thrombosis, embolism, spasm of vessels as seen in Raynaud's disease, ergot poisoning, TAO, arteriosclerosis, polyarthritis nodosa, pressure from outside due to ligature,tourniquet, tight plaster

The venous cause may be due to all above causes along with hematoma, strangulation. Venous ischemia is rarer as veins have more extensive anastamosis than arteries.

The ischemia in capillaries may be due to frost bite, haemolytic anaemia (RBC agglutination), sickle cell disease, malaria, chronic myeloid leukaemia, disseminated intravenous coagulopathy, decubetus bed sore.

Brain and heart are more vulnerable for anoxia. Anoxia for more than few minutes leads to severe damage. Retina and smaller cerebral arteries are end arteries as they do not have any anastamosing branch. Therefore ischemia can be more dangerous to these organs than others.

Effects of ischemia: Ischemia produces damage to tisssues through

1. Anoxia/deprivation of oxygen
2. Deprivation of nourishment
3. Accumulation of waste products of metabolism.

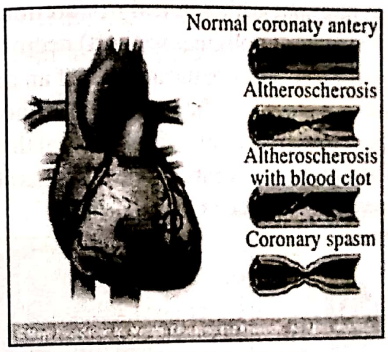
Changes After Ischemia

1. Functional damage: Angina pectoris, intermittent claudication

2. Degeneration, Atropy
3. Fibrosis
4. Infarction

Factors Determining Extent of Damage by Ischemia

1. Anatomy of vasculature of affected area (collateral circulation)
2. Nature of affected tissue.
3. Rate and degree of obstruction. Sudden obstruction has more severe effect than gradual obstruction. In gradual obstruction there is time for collateralcirculation to develop.
4. Generalised disease affecting circulation.



'INFARCTION'

This is usually due to sudden and complete deprivation of the blood supply. It is an area of necrosis produced by deprivation of blood supply. Non ischemic necrosis produced by physical, chemical, bacteria etc are not infarct. Putrifaction is absent in infarction.

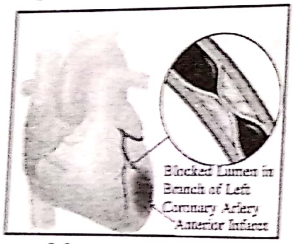
Causes: It is due to arterial or venous obstruction, sudden blockage of artery, sudden fall of BP distal to the point of block. The resultant anoxia leads to atonic dilation of capillaries , venules and increased permeability of plasma and RBC that ooze out. Hence ischemic area becomes swollen and hemorrhagic. Attempt of collateral circulation may reduce some amount of infarct. Failure to circulation leads to increased anoxia. This causes chemical injury because of accumulation of metabolites and hence results in necrosis.

Microscopic Changes of Infarction

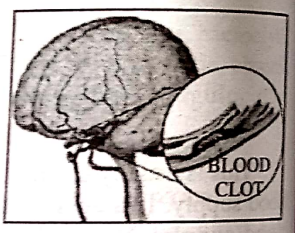
- Depletetion of muscle glucogan
- Clumping of nucleoplasm
- Myofibrils show transverse tears.
- Degenerative changes in parenchymal cell appear within few hours. Necrosis will be evident within 48hrs.
- Increase in lactic acid
- Swollen mitochondria

Gradually the excess of blood in capillaries and vein is drained away. The oozed red cells are lysed and their hemoglobin is removed by diffusion. With these events infarct become pale. Products from the necrotic tissue irritate the surrounding living tissue and set up a zone of inflammational at the periphery.

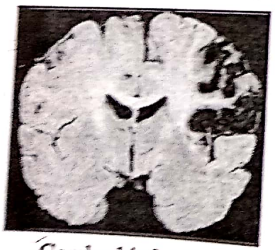
Nature of infarction: pathologically infarction is an area of either coagulative (firm) or colliquative (soft) necrosis. Infarcts are usually seen in periphery because the arteries of an organ generally branch out as fan like structure. Infarcts are commonly conical or pyramidal with base towards periphery and apex at the site of arterial obstruction. Various types of infarcts like red infarct, pale infarct, septic infarct, aseptic infarct can be found.



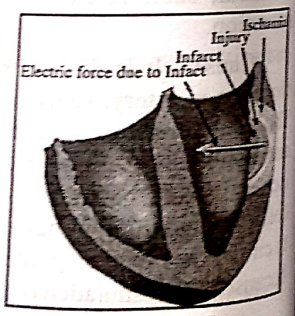
Myocardial infarction



Cerebral infarction



Cerebral infarction



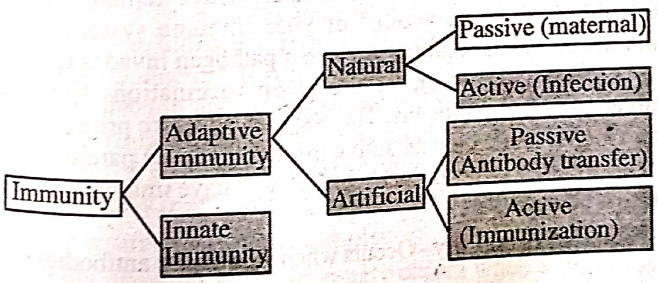
5. Types of Immunity- different types of immune responses in the body- Basic knowledge of auto immune diseases, Acquired immune deficiency disease and hypersensitivity

It is the defense mechanism in which body attains resistance against entry of antigen and prevent disease onset. It is evolved to recognize and to eliminate foreign molecules through an integrated network of cellular and molecular interactions and thus providing the protection against disease. The host defense mechanisms are:

1. Physical and chemical barriers: skin, mucus membrane, gastric acid, lysosome, lacto ferrin.
2. Mechanical removal: sneezing, coughing, secretion, ciliary action of respiratory system
3. Colonization resistance: normal flora preventing colonization with pathogenic organisms
4. Immune response : state of having sufficient biological defenses to avoid infection, disease, or other unwanted biological invasion. Immunity involves both specific and non-specific components. The non-specific components act either as barriers or as eliminators of wide range of pathogens irrespective of antigenic specificity. Other components of the immune system adapt themselves to each new disease encountered and are able to generate pathogen-specific immunity

Classification of Immunity

Innate immunity: it is an immediate response as infectious



agents enter the body, the inflammatory process directly activates. Eg : phagocytes like nutraphils, monocytes, eosinophils gets activated against parasites particularly nematodes. Basophils and mast cells contain histamine and other vassoactive amines. They bear high affinity IgG and participate in immediate hypersensitive reactions.

Specific or Adaptive immunity: If effective protection is not availed by innate immunity then adaptive immune system is induced. This response develops after period of days and is mediated by lymphocytes, which express antigen specific receptors. Features of specific immunity are as follows.

1. Specificity- Distant antigen generate specific response.
2. Diversity- Antigens are recognized by different lymphocytes.
3. Memory- Re exposure to antigen induces rapid and effective response.
4. Self regulation- Normal immune response regress with time.
5. Self and non self discrimination- during development the lymphocytes learn to distinguish between self and foreign antigens so that there is no unwanted mutual destruction.

Acquired Immunity– Its responses are antigen-specific responses in which the body recognizes a foreign substance and selectively reacts to it. This is mediated primarily by lymphocytes. Acquired immunity overlaps with the process of innate immunity. Acquired immunity can be subdivided into active immunity and passive immunity.

Active Immunity– occurs when the body is exposed to a pathogen and produces its own antibodies. Active immunity is active because it is the "activation" of your immune system. Active immunity can occur naturally, when a pathogen invades the body, or artificially, like when we are given vaccinations containing disabled or killed pathogens. The body does require prior exposure to an antigen to develop an active immunity. Some parents expose their children to some antigens so they will have immunity to these diseases later in life.

