LITERARY REVIEW
REVIEW OF PREVIOUS WORK DONE

Research work done in various institutes

In various Research Institutes of Ayurveda nearly 90 researchers had done work on Medoroga with special reference to its Kriya, Nidan, chikitsa etc. points. Out of them 12 researches are done by researchers of department of Dravyaguna. They are as follows:
**Table No. 1**

<table>
<thead>
<tr>
<th>S/no.</th>
<th>Year</th>
<th>Author</th>
<th>Subject</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>1991</td>
<td>Shah Meeta</td>
<td>A study on Lekhaneeya Dashemahi WSR to Haridra and Chitraka</td>
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<td>2</td>
<td>1994</td>
<td>Parmar R.M.</td>
<td>Study of Katushigru for its Lekhan Karma in the management of obesity (sthoulya)</td>
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<td>3</td>
<td>1995</td>
<td>Thakar (Ms)S.M</td>
<td>Pharmaco therapeutic study of Apamarg and Nirgundi in Medo vriddhi.</td>
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<tr>
<td>4</td>
<td>1997</td>
<td>Savant Rita</td>
<td>A comparative pharmacotherapeutical study on Apamargabija and Vidang in sthoulya.</td>
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<td>7</td>
<td>1989</td>
<td>Lal Kannnaiya</td>
<td>Katipaya medoghna Dravyon ka Medohar Prabhava ka Adhyayan.</td>
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<td>8</td>
<td>1997</td>
<td>Garg K.C.</td>
<td>Katipaya Lekhaniya Dravyon Ka Sthaulya roga ke paripeksha me gunakarma tmak Adhyayayan.</td>
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<tr>
<td>9</td>
<td>1987</td>
<td>Jane P.V.</td>
<td>Effect of Rassanajana Ghanawati in obesity.</td>
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<td>11</td>
<td>1990</td>
<td>Mishra M.C.</td>
<td>Medorog Ka Visheleshanatmak Adhyayan Evam Navak Guggulu Ka medorog par prabhav</td>
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<td>12</td>
<td>1995</td>
<td>Kulkarni Sadhana</td>
<td>Clinical study at Medoroga with Ayurvedic therapy.</td>
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</table>
Out of these 5 researchers had taken the reference of Lekhaniya Dashemani or Lekhan drugs. Mrs. Meeta Shah –Kotecha in 1991 at I.P.G.T. and R.A. Jamnagar had done her work on study of Lekhaniya Dashemahi with special reference to Haridra and Chitrak.

Her study was proved a good guide light for this study. She assessed the Lekhan Karma of Haridra in comparison with Chitrak, based only on weight loss. Criteria and study duration was also small i.e. 45 days and only on 19 patients result shown in this table.

In present study the duration was long as well as patient number was large and criteria are more.

**Table No. 2**

<table>
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<tr>
<th>Scholar</th>
<th>Drug and groups</th>
<th>No. of patients</th>
<th>Duration of treatment</th>
<th>Dose of treatment</th>
<th>Result</th>
<th>Conclusion</th>
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</thead>
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<tr>
<td>Shah and Sharma (DG) 1991</td>
<td>Lekhaniya Dashemahi W.R.T. Haridra Chitrak</td>
<td>19</td>
<td>45 days</td>
<td>19 m g.d.s ½ gm. g.d.s.</td>
<td>Weight</td>
<td>Both drugs possess lekhana property chitrak was more effective than Haridra</td>
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</table>
REVIEW OF HARIDRA

History of plant:

The majority of medicinal plants known today are documented over 300 years back in literature of Ayurveda. Vedas the oldest existing literature of the Hindus give us mixture of information regarding morphological description and therapeutic uses of plants.

In Athervaveda one of the synonym of Haridra i.e. ‘Rajani’ indicates its color used in depigmentation of skin and graying of hair and also the another meaning of Rajani according to its use in day to day life. But its reference have been not found in Rigveda, Yajurveda and Patanjal Mahabhashya. Some scattered reference were found in Bhramhan granthas, Paninya Ashtadhayi and Vartik as per shown in the table no.8.

Haridra is a very important herb in Indian Ayurvedic Medicine. A symbol of prosperity, it was considered as cleansing herb for the whole body. Medicinally, it was used as a digestive aid and in the treatment of fever, infections, arthritis, jaundice and other liver problems.