Passive Immunity– Occurs when we acquire antibodies made

by another human or animal. Passive immunity is passive because it requires no response from the person's immune system. In passive immunity you are not presenting the body with foreign antigens. Therefore your immune system will not need to use B cells, and we know that if the B cells are never introduced your body isn't making antibodies and it isn't making memory B cells. The transfer of antibodies from mother to fetus across the placenta is one example. Injections containing antibodies are another. Sometimes travelers going abroad may be injected with gamma globulin, but this passive immunity last only about three months. Passive immunizations are used to protect people who have been exposed to infections or toxins, like snake venom or tetanus.

Naturally Acquired Passive Immunity

Maternal passive immunity is a type of naturally acquired passive immunity, and refers to antibody-mediated immunity conveyed to a fetus by its mother during pregnancy. Maternal antibodies (MatAb) are passed through the placenta to the fetus by an FcRn receptor on placental cells. This occurs around the third month of gestation. IgG is the only antibody isotype that can pass through the placenta. Passive immunity is also provided through the transfer of IgA antibodies found in breast milk that are transferred to the gut of the infant, protecting against bacterial infections, until the newborn can synthesize its own antibodies.

Artificially Acquired Passive Immunity

Artificially acquired passive immunity is a short-term immunization induced by the transfer of antibodies, which can be administered in several forms; as human or animal blood plasma, as pooled human immunoglobulin for intravenous or intramuscular use, and in the form of monoclonal antibodies. Passive transfer is used prophylactically in the case of immunodeficiency diseases, such as hypogammaglobulinemia. It is also used in the treatment of several types of acute infection, and to treat poisoning. Immunity derived from passive immunization lasts for only a short period of time, and there is also a potential risk for hypersensitivity reactions, and serum sickness, especially from gamma globulin of non-human origin

Naturally Acquired Active Immunity

Naturally acquired active immunity occurs when a person is

exposed to a live pathogen, and develops a primary immune response, which leads to immunological memory.This type of immunity is "natural" because it is not induced by man. Many disorders of immune system function can affect the formation of active immunity such as immunodeficiency (both acquired and congenital forms) and immunosuppression.

Artificially Acquired Active Immunity

Artificially acquired active immunity can be induced by a vaccine, a substance that contains antigen. A vaccine stimulates a primary response against the antigen without causing symptoms of the disease. The term vaccination was coined by Edward Jenner and adapted by Louis Pasteur for his pioneering work in vaccination. The method Pasteur used entailed treating the infectious agents for those diseases so they lost the ability to cause serious disease. Pasteur adopted the name vaccine as a generic term in honor of Jenner's discovery, which Pasteur's work built upon.

Defenses Against Infection

1. Innate Defense- first line of defense

Physical and chemical barriers are the body's first line of defense.

Physical or Mechanical Barriers

Skin

One of the bodies first line of defenses against bacteria and other harmful organisms is the skin. Our skin is a barrier which stops infection from entering the body. Millions of microorganisms live harmlessly on the skin and in the air around us. Sebaceous glands in the skin produce sweat and sebum, which, combined help to protect the skin. Both substances contain antiseptic molecules, primarily lysozyme which breaks down bacterial cell walls.

Mucus Membranes

The mucous membranes line various body cavities that are exposed to the external environment and internal organs. It is at several places continuous with skin: at the nostrils, the lips, the ears, the genital area, and the anus. The nose and mouth serve as passageways for air going to and from the lungs. As we inhale and

exhale, the mucus membranes that line these passageways warm and humidify the air. Mucus membranes serve different functions, however, their more important job is to secrete mucus that traps bacteria and other foreign debris that irritates the lining of the respiratory tract. This mucus is produced and stored in the sinuses by other mucus membranes. We get congested when there is excessive fluid in the sinus cavities. This is a result of an increase in mucus secretions, as well as an increase in the amount of fluids that passes across the blood vessels of the mucus membranes that line the nose and sinus.

Mucociliary Escalator

The mucociliary clearance of the respiratory tract is an important defense mechanism against foreign debris and inhaled pathogens. The cilia that lines the upper and lower airways are lined with a thin layer of mucus. These beat rapidly to propel particles that are trapped in the mucus layer to the pharynx. Defective mucociliary clearance predisposes our respiratory tracts to recurrent infections. These cilial defects may be either congenital or acquired by infection, toxins or drugs.

Chemical Defenses

- Tears, saliva

Tears and saliva contain lysozyme, an antiseptic enzyme that attacks cell walls of bacteria and breaks them down.

- Stomach acids

Glands in the stomach lining produce hydrocloric acid. This acid kills most invading organisms that are swallowed and take up residence there.

2. Non-specific Responses to Infection - 2nd Line of Defense

The outermost defense our body has is our skin. The sebaceous glands produce sweat and sebum, which contain ANTISEPTIC properties which protect. This bacteria-killing substance called LYZOSOME is also found in tears and saliva. Acidic urine in the urinary tract and friendly bacteria in the genital tract prevent the multiplying of harmful organisms in these areas. Most invading organisms in the stomach are killed by gland production of

hydrochloric acid. These are a few examples of how the outer defenses protect us. All outer defenses work together as the body's first line of defense.

Inflammatory Response

Any break in the skin will allow bacteria to enter the body. These foreign microbes will cause swelling and reddening at the site of injury. This reaction by the body is called an inflammatory reaction or inflammatory response.

- **Swelling, redness, heat, and pain**

Inflammation is characterized by the following quintet: swelling (tumor), redness (rubor), heat (calor), pain (dolor) and dysfunction of the organs involved (functio laesa). When an injury occurs, a capillary and several tissue cells are apt to rupture, releasing histamine and kinins. These cause the capillaries to dilate, become more permeable, and leak fluid into these tissues. Dilation and fluid leaking into the tissues causes swelling, redness, and heat. The swelling and kinins stimulate nerve endings, causing pain. If there has been a break in the skin due to the injury, invading microbes may enter.

- **Phagocytosis by neutrophils and macrophages**

In the event of a break in the skin, neutrophils, monocytes (and macrophages) arrive and attempt to engulf and destroy the invaders. Phagocytosis is receptor-mediated event, which ensures that only unwanted particles are ingested. Stimulated macrophages can bring about an explosive increase in the number of leukocytes by producing Colony Stimulating Factors (CSFs). The CSFs pass by way of the blood to the bone marrow, where they stimulate the production and the release of white blood cells (WBCs), primarily neutrophils. Lymphocytes in nearby lymph nodes produce specific antibodies to attack the microbes. During the conflict, some neutrophils die and become mixed with dead tissue, bacteria, living white cells, etc. This thick yellow-white fluid is called pus. When a person has an illness, an examination of the numbers and types of WBC's in their blood can be very useful.

- **Complement System**

The complement system is a biochemical cascade of the immune system that helps clear pathogens from an organism, and promote healing. It is derived from many small plasma proteins that work together to form the primary end result of cytolysis by disrupting the target cell's plasma membrane.

Complement is activated by antigen-antibody complexes and causes holes to form in the plasma membrane of foreign microbes or cells (lysis). The complement system is considered a nonspecific defense, but it can be activated against specific microbes that have been marked with antibodies. Hemolytic transfusion reactions are caused by complement activation after a person expresses antibodies against the antigens found on the inappropriately donated blood. Hemolytic Disease of the Newborn (HDN) is due to maternal antibodies against the Rh factor crossing the placenta, binding to the baby's red blood cells, and stimulating the baby's own complement system to lyse its red blood cells.

3. Adaptive Defense (Specific Defense-third line of defense)

This part of the immune system directly targets invading microbes. Our specific immune defenses respond to antigens. An antigen is a protein (or polysaccharide) molecule, typically on the cell membrane, that the body recognizes as nonself. They are found on microbes, foreign cells, or on cancer cells. Normally our immune system does not respond to our own antigens (if it does, then this is an autoimmune disease). Sometimes we develop an immune response to a harmless antigen, such as pollen or cat dander (this is an allergic response).

- **Lymphocytes**

Specific immunity is dependent upon two types of lymphocytes, the B cells and the T cells. Their names are based on where in the body they mature. B cells mature in the bone marrow, and T cells mature in the thymus gland. In comparison, both B and T cells can recognize and target antigen-bearing cells, although they go about this in different ways. B and T cell lymphocytes are capable of recognizing an antigen because they have specific receptor

molecules on their surface which exactly fit individual antigens (like a lock and a key). Any B or T cell can only respond to one type of antigen. The body does not know ahead of time which antigens it will encounter, but rather makes receptor sites for a huge number of possible antigens. It is estimated that for the million or so antigens we encounter in our lifetime we have an equal number of specific lymphocytes for each possible antigen.

B cell lymphocytes are responsible for antibody-mediated immunity (humoral immunity). They produce antibodies, which are proteins that bind with and neutralize specific antigens. Antibodies do not directly kill bacteria, but mark them for destruction. When antibodies bind to viruses they can prevent the viruses from infecting cells. When antibodies bind to toxins they can neutralize the toxin (why we get immunized against the tetanus toxin). Humoral immunity works best fighting against target viruses, bacteria, and foreign molecules that are soluble in blood and lymph before the bacteria or viruses have entered into cells (extracellular bacteria and extracellular viruses).

B cells produce two different types of cells:

- Plasma cells
- Memory cells

Plasma Cells

As B cells mature during embryonic development, they develop surface receptors that allow them to recognize specific antigens. Then they travel in the bloodstream, distributing throughout the lymph nodes, spleen, and tonsils. Once B cells reach their destination, they remain inactive until they encounter a foreign antigen with an antigen that matches their particular receptor site (most B cells remain inactive for your entire life). The foreign antigen must be presented to the B cell directly, but usually macrophages and T cell lymphocytes (helper T cells) interact with B cells as Antigen Presenting Cells to bring about antibody production. Upon such an encounter, the B cell's receptors will bind to the antigen. The appropriate B cell is turned on or stimulated. It then grows bigger and rapidly multiplies into a large homogenous group (clone). Most of these cells are plasma cells, which actively secrete antibody that will bind with the original stimulating antigen. While most of the cells remain in the lymphatic system, the antibodies are secreted

into the lymph fluid which then enters into the blood plasma to circulate throughout the body. Although the clone cells only live a few days, their antibodies remain and circulate in the blood and lymph, gradually decreasing in number.

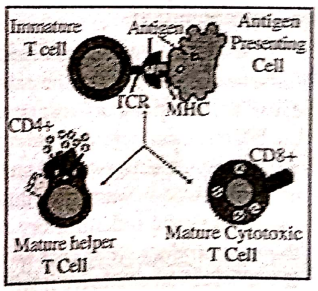
Antibody Structure and Function

There are different classes of antibodies, or immunoglobulins (Ig), such as IgA, IgG, IgE, and IgM. They can attach to the surface of a microbe and make it more easily phagocytized by neutrophils, monocytes and macrophages. Anything that simplifies phagocytosis is called an *opsonin*. The process of antibodies attaching to invaders can be termed 'opsonization.' Some antibodies can bind and inactivate certain poisons or toxins and are called antitoxins (tetanus immunizations stimulate your body to produce antibodies against the tetanus toxin rather than against the bacteria that produces the toxin). Still other antibodies can bind to the surface of microbes and prevent their attachment to the body's cells (thus preventing viruses from entering host cells). Also, some of them can stimulate nine proteins found in plasma, called complement.

Memory B Cells

At the time of activation some of the clones become memory B cells. These cells are long lived and have recorded the information about the foreign antigen so antibodies can be made more quickly, and in greater amount, in case a second exposure should occur. Since the second response is much stronger than the first and puts more antibodies into circulation, we often receive "booster shots" for immunizations.

T Cells Attack Infected Cells



Defending the body against intracellular pathogens is the role of T lymphocytes, which carry out cell-mediated immunity (CMI). Macrophages phago-cytize invading microbes and present parts of the microbe (antigens) to the T cell lymphocytes. The appropriate T cell is turned on or stimulated. The

activated T cell rapidly multiplies into a large homogenous group (clone) of cytotoxic T cells (Tc cells).

- (a) Attack organisms directly, Also kill infected cells

These cytotoxic T cells migrate to the site of infection (or disease) and produce chemicals which directly kill the invader. Cytotoxic T cells release "perforin" that causes pores to form in the plasma membrane of the target cell, resulting in lysis.

- (b) T cells develop in the thymus gland from immature precursor cells that migrate there from the bone marrow.
- (c) Killer and helper T cells • (d) Memory T Cells

A portion of these activated T cells become memory T cells (Tm). These cells record the information about the foreign antigen so T cells can respond more quickly, and more strongly, if a second exposure occurs. A portion of the T cells become T helper cells (TH) or T suppressor cells (Ts). TH cell stimulate other T cells and B cells by releasing cytokines and other stimulatory chemicals. Ts cells suppress the immune response. Experience has shown that cell mediated immunity is most useful to the body by: Protecting against microbes which exist inside of our body's cells (intracellular bacteria and intracellular viruses). Protecting against fungal infections. Protecting against protozoan parasites. Protecting against cancer cells.

Immune Response Pathways

The innate response starts first, and it is reinforced by the more specific acquired response. The two pathways are interconnected so cooperation and communication is essential.

- **Inflammation**

What happens when bacteria invade? If the first line of defense fails, bacteria can reach the extracellular fluid. There they usually cause an inflammatory response. This response coats antigens on the bacterial surface, with antibodies. Then in return the antibodies will ingest the antigens with phagocytic cells. This is characterized by a red, swollen warm area that is tender or painful. In addition to the nonspecific inflammatory response, lymphocytes attracted to the area produce antibodies keyed to the specific type of bacteria. If the infection continues it will produce a fever.

During an infection macrophages may release cytokines, such as interleukin-1, that travel to the hypothalamus and induce a change in the thermostat setting. When the thermostat is raised to a new normal temperature, the previous body temperature now registers as too cold. To increase the temperature to the new level, our body shunts blood away from the skin (leaving it feeling cold and clammy), the heart rate increases, and we shiver to generate heat until we reach the new set point. The hypothalamus may subsequently lower the thermostat, in which case we suddenly feel hot and start to sweat as our body attempts to cool off. A person may cycle between chills and sweats during the course of an infection. While a fever can be dangerous if it gets too high, or if a patient is weak or has heart trouble, there is some evidence suggesting that the body may overcome an infection faster if a fever is allowed to run its course.

'IMMUNE SYSTEM DISORDERS'

The immune system is a very complex and highly developed system, yet it has a very simple mission, that is to find and destroy the invaders. When the immune system does not function properly it leaves the body open for attacks from an vast array of diseases. This can be classified these into three broad categories; Autoimmunity, Immunodeficiencies, and Hypersensitivities.

"AUTO IMMUNITY"

Anything that can trigger the immune response is called an antigen. An antigen can be a microbe such as a virus, or even a part of a microbe.

Immune response against one's own antigen is called auto immunity. It is an endogenous immune response to an endogenous antigen. Body's immune system fails to distinguish between self and non self and reacts by formation of auto antibodies against one's own tissue antigens. This may turn fatal. The targets of auto immune reactions may be intracellular components, extracellular components, receptors, cell membranes components, plasma proteins or hormones. Both antibody and T cell response to self antigen cause disease.

1. Normal auto immunity. It is needed so as this allows the

clearance of self antigen debris from circulating and production of complex network of immune regulation called diotype network. It plays important role in wound healing and clearing cell debris.

2. Disease associated auto immunity. There is breakdown in the control mechanism of self tolerance that could lead to disease.

Fundamental Principles

- Results from failure from discrimination between self and non self, demonstration of immune response to an auto antigen.
- Certain HLA genes are associated with increased susceptibility of such tendency.
- There is loss of immunological tolerance in auto immunity.

Autoantigen → Auto antibody → Auto immune disease.