References from the classical books of Ayurved (Samhita Era) reveal that Haridra is good blood purifier and complexion promoting antiseptic agent. It is used in the formulations used in the treatment of diseases like Kushtha, Prameha, Kamala, Pandu, Arsha, Shotha etc.

Sushruta has described Haridra under Kushtha, Shleshma samshaman, Vrana, Grahani and Arshadhikar. Vagbhata in Ahtang Hridaya has used Haridra in the treatment of various diseases and various conditions.

After Samhita period, several nighantus were composed by different scholars giving details of the drugs of vegetable origin. In most of nighatus Haridra is mentioned under “Oshadhi Varga. They have tried to explain the drug in detail along with its taxonomical descriptive testing in the form of symptoms, pharmacological properties and therapeutic uses.
In modern era the writers like along with many National & International Writers Kirthikar Basu, P.V. Sharma, Bapalal Vaidya etc. have explained Haridra in detail and classified by using binomial nomenclature.

In the present study amongst 4 species of curcuma, Curcuma longa, Linn is used as “Haridra”.

**Classification – Vargeekaran :**

For easy identification of the drugs, Acharyas of Ayurveda have described the drug under different classification. Though the classification started during Samhita period in which the drugs have been classified on the basis of its source, properties, actions etc. In Nighantus more scientific approach has been adopted for classification.

The classification of ‘Haridra’ according to different Samhitas has been completed in the table.

**Table No. 3: Vargeekaran of Haridra (Samhitokta Ganas)**

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<th>Gana</th>
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<tr>
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<td>Charak Samhita</td>
<td>Lekhaniya, Kushthaghna, Vishaghana, Prajasthapan , Shirovirechana, Vat- Samshaman, Shleshma - samshaman</td>
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<td>2</td>
<td>Sushruta Samhita</td>
<td>Shleshma-samshaman, Kushthaghna, Haridradi, Lakshadi Mustadi, Ratapravartak, Arshapatan, Prameshaghn.</td>
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<td>Ashtang Hridaya</td>
<td>Haridradi, Mustadi, Arshoghna, Kandughna</td>
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<td>Ashtanga Sangraha</td>
<td>Hridradi, Mustadi, Arshoghna, Kandughna</td>
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Table No. 4:- Classification according to Nighantus:

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<td>Pippalyadi , Paribhadradi</td>
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<td>Madanpal</td>
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<td>Kiiyadeva</td>
<td>Oshadhi Varga</td>
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<td>Dhanvantari</td>
<td>Guduchyadi</td>
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<td>7</td>
<td>Shaligram</td>
<td>Ashtavarga</td>
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</table>

Classification according to taxonomy

The dravya Haridra morphologically is from Zinziberaceae family hence must have to know Morphological characters of Zinziberaceae as follows

Habitat : Herbs often large, frequently with a pseudostem of convolute leaf sheaths (rarely with a woody caudex).

Leaves : Radical or cauline, usually membranous sheaths generally large , clasping the stem, lamina with a strong central nerve and pinnate close secondary nerves ; petioles short or O.

Flowers : Hermaphrodite rarely 1 sexual (MUSA) irregular.

Inflorescence : Solitary or Spicate, bracts membranous or herbaceous; bracteoles membranous or O.

Perianth : 2 seriate, superior; outer segment 3 calycine (rarely petaloid ), free and imbricate or connate in an entire toothed or spathaceous tube; inner segments petaloid staminides.

Limb : 3 partite, the segments free or connate.

Stamens : Only 1 perfect, the rest replaced by petaloid staminodes or 5 perfect with a sixth imperfect or obsolete.
Anthers : Linear, 2 celled, (rarely of one cell on the margin of a petaloid connective.)

Ovary : 3 (rarely 1 or 2 ) celled, inferior .

Ovules : Many (rarely few) anatropous axile (rarely parietal).

Style : Usually entire or subentire.

Fruit : A loculicidally 3 valved capsule or indehiscent and membranous or fleshy, usually crowned by the remains of the perianth.

Seeds : Often arillate ; albumen flouky ; embryo small. General about 50 – throughout the warm regions of both hemispheres.

**Curcuma Linn**

Morphological Characters :

Habitat : Stemless herbs with tuberous rootstock bearing sessile and longsetipitate tubers.

Leaves : Usually ablong often very large, flowers in dense compound spikes, vernal or aerival and preceding or autumnal and contemporaneous with the leaves crowned by a coma of enlarged colour bracts.