Tolerance is specific immunological unresponsiveness. That is immune response to a certain antigen does not occur although the immune system is otherwise functioning normally. Immune system distinguishes between antigen that is foreign and one that is normally present on the cell in the body by selection in the thymus during fetal life. Stem cells in the bone marrow produce prothymocytes which are attracted to thymus by the chemotaxic agent called thymotaxin. There they mature producing cells which recognize 'self' antigens as well as 'foreign' antigens. But self reacting T cells are eliminated or inactivated. This mechanism induces tolerance so that antigens that are exposed to the immune system during fetal life are not capable of eliciting an immune response in later life. Parts of the body that are not exposed to the immune system during fetal life can produce an immune response later on. Eg- lense protein, spermatozoa. Even antigens exposed during fetal life may provoke immune activation much later in life leading to group of diseases called auto immune disease.

Certain examples for auto immune diseases are. Rheumatoid Arthritis, Multiple sclerosis, immune mediated or type 1 diabetes, inflammatory bowel disease, systemic lupus erythematosus, psoriasis, scleroderma, auto immune thyroid disease, antisperm antibody leading to infertility.

For reasons we do not fully understand, sometimes the immune system attacks the body the way it normally would attack a germ or foreign substance. The genes some people inherit can contribute to their susceptibility to develop an autoimmune disease. Most autoimmune diseases affect woman more than men.

- In **Juvenile-onset diabetes** the immune system starts attacking and eliminating the cells in the pancreas that make insulin.
- **Multiple Sclerosis** is a chronic degenerative disorder of the central nervous system where the immune system starts attacking and destroying vital myelin in the brain and spinal cord. This causes multiple sclerosis (scars) on the myelin sheath resulting in loss of nerve function.
- Another fairly known disorder is **Rheumatoid Arthritis** this is when the immune system starts attacking the tissue inside your joints.
- In **Organ and Tissue Transplants,** foreign tissue are placed inside the body. These tissues do not perfectly match the surrounding cells. The body sees this as something that should not be there and sends messages to attack and kill it. This can make transplanting nearly impossible. This problem can not be completely prevented but it can be diminished by making sure the donor tissue is a close match to the recipient tissue. In addition, the recipient is placed on immuno-suppressing drugs to try and prevent the immune system from attacking and rejecting the new organ or tissue.
- **Vitiligo** is an autoimmune disorder in which the immune system destroys pigment-making cells called melanocytes. This results in irregularly shaped milky-white patches of skin on different parts of the body. This is the condition which Michael Jackson claims to have. Thus it is a state in which the body's immune system fails to distinguish between self and non self and reacts by formation of auto antibodies against one's own tissue antigen.

Immunodeficiency Diseases

When the immune system is presented with foreign antigens in association with dendritic cells, a vigorous immune response ensues. (Antigens are the molecules on the surface of invader cells that announce them as different from the body's cells.). Alternatively,

dendritic cells can be exploited during the development of many immune based diseases.

- **AIDS and HIV**

Acquired immunodeficiency disease (AIDS) is a well-known immune system disease. It is a collection of symptoms and infections resulting from the specific damage to the immune system caused by the human immunodeficiency virus (HIV).The late stage of the condition leaves individuals prone to opportunistic infections and tumors. HIV is transmitted through direct contact of a mucous membrane or the bloodstream with a bodily fluid containing HIV such as blood, semen, vaginal fluid, preseminal fluid, and breast milk. This transmission can come in the form of anal, vaginal or oral sex, blood transfusion, contaminated hypodermic needles, exchange between mother and baby during pregnancy, childbirth or breastfeeding, or other exposure to one of the above bodily fluids. AIDS is the most severe manifestation of infection with HIV. HIV is a retrovirus that primarily infects vital components of the human immune system such as CD4+ T cells (a subset of T cells), macrophages and dendritic cells. It directly and indirectly destroys CD4+ T cells. CD4+ T cells are required for the proper functioning of the immune system. When HIV kills CD4+ T cells cellular immunity is lost, leading to the condition known as AIDS. Acute HIV infection progresses over time to clinical latent HIV infection and then to early symptomatic HIV infection and later to AIDS, which is identified on the basis of the amount of CD4+ T cells in the blood and the presence of certain infections.

The median time of progression from HIV infection to AIDS is nine to ten years, and the median survival time after developing AIDS is only 9.2 months. However, the rate of clinical disease progression varies widely between individuals, from two weeks up to 20 years. Many factors affect the rate of progression. These include factors that influence the body's ability to defend against HIV such as the infected person's general immune function. Older people have weaker immune systems, and therefore have a greater risk of rapid disease progression than younger people. Poor access to health care and the existence of coexisting infections such as tuberculosis also may predispose people to faster disease progression. The infected person's genetic inheritance plays an important role and some people are resistant to certain strains of HIV.

"HYPERSENSITIVITY"

This is defined as exaggerated or inappropriate state of normal immune response with onset of adverse effects on the body. The lesions are called as hypersensitive response or immunological tissue injury. Reaction depends upon rapidity, duration and type of immune response.

1. Immediate type : reaction occurs immediately as antigen enters (within seconds or minutes) this is mediated by humoral antibodies (Bcell) 1,2,3
2. Delayed type : reaction is slower 24-48 hours. And effect is prolonged mediated by cellular response (Tcell) type 4

Type 1 response : it is form of anaphylactic reaction. It is the state of rapidly developing immune response to an antigen to which an indivisual is previously sensitised. It can manifest in local irritation as seen by skin, nose, troat, lungs. This can be life threatening. The clinical presentation of type 1 hypersensitivity are itching,erythema,contraction of respiratory bronchioles, diarrhoes, pulmonary oedema, pulmonary haemorrhage shock and death.

Type 2 response. This is cytotoxic reaction. They attack cell surface antigen on specific cells and causes lysis of target cells. This response is seen in autoimmune hemolytic anaemia, transfusion reaction,thrombocytopenic purpura, leucopenia. They are cytotoxic to tissue components. Some of the conditions are Grave's disease in which auto antibodies are produced against TSH receptors. In Myasthenia gravis autoantibodies are produced against acetylcholine receptors. Antisperm antibody can be produced against spermatozoa to cause male sterility. Autoantibodies against islets of pancreas can produce type 1 diabetes mellitus.

Type 3 response. This is immune complex mediated reaction resulting from deposition of antigen antibody complex on tissue followed by activation of the complement system and inflammatory reaction. This leads to cell injury. The causes are persistant low grade microbial infection, extrinsic environmental antigen and auto immune process. Few of the examples are immunecomplex glomerulo nephritis, goodposture syndrome, SLE,Rheumatoid arthritis, farmers lung, poly arthritis.nodosa, wegener's granulomatosis, henoch-scholein purpura, Drug induced vasculitis.

- Type 4 response is delayed response. The tissue injury by cell mediated immune response without formation of antibodies. But it is instead slow and prolonged response of specifically sensitized T lymphocytes. The reaction occurs about 24 hrs after exposure to antigen.

6. Nomenclature and classification of tumors- difference between benign and malignant tumors

Neoplasia means new growth but not those which undergo process of embryogenesis, regeneration and repair, hyperplasia and hormonal stimulation. The proliferation and maturation of cells in normal adult is controlled.

- Liable cells - proliferate throughout
- Stable cells - limited proliferation
- Permanent cells - do not replicate.

The neoplastic cells lose control and regulation of replication and form abnormal mass of tissue. Thus it can be defined as "A mass of tissue formed as a result of abnormal, excessive, uncontrolled, uncoordinated autonomous and purposeless proliferation of cells even after cessation of stimulus for growth which caused it."

Components of Tumours

1. Parenchyma : The tissue
2. Stroma : fibrous connective tissue and blood vessels, it provides framework on which the parenchymal tumor cells grow.

Causes of Cancer

- Cancers are caused by abnormalities in the genetic material of the transformed cells.These abnormalities may be due to the effects of carcinogens, such as tobacco smoke, radiation, chemicals, or infectious agents.
- Other cancer-promoting genetic abnormalities may randomly occur through errors in DNA replication, or are inherited, and thus present in all cells from birth. The heritability of cancers is usually affected by complex interactions between carcinogens and the host's genome.

1. Mutation: Chemical Carcinogen

- The incidence of lung cancer is highly correlated with smoking.
- Cancer pathogenesis is traceable back to DNA mutations that impact cell growth and metastasis. Substances that cause DNA mutations are known as mutagens, and mutagens that cause cancers are known as carcinogens.
- Particular substances have been linked to specific types of cancer. Tobacco smoking is associated with many forms of cancer, causes 90% of lung cancer. Prolonged exposure to asbestos fibers is associated with mesothelioma

2. Mutation: Ionizing Radiation

- Sources of ionizing radiation, such as radon gas, can cause cancer. Prolonged exposure to ultraviolet radiation from the sun can lead to melanoma and other skin malignancies. It is estimated that 2% of future cancers will be due to current CT scans
- Non-ionizing radio frequency radiation from mobile phones and other similar RF sources has also been proposed as a cause of cancer.

3. Viral or Bacterial Infection

- Some cancers can be caused by infection with pathogens.
- Many cancers originate from a viral infection, this is especially true in animals such as birds, but also in humans, as viruses are responsible for 15% of human cancers worldwide.
- The main viruses associated with human cancers are human papillomavirus, hepatitis B and hepatitis C virus, Epstein-Barr virus, and human T-lymphotropic virus.

4. Hormonal Imbalances

- Some hormones can act in a similar manner to non-mutagenic carcinogens in that they may stimulate excessive cell growth. E.g; role of hyperestrogenic states in promoting endometrial cancer.

5. Immune System Dysfunction

- HIV is associated with a number of malignancies, including Kaposi's sarcoma, non-Hodgkin's lymphoma, and HPV-associated malignancies such as anal cancer, cervical cancer. Certain other immune deficiency states (e.g. common variable immunodeficiency and IgA deficiency) are also associated with increased risk of malignancy.

6. Heredity

- Most forms of cancer are sporadic, There are a number of recognised syndromes where there is an inherited predisposition to cancer, often due to a defect in a gene that protects against tumor formation. Famous examples are:
- Hereditary nonpolyposis colorectal cancer (HNPCC, also known as Lynch syndrome) can include familial cases of colon cancer, uterine cancer, gastric cancer, and ovarian cancer, without a preponderance of colon polyps.
- Retinoblastoma, when occurring in young children, is due to a hereditary mutation in the retinoblastoma gene

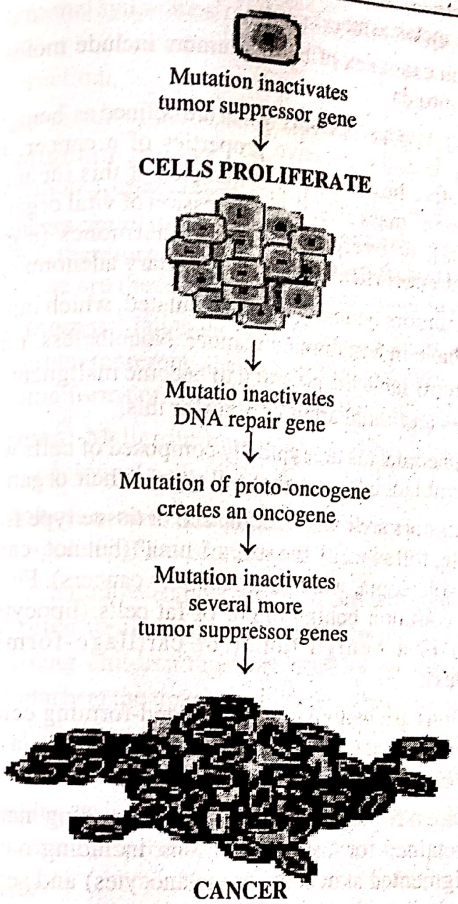
7. Other causes

- Transplacental transmission of acute leukaemia, lymphoma, melanoma and carcinoma from mother to fetus has been observed
- Diet :Reduced meat consumption is associated with decreased risk of colon cancer, and reports that consumption of coffee is associated with a reduced risk of liver cancer. Studies have linked consumption of grilled meat to an increased risk of stomach cancer, colon cancer, breast cancer, and pancreatic cancer, a phenomenon which could be due to the presence of carcinogens such as benzopyrene in foods cooked at high temperatures.

Pathogenesis :-

Stages of Cancer

- Stimuli: Achieved from above said causes
- Cell proliferation: Uncontrolled un coordinated cell division or replication
- Angiogenesis: New vascular tree grows for the mass and supply oxygen and nutrition for neoplastic cells.
- Growth: The continuous cell proliferation leads to mass or lump formation which is also called as tumors.
- Spread (metastasis): both Benign and malignant forms of tumors grow in the stroma among them malignant tumors spread to distant new area and produce new mass.
- Growth : The new mass called secondary tumour in the new place derived from primary tumor after metastasis make a new beginning in another mass formation due to cellular proliferation.



Classification of Neoplasia

1. Benign Tumor

The term benign implies a mild and nonprogressive disease, and indeed, many kinds of benign tumors are harmless to the health. It is a tumor that lacks all three of the malignant properties of a cancer. Thus, by definition

- Benign tumor does not grow in an unlimited, Aggressive manne
- Does not invade surrounding tissues,

- Does not metastasize.

Common examples of benign tumors include moles, lipoma and uterine fibroids.

However, some neoplasms which are defined as 'benign tumors' because they lack the invasive properties of a cancer, may still produce negative health effects. Examples of this include tumors which produce a "mass effect" (compression of vital organs such as blood vessels), or overproduce certain hormones (e.g; thyroid adenomas, adrenocortical adenomas, pituitary adenomas).

Benign tumors typically are encapsulated, which inhibits their ability to behave in a malignant manner. Nonetheless, many types of benign tumors have the potential to become malignant and some types, such as teratoma, are notorious for this.

Benign neoplasms are typically composed of cells which bear a strong resemblance to a normal cell type in their organ of origin.

These tumors are named for the cell or tissue type from which they originate, followed by the suffix "-oma" (but not -carcinoma, -sarcoma, or -blastoma, which are generally cancers). For example, a lipoma is a common benign tumor of fat cells (lipocytes), and a chondroma is a benign tumor of cartilage-forming cells (chondrocytes).

Adenomas are benign tumors of gland-forming cells, and are usually specified further by their cell or organ of origin, as in hepatic adenoma (a benign tumor of hepatocytes, or liver cells).

There are a few cancers with 'benign-sounding' names which have been retained for historical reasons, including melanoma (a cancer of pigmented skin cells, or melanocytes) and seminoma (a cancer of male reproductive cells).

Signs and Symptoms of Benign Tumor

- Benign tumors,may be asymptomatic or may cause specific symptoms depending on their anatomic location and tissue type. Symptoms or pathological effects of some benign tumors may include:
- Bleeding or occult blood loss causing anemia
- Pressure causing pain or dysfunction
- Cosmetic changes, Itching

- 'Hormonal syndromes' resulting from hormones secreted by the tumor.
- Obstruction,
- Compression of blood vessels or vital organs

2. **Malignant tumors** (cancers) are usually named using-carcinoma, -sarcoma or -blastoma as a suffix,

Cancers are classified by the type of cell that resembles the tumor and, therefore, the tissue presumed to be the origin of the tumor. These are the histology and the location, respectively.

- **Carcinoma:** Malignant tumors derived from epithelial cells. This group represents the most common cancers, including the common forms of breast, prostate, lung and colon cancer.

Sarcoma: Malignant tumors derived from connective tissue, or mesenchymal cells.

- **Lymphoma and leukemia:** Malignancies derived from hematopoietic (blood-forming) cells
- Germ cell tumor: Tumors derived from totipotent cells. In adults most often found in the testicle and ovary in fetuses, babies, and young children most often found on the body midline, particularly at the tip of the tailbone; in horses most often found at the poll (base of the skull).
- Blastic tumor or blastoma: A tumor (usually malignant) which resembles an immature or embryonic tissue. Many of these tumors are most common in children.