Bracts : Lower bracts ovute, membranous, enclosing several bracteolate fugacious flowers which open in succession.

Calyx : Short, cylindric, minutely toothed.

Corolla : Tube funnel shaped; corolla-lobes usually ovate or oblong, the upper longer and somewhat concave.

Stamen : 1 perfect ; filament short.

Anther : Not ceased, with contiguous cells spurred at the base, lateral staminodes oblong.pataloid, connate with the filament, Lip orbicular with a deflexed lip.

Ovary : 3 celled
Haridra Curcuma longa

Haridra - Curcuma longa, Linn.
Ovules: Numerous on axile placentas.

Stigma: Filiform.

Stigma: 2 lipped, the lips ciliate.

Fruit: A tardily dehiscent globase membraneous 3 valved capsule.

Seeds: Ovoid or oblong usually arillate. Species 35 Palaeotropics

Characters: The rhizome is aromatic stomachic and carminative.

**Cucurma aromatica**

Habitat: Large rootstalk, of palmately branched, sessile annulate bienniaall tubers yellow and aromatic inside.

Leaves: 38 – 60 by 12-20 cm, oblong-elliptic or oblong lanceolate, caudate acuminate, green often variegated above pubescent beneath base deltoid petioles as long as longer than the blade. Flowering spike lateral, apert from and usually aerival.

Flowers: Fragrant, shorter than the bracts spikes 15-30 cm long.; flowering bracts 3.8 5 cm long ovate, recurved, cymbiform, rounded at the tip, pale green, connate below forming pouches for the flowers.

**Curcuma Caesia:**

Morphological Characters:

Habitat: Whole height about 1.2 m.

Leaves: 30-60 by 12.5 – 15 cm broadly lanceolate or oblong, glabrous with a a deep ferruginous purple cloud down the middle which penetrates to the lower surface.

Petiole: Petiole and sheath about as long as the blade.

Spike: These appearing rather before the leaves, about 15 cm or altogether about 30 cm long with the peduncle.
Coma : Deep bright red tending to crimson.

Flowers : Pale yellow, reddish at the outer border rather shorter than their bracts.

Uses : It is used in the fresh state like turmeric. Its roots are used as a rubefacie.

Common name : Marathi : Kalihaladi
              English : Black Zedeory
              Bombay : Narakachura

**Curcuma amada :**

**Morphological characters :**

Habitat : Large rootstock sessile tubers thick cylindric or ellipsoid pale yellow in yellow inside.

Leaves : Long – petiolate, in tufts the blade 30-45 by 7.5 –12.5 cm, oblong lanceolate, acute or acuminate, narrowed to the base, glabrous and green on both sides.

Petioles : These are as long as the leaf blade.

Flowers : In autumnal spikes 7.5-15 by 3.8-5 cm in the centre of the tuft of leaves.

Peduncle : 15 cm long or more.

Flowering bracts : 2.5 long, greenish white, Bracts of the coma longer and narrower, tinged with pink or red.

Calyx : Nearly 13 mm long, abtusely 3 toothed.

Corolla : White or very pale yellow, tube about 2.5 cm long, lobes oblong, acute.

Lip : Semielliptic, yellow, 3 lobed, the middle lobe emarginate.
Character and uses:

1) The rhizome is sweet, bitter, cooling, appetiser, alexiteric, antipyretic, aphrodisiae, laxative, causes “Vata” useful in biliousness, all kinds of itching and skin diseases, bronchitis, asthma, hicough, inflamations due to injuries.

2) The roots are expectorant and astringent useful in diarrhoea and ghee.

Common name: Marathi: Ambahaladi

Hindi: Amahaldi, Kapura Haldi

English: Mango, Ginger

Taxonomy of Haridra (Curcuma longa Linn)(Ayurvedic Taxonomy)

Synonyms of Haridra:

In Ayurvedic classical text, the pharmacological details about drugs are not available as such, instead several synonyms are used to describe the drug which themselves are self explanatory regarding the morphological characters, pharmacogastical properties and therapeutic uses of drug. Synonyms of Haridra as given by different texts as listed in the table No.5.

Etymological analysis and Interpretation of the synonyms:

1) Synonyms denoting medicinal properties of Haridra:

(a) Krimighna – It is antihelminthic in action, which subsides worm infestation.

(b) Mehaghni – Specially coated that it is a drug of choice in the treatment of Prameha.

(c) Vishaghni – Destroys the toxins and purifies the blood in the body.

(d) Varnavati, Varnada, Varnapradayini, Varnavilasini, Varyarnini these all gives colour to any cloth as well as skin. Hence used as dying agent for cloths and increasing complexion.
2) **Synonyms describing the whole plant**

a) Haridra – Yellow colored plant which spreads green color.

b) Nishakhya – It looks beautiful in moonlight.

3) **Synonyms describing morphological characters**

a) Habit - Bhadrada - Holly climber like shrub

b) Habitat - Vanvilasini - Grows wildly in forests

c) Roots - Romashmoolika - Having hairy roots

d) Pindaharidra/Pindabhadra – To differentiate from Daruharidra it is named as Pindabhadra by Kaiyadev. It means roofs are cylindrical/tubular.

3) **Color – Synonyms describing Root Colour**

a) Pita, Pitangi, Gauri, Varangi Roots have attractive yellow colour.

b) Suvarna, Kanchani – Roots are golden yellow in colour.

5) **Religious Significance**

a) Pavitra – Holly plant or used in holly events (daily pooja karma)

b) Mangalya – Laxmi, Bhadra

c) Subhaga – Roots powder is auspicious and used for religious purposes.

6) **Other**

a) Hattavilasini – It attracts towards itself and enhances beauty of the market.

b) Yoshitpriya, Shreevallabha, Shobhana Like by all women and is used as cosmetic (udarrtana) enhances the complexion and color of skin.

c) Vaishya – it sells in daily market.
## Table No. 5: Synonyms of Haridra:

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**Varnacular Names:**

- English: Turmeric
- Hindi: Haldi
- Marathi: Halad
- Gujarathi: Halder
- Bengali: Haldi
- Punjabi: Haldar, Halja
- Telegu: Pusupu
- Kannad: Haldi
- Malayalam: Kooneit - Manjal
- Tamil: Harjal - Manjal
- Bombay: Halad

**Taxonomical Classification:**

- Kingdom: Plant Kingdom
- Division: Phenerogame
- Subdivision: Angiosperms
- Order: Scitaminaceae (zingiberaceae)
- Genus: Curcuma
- Species: longa (Linn.) Linn.

The nomenclature of curcuma xanthorhiza Roxb. has been renamed as Curcuma longa (Linn.) Linn.

In the present study Curcuma longa (Linn.) syn Curcuma xanthorhiza Roxb has been identified and used as Haridra.
Habitat

Commonly found everywhere especially on black, fertile, porous soil in 20°-35° C temperature and hot dry climatic conditions. Rain fall 70-250 cms is needed. Cultivated in abundance all over India, but more in Maharashtra.

India is a largest producer of Curcuma. In Maharashtra. City Sangli is an important marketing center. When stored in basement it is well preserved for longer time.

Taxonomical Description : Curcuma longa linn.

Habit : A tall herb 2-4 ft in height.

Root stock : Large ovoid with sessile cylindrical tubers orange coloured inside. Appearance looking like zinger tubers.

Leaves : Very large, in tufts up to 1.2 m or more long, including the petiole which is about as long as the blade, oblong lanceolate tapering to the base, greenish yellow in colour.

Flowers : In autumnal spikes, 10-15 cm long penduncle 15 cm or more, concealed by the sheathing petiole.

Bract : 3 in no. ovate membranous enclosing several Bractiolate fugitive flowers which open in succession. Spikes 10-12 cm long 5 cm in dia flower bracts pale green. Those of cornia tinged with pink flowers as long as bracts pale yellow.

Calyx : Short cylindrical sepals free and imbricate whitish obtusetry toothed, sepals are 3 in nos.

Corola : Petals are 3 in nos. & joined to form funnel shaped corolla tube.

Stamen : Forming petaloid staminodes (3+3)

Ovary : 3 celled, many ovuled, inferior.

Style : Filiform stigma, 2 lipped, lips ciliate

Fruit : Capsule, ellipsoid seeds are rare.
Rhizome: The primary rhizomes are ovate or pear shaped and are known as bulb or round turmeric while the more cylindrical secondary lateral rhizomes are about 9-7 cm long 1-1-5 cm long. The latter are known as finger and contain more yellow colouring matter than the bulb variety.

Varieties: We find three varieties of Haridra mentioned only in Nighantus. Bhavaprakash Nighantu, described 3 varieties of Haridra.