Metastasis

It is displacement, is the spread of a disease from one organ or part to another non-adjacent organ or part. It had been previously thought that only malignant tumor cells and infections have the capacity to metastasize.

- Cancer cells can break away, leak, or spill from a primary tumor, enter lymphatic and blood vessels, circulate through the bloodstream, and be deposited within normal tissue elsewhere in the body. Metastasis is hallmark of malignancy. Most tumors and

other neoplasms can metastasize, although in varying degrees (e.g., glioma and basal cell carcinoma rarely metastasize)

- When tumor cells metastasize, the new tumor is called a secondary or metastatic tumor, and its cells are like those in the original tumor. For example, that, if breast cancer metastasizes to the lungs, the secondary tumor is made up of abnormal breast cells, not of abnormal lung cells. The tumor in the lung is then called metastatic breast cancer, not lung cancer.

Mode of Spread

- Blood stream
- Lymphatic drainage
- Both
- Direct spread

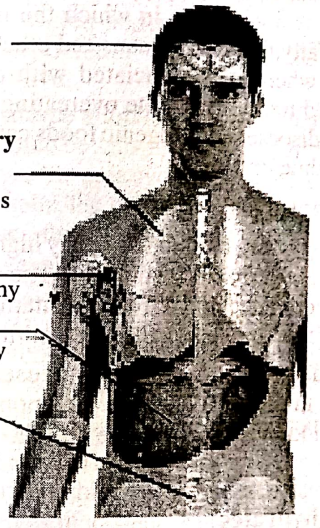
Cancer cells may spread to lymph nodes (regional lymph nodes) near the primary tumor. This is called nodal involvement, positive nodes, or regional disease. ("Positive nodes" is a term that would be used by medical specialists to describe a patient's condition, meaning that the patient's lymph nodes near the primary tumor tested positive for malignancy.

- Localized spread to regional lymph nodes near the primary tumor is not normally counted as metastasis, although this is a sign of worse prognosis. In addition to the above routes, metastasis may occur by direct seeding, called transcoelomic spread. This is generally only seen in the peritoneal or pleural cavities by ovarian tumours and mesotheliomas respectively.
- Metastatic tumors are very common in the late stages of cancer. The spread of metastases may occur via the blood or the lymphatics or through both routes. The most common places for the metastases to occur are the lungs, liver, brain, bones There is also a propensity for certain tumors to seed in particular organs. This was first discussed as the "seed and soil" theory by. For example, prostate cancer usually metastasizes to bones. In a similar manner, colon cancer has tendency to metastasize to the liver. Stomach cancer often metastasizes to the ovary in women.

Signs and Symptoms: They Depend on the Location of the Tumor

- Local symptoms: unusual lumps or swelling, hemorrhage, pain and/or ulceration. Compression of surrounding tissues may cause symptoms such as jaundice.
- Symptoms of metastasis (spreading): enlarged lymph nodes, cough, hemoptysis, hepatomegaly, bone pain, fracture of affected bones, neurological symptoms. Sever pain.
- Systemic symptoms: wt loss, poor appetite, fatigue cachexia, night sweats, anemia and specific paraneoplastic phenomena, i.e. specific conditions that are due to an active cancer, such as thrombosis or hormonal changes.

Common sites and symptoms of Cancer metastasis



Diagnosis of tumor

- Clinical observation,
- Histopathological study or biopsy,
- Fine needle aspiration cytology,
- Radiological investigation through X-ray, CT scan,
- Tumor markers.

7. Introduction to Nutritional disorders- disorders of macro nutrients

A nutritional disorder is a disease that results from excessive or inadequate intake of food and nutrients which leads to conditions such as obesity, kwashiorkor and rickets. Nutritional disorders usually result from long-standing states and habits such as malnutrition, compulsive disorders and abnormal intake of specific nutrients and minerals.

Overnutrition

Metabolic

Obesiy is caused by consuming too many calories compared to the amount of exercise the body is performing, causing a distorted energy balance. It can lead to diseases such as cardiovascular disease and diabetes. Obesity is a condition in which the natural energy reserve, stored in the fatty tissue of humans and other mammals, is increased to a point where it is associated with certain health conditions or increased mortality. Acute overeating can also be a symptom of an eating disorder. Goitrogenic foods can cause goitres by interfering with iodine uptake.

Vitamins and Micronutrients

Vitamin poisoning is the condition of overly high storage levels of vitamins, which can lead to toxic symptoms. The medical names of the different conditions are derived from the vitamin involved: an excess of vitamin A, for example, is called."hypervitaminosis A".

Iron overload disorders are diseases caused by the over accumulation of iron in the body. Organs commonly affected are the liver, heart and endocrine glands in the mouth.

Deficiencies

Proteins/fats/carbohydrates

- Protein-energy malnutrition

Kwashiorkor : Kwashiorkor is a severe form of malnutrition, caused by a deficiency in dietary protein. The extreme lack of protein causes an osmotic imbalance in the gastro-intestinal system causing swelling of the gut diagnosed as an edema or retention of water.

Extreme fluid retention observed in individuals suffering from kwashiorkor is a direct result of irregularities of the lymphatic system

and capillary exchange. The lymphatic system serves three major purposes: fluid recovery, immunity, and lipid absorption. Victims of kwashiorkor commonly exhibit a reduced ability to recover fluids, immune system failure, and low lipid absorption, all of which result from severe undernourishment. Fluid recovery in the lymphatic system is accomplished by re-absorption of water and proteins which are returned to the blood. However, due to the lack of proteins, no substantial pressure gradient can be established to draw fluids from the tissue back into the blood stream. This results in the pooling of fluids, causing the swelling and distention of the abdomen.

The low protein intake leads to some specific signs:

- Edema of the hands and feet,
- Irritability, anorexia,
- Desquamative rash, hair discolouration,
- Large fatty liver.
- The typical swollen abdomen is due to two causes: ascites because of hypoalbuminemia (low oncotic pressure), and enlarged fatty liver.

Protein should be supplied only for anabolic purposes. The catabolic needs should be satisfied with carbohydrate and fat. Protein catabolism involves the urea cycle, which is located in the liver and can easily overwhelm the capacity of an already damaged organ. The resulting liver failure can be fatal. This means in patients suffering from kwashiorkor, protein must be introduced back into the diet gradually. Clinical solutions include weaning the affected with milk products and increasing the intake of proteinaceous material to daily recommended amounts.

Marasmus

Marasmus is caused by a severe deficiency of nearly all nutrients, especially protein and carbohydrates. It can be distinguished from kwashiorkor in that kwashiorkor is protein deficiency with adequate energy intake whereas marasmus is inadequate energy intake in all forms, including protein. Protein wasting in kwashiorkor may lead to edema. The malnutrition associated with marasmus leads to extensive tissue and muscle wasting, as well as variable edema. Other common characteristics include.

- Dry skin, loose skin folds hanging over the buttocks (glutei) and armpit (axillae), etc.
- There is also drastic loss of adipose tissue (body fat) from normal areas of fat deposits like buttocks and thighs.

• The afflicted are often fretful, irritable, and voraciously hungry

Intellectual disability

The signs and symptoms of intellectual disability are a behavioral. Most people with intellectual disability do not look lik they are afflicted with such, especially if the disability is caused b environmental factors such as malnutrition or lead poisoning. Th so-called typical appearance ascribed to people with intellectu disability is only present in a minority of cases, all of which a syndromic.

Children with intellectual disability may learn to sit up, crawl, or to walk later than other children, or they may learn to ta later. Both adults and children with intellectual disability may als exhibit some or all of the following characteristics:

- Delays in oral language development
- Deficits in memory skills
- Difficulty learning social rules
- Difficulty with problem solving skills
- Delays in the development of adaptive behaviors such as self help or self-care skills
- Lack of social inhibitors

Dietary Minerals

Calcium

Osteoporosis : Osteoporosis itself has no symptoms; its ma consequence is the increased risk of bone fractures. Osteoporoti fractures occur in situations where healthy people would no normally break a bone; they are therefore regarded as fragili fractures. Typical fragility fractures occur in the vertebral colum rib, hip and wrist.