Table No.6 –Varities of Haridra:

<table>
<thead>
<tr>
<th>Varieties</th>
<th>Bhavaprakash Nighantu</th>
<th>Kaiyadev Nighantu</th>
<th>Shaligram Nighantu</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Haridra</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>2. Amragandhi Haridra (Karpura Haridra)</td>
<td>✓</td>
<td>✓</td>
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</tr>
<tr>
<td>3. Vana Haridra</td>
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</tbody>
</table>

Types according to Bhavprakash

1) Haridra: (Cucurma longa linn)

Paryaya 1-

Haridra, Kanchani, Pita, Nishakhya, Varvarmini, Krumighni, Haldi, Voshitpriya, Halta Vilasini

Guna Karma 2 –

Katu and Tikta rasa, Ruksha, Ushna Virya, Kafa – pitta nashak, Varnya.

Vyadhignata:

Twak – dosha, Prameha, Rakta vikar, Shotha, Pandu
2) **Karpura Haridra (Cucuruma amuda Roxb)**

Paryaya^3^  
Amahara Darvibheda, Amragandha, Surabhi Daru, Daru, Karpura, Padmapatra, Sarim, Surtarka.

**Guna Karma^4^**  
Shit, Vatakarak, Pitta nashak, Madhura and Tikta rasa.

**Guna Karma^5^**  
Kushtha and Vata Rakta Vinashini

3) **Van Haridra (Curcuma aromatica Sallish)**  
Kushtha & Vata Rakta nashak.

**PROPERTIES:**

In Ayurvedic classics the pharmacological action of the drugs are explained on the basis of Rasa, Guna, Virya, Vipaka and Prabhawa.

Rasapanchak

Guna : Ruksha, laghu  
Rasa : Tikta, Katu  
Virya : Ushna  
Vipak : Katu

**PROPERTIES:**

In Ayurvedic classics the pharmacological action of the drugs are explained on the basis of Rasa, Guna, Virya, Vipaka and Prabhawa.
Table No. 7:- Properties & Action of Haridra:

<table>
<thead>
<tr>
<th>Ref</th>
<th>Rasa</th>
<th>Vipak</th>
<th>Virya</th>
<th>Guna</th>
<th>Laghu</th>
<th>Doshghnata</th>
<th>Action</th>
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Actions of Haridra

Formulations and Preparations:
Haridra Khanda, Kalyanakawaleha, Pathyadi Kwatha, Panchanimba, Churna, Dashamool taila, Marichadya taila, Vridhihara lepa, Vyoshadi Saktu etc.

Substitute and Adulterants:
Curcuma longa is rarely adulterated or substituted. However finger print profile using TLC and GLC can distinguish the drug form other species of Curcuma. Haridra is a substitute of Daruharidra.

Trade and Commerce:
Indian turmeric (Madras variety) is selling in UK market for Rs. 27,000=00/tonne. The domestic and export consumption of turmeric is around 41 lakh bags and total crop production in 1997 was just 35 lakh bags. Turmeric grown in Kerala is called Alleppey finger turmeric (AFT). It commands a records price of Rs. 3,600 per quintal as against the Sangli city variety (Price of Rs. 2,500=00/quintal)

Retail market price of Rhizome powder Rs. 80=00/kg.
**Propogation and cultivation:**

Turmeric is planted during June to August. The land is prepared to a fine tilth by cross ploughing and harrowing and 20 tonnes of FYN is incorporated with the soil.

The land is divided into flat beds of convenient sizes. Sandy loam soil is more suitable to this crop. Rhizomes are used as planting material and about 500kg is required for one hectare. Rhizomes are plated 7 cm deep at a distance of 30 cms X 45 cms. A fertilizer mixture of 300 kg urea, 850 kg super phosphate and 400 kg muriate of Potash is added to the soil per hectare for better yield. Depending on the weather conditions the crop is irrigated at weekly intervals. Inflorescence are removed as and when they appear. The crop is ready for harvest after 8 months of planting. When the leaves and stems are dried crop is harvested. Light irrigation is given before digging. Rhizomes are dug without any injury thereafter. From 100 kg of raw rhizomes, 25 kg cured product can be produced. It can be micro propagated through tissue culture technique by inoculating sprouting buds (collected in the month of April or May) on MS medium with 10% coconut milk + 0.2 mg /IBAP + 0.1 mg /IKh.

**Collection**

When its leaves become yellowish and dried the rhizomes should be collected.

**Storage and Preservations**

The primary and secondary rhizomes are dug up and it should be boiled and dried in shed before storage and stored in closed conditions.

**Therapeutic Uses:**

Haridra has been mentioned as a single drug as well as in combination with other drugs in different forms in various diseases.

Actions Haridra: Haridra having external & internal actions.
Local application of turmeric and anti inflammatory, analgesic and complexion enhance. It cures skin disorders has wound cleaning and healing properties. Turmeric smoke relieves hiccups, lower respiration and also relieves pain caused due to scorpion bite. It is very strong in nature. Its paste acts as an antidote. Those who develop cough and other respiratory disorders by drinking milk can be treated by turmeric.

**Internal Actions : It includes following eight actions.**

1) **Nervous system**: Analgesic by Ushna veerya. In case of injury a mixture of turmeric and jaggery administered orally reduces pain and promotes circulation.

2) **Digestive System**: Turmeric is better in taste, appetizer, laxative, chologogue and anthelmintic. Because of these properties it is used for treating loss of appetite, hepatitis, constipation, ascites and worms. There is a wrong belief that turmeric causes jaundice or it is harmful in jaundice.

3) **Circulatory System**: Turmeric stimulates blood formation, circulation and it is also homeostatic. Turmeric is useful in treating anemia, bleeding disorders and other blood diseases.

4) **Respiratory System**: Because of its tikta and tiksha properties turmeric is useful as an expectorant, Kapha (mucous) developed due to drinking milk and other aliments due to Kapha can be treated with turmeric. Inhalation of turmeric smoke reduces kapha.

5) **Urinary System**: Turmeric is a good amtiuretic but this action is carried out by digestion of ama, Kapha and meda. Turmeric should be used in the form of decoction or powder in prameha. Bhasma of tin riturated with turmeric is an effective for prameha.

6) **Reproductive System**: Haridra khanda pak is administered in postpartum period because of its purifying effect on uterus and breast milk. Turmeric is also useful in Shukrameha.

7) **Skin**: Turmeric is used in several skin diseases. It improves skin complexion. Itching and skin eruption due to sheetapitta can be treated with turmeric. It is also useful in pruritus and urticaria.
8) Temperature: Because of its pittashamak and amapachak properties, turmeric is useful in high fever.

9) Satmikaram: Turmeric reduces and helps in treating weakness.

USES OF HARIDRA IN CLINICAL TEXT:

Charak samhita:

1) Ingrediant of Mustakdi kwatha used as blood purifier in skin diseases (CHS/SU.ST23/12)

2) Haridra churma is one of the main ingredients of Vyoshadi Saktu in the treatment of Prameha and Sthaulya (CHS/SU.ST23/19)

3) Ingredient of preparation with old ghee forming Maha Tikta Ghrita used in Kushthadhikar (CHS/CHI.ST 7/145)

4) Haridra is one of the main constituent of Triphaladi yog used in chikista of Kamala (CHS/CHI.ST 16/19)

5) Single drug used of churna for bahualepan in Kushtha. (CHS/SU.ST 3/3, 8,14)

6) Single drug used Kwatha for Kaphaja Prameha. (CHS/CHI. 6/27)

Sushruta Samhita:

1) Ingredients of Gutikanjan used in Drishtigata Roga (SHS/US.ST.17/27)

2) One of the main ingredients in Manshiladi Anjan useful in Netra kandu ptergium as it has antipuritic property.

3) Mostly Susrutacharya mentioned in if in the treatment of Neterroga and manasroga along with Daruharidra as eg.:
   - Amanushopsarya Pratischedha (SHS/US-60/44)
   - Apsmara Pratischedha (SHS/US/ST-60/44)
   - Main ingrediant Panchagavya Ghrita Apsmar Pratischedha (SHS/US ST 61/33)
Because of its Jwarghna action it is useful in Jwaraghna formulations. (SHS/CHI – ST 38/229,38/234)