Rickets: Rickets is defective mineralization of bones befor epiphyseal closure in immature mammals due to deficiency o impaired metabolism of vitamin D, phosphorus or calcium, potentiall leading to fractures and deformity. The predominant cause is a vitam D deficiency, but lack of adequate calcium in the diet OR cases o severe diarrhea and vomiting may be the cause of the deficienc Although it can occur in adults, the majority of cases occur in childre suffering from severe malnutrition, usually resulting from famine starvation during the early stages of childhood.

Signs and symptoms of rickets include:

- Bone tenderness
- dental problems
- muscle weakness (rickety myopathy) increased tendency for fractures (easily broken bones), especially greenstick fractures
- Skeletal deformity
- Toddlers: Bowed legs (genu varum)
- Older children: Knock-knees (genu valgum) or "windswept knees"
- Cranial deformity (such as skull bossing or delayed fontanelle closure)
- Pelvic deformity
- Spinal deformity (such as kyphoscoliosis or lumbar lordosis)
- Growth disturbance
- Hypocalcemia (low level of calcium in the blood)
- Tetany (uncontrolled muscle spasms all over the body)
- Craniotabes (soft skull)
- Costochondral swelling (aka "rickety rosary" or "rachitic rosary")
- Harrison's groove
- Double malleoli sign due to metaphyseal hyperplasia
- Widening of wrist
- Tetany: Tetany or tetany seizure is a medical sign consisting of the involuntary contraction of muscles, which may be caused by disease or other conditions that increase the action potential frequency of muscle cells or the nerves that innervate them. Muscle cramps that are caused by the disease tetanus are not classified as tetany; rather, they are due to a blocking of the inhibition to the neurons that supply muscles.

Iodine deficiency

Goiter : Goitre is associated with hypothyroidism or hyperthyroidism may be present with symptoms of the underlying disorder. For hyperthyroidism, the most common symptoms are associated with adrenergic stimulation:

- Tachycardia,
- Palpitations,
- Nervousness,
- Tremor, and increased blood pressure.

Clinical manifestations are often related to hypermetabolism, including increased metabolism, excessive thyroid hormone, an increase in oxygen consumption, metabolic changes in protein metabolism, immunologic stimulation of diffuse goiter, and ocular changes (exophthalmos). Hypothyroid individuals have

- Weight gain despite poor appetite,
- Cold intolerance,
- Constipation and lethargy.

Selenium Deficiency

Keshan disease : Keshan disease is a congestive cardiomyopathy caused by a combination of dietary deficiency of selenium and the presence of a mutated strain of Coxsackievirus. Often fatal, the disease afflicts children and women of child bearing age, characterized by heart failure and pulmonary edema. Over decades, supplementation with selenium reduced this affliction

Iron Deficiency

Iron deficiency anemia: Iron-deficiency anemia is characterized by the sign of pallor (reduced oxyhemoglobin in skin or mucous membranes), and the symptoms of fatigue, lightheadedness, and weakness. None of the symptoms (or any of the others below) are sensitive or specific. Pallor of mucous membranes (primarily the conjunctiva) in children indicates anemia with best correlation to the actual disease,

Because iron deficiency tends to develop slowly, adaptation occurs and the disease often goes unrecognized for some time, even years; patients often adapt to the systemic effects that anaemia causes. In severe cases, dyspnea (trouble breathing) can occur. Unusual obsessive food cravings, known as pica, may develop. Other features are

- Anxiety, Irritability or a low feeling
- Angina
- Constipation
- Sleepiness/Hypersomnia
- Tinnitus
- Mouth ulcers
- Palpitations
- Hair loss
- Fainting or feeling faint
- Depression
- Breathlessness
- Twitching muscles
- Pale yellow skin
- Tingling, numbness, or burning sensations

- Missed menstrual cycle
- Koilonychia (spoon-shaped nails) or nails that are weak or brittle

Zinc

May exhibit with physical and mental Growth retardation

"VITAMINS"

Thiamine (Vitamin B1)

Beriberi : Symptoms of beriberi include weight loss, emotional disturbances, impaired sensory perception, weakness and pain in the limbs, and periods of irregular heart rate. Edema(swelling of bodily tissues) is common. It may increase the amount of lactic acid and pyruvic acid within the blood. In advanced cases, the disease may cause high output cardiac failure and death. Symptoms may occur concurrently with those of Wernicke's encephalopathy, a primarily neurological thiamine-deficiency related condition.

Beriberi is divided into three historical classifications:

1. Dry beriberi specially affects peripheral nervous system: Difficulty in walking

- Tingling or loss of sensation (numbness) in hands and feet
- Loss of tendon reflexes
- Loss of muscle function or paralysis of the lower legs
- Mental confusion/speech difficulties
- Pain
- Involuntary eye movements (nystagmus)
- Vomiting.

2. Wet beriberi specially affects the cardiovascular system and other bodily systems :

Increased heart rate

- Vasodilation leading to decreased systemic vascular resistance, and high output cardiac failure, Elevated jugular venous pressure, Dyspnea (shortness of breath) on exertion.
- Paroxysmal nocturnal dyspnea
- Peripheral oedema (swelling of lower legs)

3. Infantile beriberi affects also the children of malnourished mothers.

- Hoarseness, where the child makes moves to mourn but emits no sound or just faint moans, caused by nerve paralysis.
- Weight loss, becoming thinner and then marasmic as the disease progresses.

- Vomiting, Diarrhea
- Occasionally convulsions were observed in the terminal stages
- Pale skin
- Edema, Ill temper
- Alterations of the cardiovascular system, especially tachycardia (rapid heart rate).

Niacin (Vitamin B3)

Diarrhea, dermatitis, dementia and death. A more comprehensive list of symptoms includes:

- High sensitivity to sunlight
- Aggression
- Dermatitis, alopecia (hair loss), edema (swelling)
- Smooth, beefy red glossitis (tongue inflammation)
- Red skin lesions
- Insomnia
- Weakness
- Mental confusion
- Ataxia (lack of coordination), paralysis of extremities, peripheral neuritis (nerve damage)
- Diarrhea
- Dilated cardiomyopathy (enlarged, weakened heart)
- Eventually dementia

Vitamin C

Scurvy: Early symptoms are malaise and lethargy. After 1-3 months, patients develop shortness of breath and bone pain. Myalgias may occur because of reduced carnitine production. Other symptoms include skin changes with roughness, easy bruising and petechiae, gum disease, loosening of teeth, poor wound healing, and emotional changes. Dry mouth and dry eyes similar to SjÏgren's syndrome may occur. In the late stages, jaundice, generalized edema, oliguria, neuropathy, fever, convulsions, and eventual death are frequently seen.

Vitamin D

- Osteoporosis
- Rickets

8. Introduction to Infections

An infectious disease or communicable disease is caused by biological agent such as by virus, bacteria, fungus or parasites. Infectious disease are invasion of a host organism by a foreign replicator, generally often called as microbes that are invisible to naked eye. An organism that a microbe infects is known as host for that microbe. In the humans host a microbe cause disease by either disrespecting a vital body process or stimulating the immune system to mount a defensive reaction. An immune response against pathogen can include increased fever, inflammatory reaction and other damaging symptoms. An infection is the detrimental colonization in a host organism by a foreign species. The infectious agents are Virus, Bacteria, Fungus.