**Vagbatta (AHS):**

1) Used as single drug Churna in Shirovirechana karma (AHS/SU.ST.15/4) Malapachan Krma (AHS/SU.ST 15/40)
2) Ingradiant of SaktuPanak for the treatment of Atishaulya, Hridroga, Kamala and mental disorders. (AHS/SU.ST 14/29)
3) Ingradiant of Vasishtha Haritaki in Kasa Chikista (AHS/CHI.ST 3/8)
4) Used in Grahani Chikitsa with Daruharidra, Bhunimba and Kutaki in the form of Kshara. (AHS/CHI.ST.10/57)
5) Used in treatment of Udar with Daruharidra, Hingu, Krishnajeerak in the form of Kshara (AHS/chi.ST.15/73)

**Sharangdhar Samhita**

1) Ingredient of many Kwatha as –
   a) Punernavadi Kwatha – in the treatment of Pandu, Kasa, Udar, Shwas, Shotha (S.S 2/76)
   b) Phalatrikadi Kwatha – Prameha nashak (S.S 2/109)
   c) LagnuManjeeshthadi Kwatha – Vata Rakta Pama etc. blood disorders (S.S 2/136)
2) Ingredient of many Guti Vaties as –
   a) Mandur Uatak – Kamala, Pandu, Prameha, Urustambha, Arsha, Shotha, Kushtha (S.S 7/34)
   b) Chandraparabha Vti – Prameha, Medorog Arsha, Mutravikar etc. (S.S 7/HO)
   c) Yograj Guggulu – Prameha (S.S 7/56)
3) Ingredient of many Siddhaghrita and Tailas –2
   a) Maha Pancha Tikta Ghrita – Kushtha, Pandu, Hridrog, Arsha etc. (S.S 9/50)
   b) Kasisadi Ghrita – Vicharchika, Pama, Kshudrarog etc. (S.S 9/51)
   c) Phalaghrita – Striroga (S.S 9/87)
4) Ingredient of Asawa -
5) Ingredient of Devdaruyadi Arishta –
Prameha, Kushtha, Grahani, Arsha (S.S 10/62)

**Bhava prakash**

In this Samhita Haridra is mentioned under most of the Rogadhikar some are of them as

1) Prameh Pidakadhikar - 38

Haridra along with Daruharidra and Honey is useful in sandra meha (B.P.2 - 38/45)

2) Main ingredient in most of formulations as for the treatment of Prameha pidaka.
   - Phalatrikadi kwatha (B.P.3 - 38/59)
   - Trikatukadya Modak (B.P.3 - 38/62)
   - Gokshura churna vatika (B.P. 3 - 38/82)

After Charak Samhita, Vaghbhatta Samhita only Bhawamishra mentioned Haridra in the treatment of sthauthya. Some of the e.g. are as follows

1) Main ingredient of Vyoshadi Saktu (B.P.3 - 39/53)

2) Triphaladhya Taila (B.P. 3 - 39/58)

3) Medolekhan Udvertana (B.P.3 - 39/82)

**USES AND AMAYEEK PRAYOGA**

- Juice of fresh rhizome is applied to revent wounds, buries and bites. Internally it is used as an anti helminthic.

- Root is usually administered in intermittent fevers. In doses of 15 to 20 grams twice a day it is given for flatulence, dyspepsia and weak state of stomach it is used both externally & internally in skin diseases due to impurity of blood.

- A paste of turmeric and leaves of Justicia Adhatoda with cow’s urine rubbed on the skin in several other combinations of the root are in vogue such as turmeric and nim leaves, turmeric and the ashes of the plant tree etc. Turmeric is also given internally with cow’s urine in eczema. Mixed with gingelly oil it is applied to the body to prevent skin eruptions.
• Turmeric paste mixed with a little lime and salt, applied hot is a popular application to sprains, bruises, wounds and inflammatory troubles of the joints.

• In small pox and chicken pox a coating of turmeric powder or thin paste is applied to facilitate the process of scrubbing and decoction of turmeric is applied as a lotion to relieve the burning in catarrhal and prevent opthalmia. Popularly known as country sore eye or conjunctivitis. A piece of rag soaked in it and kept constantly over affected eye relieves the burning and moderates the urgency of the symptoms.

• Its powder is sprinkled on ulcers to stimulate healthy action.

• Tubers sold in the market for dietary purposes are boiled and are on no account used for dyeing.

• Turmeric for dyeing is sold separately and Indian women use it to smear their hands and faces with and is called in Tamil "Kappumanial"

• Manual of Tail industries Ghee mixed with powdered turmeric is given to relieve cough.

• A paste of turmeric alone or combined with the pulp of neem leaves is used in ringworm, obstinate itching eczema and other parasitic skin diseases.