Virus

Virusus are strict parasites of other living cells, not only of mammalian and plant cells, but also of simple unicellular organism, including bacteria. Viruses are simple forms of replicating, biologically active particles that carry genetic information in either DNA or RNA molecules, but never in both. Most mature viruses have a protein coat over their nuclic acid and sometimes a lipid surface membranes derived from the cell they infect. Because viruses lack the protein synthesizing enzymes and structural apparatus necessary for their own replication, they bear essentially no resemblance to a true eukaryotic or prokaryotic cell. Virus replicate by using their own genes to direct the metabolic activities of the cell they infect to bring about synthesis and reassembly of their component parts. Viruses are approximately 100-1000fold smaller than cell they infect. They are approximately 20nm - 300nmin diameter. They contain protein shell called capsid with lipid membrane or envelop which is usually acquired from cytoplasmic membrane of infected cell. Protein or glycoprotein structure called spikes protrude from surface o9f virus particles. Te protein shell forming capsid assumes cylindrical or helical shape. They protect nuclic acid genome from damage, help in process of entry into the cell, and also package enzymes essential for infection process. Pox virus, hepatitis virus, erpes virus, retro virus, arbo virus are examples for virus.

Bacteria

They are smallest living cells of 0.1-10 micrometer. They have cytoplsmic membrane surrounded by a cell wall, a unique interwoven polymer called peptidoglycan which makes the cell wall rigid. The simple prokaryotic cell plan includes no mitocondria, lysosomes, endoplasmic reticulum, or other organells. Teir cytoplasm contains only ribosomes and a single, double- stranded DNA chromosome. Bacteria have no nucleus, but all chemical elements of nucleic acids and protein synthesis are present. Their nutritional requirement vary greatly, most of them are free living. Tiny metabolic factories divide by binary fission.

Vibrio, coccus, bacilli, spiral are varieties of bacteria. Tubercular bacilli, lepromatus bacilli, streptococcus are few examples of bacteria.

Fungi

They exist either in yeast or mold forms. The smallest yeast are similar to the size of bacteria but most are larger. Sizes vary from 2-20 micrometer. They multiply by budding. Molds form tubular extensions called hyphae which when linked together in a branched network form a fuzzy structure as seen on neglected bread. Fungi are eukaryotic and both yeasts and molds have a rigid external cell wall composed of their own polymers called glucan, mannan and chitin. Their genome may exist in a haploid or diploid state and replicate by meiosis or simple mitosis. Most fungi are free living and well distributed in nature. Generally fungi grow more slowly than bacteria.

Mycoses are diseases caused by fungi. Because of the similarity between human cells and fungal cells, it has been difficult for scientists to design antibiotics that are effective against fungi and do not harm humans. Some of the diseases caused by fungi are: Tineas, vaginal infection (candidiasis), and histoplasmosis.

9. Introduction and Classification of Microorganisms such as Virus-Bacteria-Fungus

Microbiology is the branch of science that deals with micro organisms or those which are not visible to naked eye. Microbiology

is the science defined by smallness. It was made possible by the invention of microscopes, which allowed the visualization of structures too small. One can know the microscopic living forms life cycle, morphology, chemical characters and other relevant information. The science of medical microbiology dates back to the pioneering studies of pasture and Koch, who has isolated specific agents and proven that they could ease disease by introducing the experimental method. The scientists studied the structure, physiology and genetics of microbes in detail and began to answer questions relating to the links between specific microbial properties and disease . These gradually extended from bacterial disease to Fungal, Parasitic and finally Viral infections.

Some characteristic features of microbes

- Some micro organisms synthesize nitrogen containing compounds that contribute to the nutrition of living things that lack this ability.
- Some like oceanic algae contribute to the atmosphere by producing oxygen through photosynthesis.
- Micro organisms have astounding range of metabolic and energy yielding abilities.
- Some can exist under conditions that are lethal to other forms for example, some bacteria can oxidize inorganic compounds such as sulfur and ammonium ions to generate energy and some can survive and multiply in hot springs at temperature higher than 75 degree centigrade.
- Some microbial species have adapted to symbiotic relationship with higher forms of life.

The major classes of micro organisms in terms of ascending size and complexity are viruses, bacteria, fungi and parasites. Parasites exists as single or multi cellular structures with the same eukaryotic cell plan of our own cell. Fungi are also eukaryotic, but has rigid external walls that make them seem more like plants than animals. Bacteria also has cell wall but their cell plan is prokaryotic and lacks the organelles of eukaryotic cells. Virus have a genome and some structural elements, but must take over the machinery of another living cell (prokaryotic or eukaryotic cell) to replicate.

Viruses:

Viruses are strict intracellular parasites of other living cells, not only of mammalian and plant cells, but also of single unicellular

organisms including bacteria. Viruses are simple forms of replicating, biologically active particals that carry genetic information in either DNA or RNA molecules, but never in both. Most mature viruses have a protein coat over this nucleic acid and sometimes a lipid surface membrane derived from the cell they infect. A single cell with infected viral partical may yield many thousands of viral particles. With many viruses, cell death and infections of other cells by the newly formed viruses results.

Bacteria

They are microbes of size 0.1 to 10 micro meters living cells. They have cytoplasmic membrane surrounded by cell wall as a unique interwoven polymer called peptidoglycan makes the wall rigid. The simple prokaryotic cell plan includes no mitochondria, lysosomes, endoplasmic reticulum or other organelles. The cytoplasm contains only ribosomes and single and double stranded DNA chromosomes. Bacteria have no nucleus, but all chemical elements of nucleic acid and protein synthesis are present. They are divided by binary fission and can be grown in artificial culture.

Fungi

Fungi exist in either yeast or mold forms. The smallest of yeasts are similar in size to bacteria, but most are larger 2-12 micro meter. They multiply by budding. Molds form tubular extension called hyphae. When linked together in a branched network, they form fuzzy structure. They are eukaryotic, both yeast and molds have external rigid cell wall composed of their own unique polymers called as glucan, mannan and chitin. Their genome may exist in a diploid or haploid state and replicate by meiosis or simple mitosis. Most fungi are free living and widely distributed in nature. Generally, fungi grow more slowly than bacteria, although their growth rates sometimes overlap.

Parasites

Parasites are the most diverse of all micro organisms. They range from unicellular amoebas of 10 to 12 micrometer to multi cellular tape worms of 1 meter long. The individual cell plan is eukaryotic, but organisms such as worms are highly different and have their own organ system. Most worms have microscopic egg or larval stage, and part of their life cycle may involve multiple vertebrae and invertebrate host. Most parasites are free living, but some dependent on combination of animal, arthropod or crustacean host for their survival.

PAPER - I
Part - B

Chapter-4

NIDANA PANCHAKA VIGYANA

1. Difference between Roga and Rogi Pariksha

परीक्ष– प्रमाणै:अर्थावधारणंपरीक्षा। (वात्सायनभाष्य)

The thoroughknowledge gained from pramaanas through examination and investigation of disease and the patient is called as pareeksha

• ज्ञानपूर्वकं हि कर्मणां समारम्भंप्रशंसन्ति कुशलाः (च.वि. ८)

A physician must have knowledge, good skill about examination of patient and analyzing the disease for proper diagnosis. Then only he can treat the disease properly.

• रोगमादौपरीक्षेततोअनन्तरंऔषधं (च.सू. २०)

The first and foremost step is to examine the patient and study the disease form in the patient, diagnose the disease and then start the treatment on the basis of pareeksha.

• व्याधेस्तुएषुपरिज्ञानंवेदनयाश्चनिग्रहः (भै.र.)

Vyadhi is identified by its clinical features expressed in the patient. A physician having complete knowledge must examine the patient before treatment.

• हेतौलिङ्गेप्रशमनेरोगाणाम् अपुनर्भवे ज्ञानंचतुर्विधंयस्यसराजार्होभिषक्तमः। (च.सू. १९)

A physician is called as a royal physician he who knows the knowledge of etiology, symptomatology of a disease and its respective treatment.

परीक्ष्यास्तु प्रयोजनं प्रतिपत्तिज्ञानम्।
प्रतिपत्तिर्नाम यो विकारो यथा प्रतिपत्तव्यस्तस्य तथानुक्तम् ।। (च.वि. ८/१३२)

Pareeksha helps us to gain pratipatti jnaana. In Ayurveda for particular disease they have mentioned particular utpatti (Nidana), lakshana or presenting features. Thus gaining knowledge about that and planning for treatment according to it is called as