• In piles an ointment made of turmeric, hemp leaves, onions and warm mustard or linseed oil gives great relief when the piles are painful and protruding also effective in eczema itching etc.

• In pemphigus and shingles the part first smeared with a thick coating of mustard oil and ten dusted on with turmeric powder is cured within ¾ days.

• In catarrh and corrhysa the inhalation of the fumes of the burning turmeric from the nostrils causes a copious mucous discharge and gives instant relief the fumes are also used to relieve hysterical feats. The inhalations taken at night and no fluid is allowed for some hours afterwards smoke produced by sprinkling powdered turmeric over burnt charcoal will relieve scorpion sling when the part affected is exposed to the smoke for a few minutes.

• Turmeric and alum powder in the proportion of 1 to 20 is blown into the ear in chronic discharge.
• With borax as a paste it is applied to reduce insolvent swellings. It is given in urinary diseases.

• Milk boiled with turmeric rhizome added to it and then sweetened with sugar is a popular remedy for cold.

• Internally turmeric is given in infections of the liver and in jaundice.

   Good Digestive: Turmeric + long pepper + Ginger + Cardamom + 10 g each powder and black pepper powder 5 grain. Mix well and make a compound powder. Following confections is highly recommended in obstinate skin complaints – HaridraKhand.

   Turmeric 6.4 tolas clarified better 48 tolas milk 16 seers, sugar 12 tolas and boil them together over a gentle fire in an earthen pot.

   Then add black pepper, long pepper, ginger, cinnamon, cardamom, tejapatra baberrang seeds root of Ipomea, Turethum the three myrobalans, flowers of Mesua ferra tubers of cyperus rotundas and prepared iron each 8 tolas in fine powder and prepare a confection. Dose: One tola every morning in prurugo, and chronic skin eruptions.

   Bhaishajyarotnavali – A cure is affected in seven days.

   Tests: Goods turmeric should be of reddish orange appearance when broken or cut into two and should have a moist feeling.

**MICROSCOPICAL DESCRIPTION**

   In transverse section rhizome consists of cortex an endodermid ring closely covering.

   In transection rhizomes consist of outer zone of cork followed by wide zone of cortex an endodermoid ring closely covering vascular bundle are scattered throughout section.

   Parenchymatus cells of cortexd and pith are full of starch grains yellow pigment being present at some places only.
In uncured sample cork is intact through while in cured sample it may be broken at many places. Cortex of rhizomes in demarked into two zones. Outer cortex contains few layers of irregular shaped rounded parenchyma full of starch grains while inner cortex which is large has parenchyma full of starch grains set selatinised to form compact mass in each cells.

Vascular bundles present in cortex have been called as cortical vascular bundle leaf trace bundles or cortical meristetes. There bundles are of closed type consisting of xylem and phloem only. There is no fibrous size zone above vascular bundles. These vascular bundles measures 80-115-240 u along their major axis, vascular bundle present towards, outer side are smaller having 3-7 xylem present element.

While those present towards inside may be comparatively bigger having up to 12 vascular bundles may be commerced with each other.

Vascular bundles arranged along circle under endodermoid ring. Ring enclosed central medulla in which are also scattered numerous vascular bundles. Those vascular bundles resembles cortical vascular bundle in all respects.

Starch grains of turmeric are oval. Circular elliptical some of grains have back like protrubrance. They are simple with exentric hilum and measure 19-2040 u along major axis. Vessels elements are transversely perforated having wide pits. There may be circular or angular. They are 40-52-80 u long. In one of samples crystals of calcium configuration measuring 10-10-22 u along their major axis.

In unstained section deep yellow lustrous spot seen scattered through out section. These probably consists of Curcumin yellow pigment of turmeric if an alkaline solution is put on section these spots turn brown.

**Phytochemistry :**

Turmeric has an aromatic odour and a warm somewhat bitter test.

Turmeric contains about 5% of diaryl heptanoid colouring materials known as curcuminoinds the chief of which is curcumin.
Together with smaller quantities of dicafeoyl methane and coffeocyl feruloye methane. Dihydo curcumin was reported in 1980. The volatile oil (5%) contains sesquite perns, sesquiter pene alcohols and ketones and monoturpenes.

In a series of recent papers Japanese workers reported on the characterization of the constituents of a polysaccharide fraction of the drug which has a marked immunological activity. The new acid glycon designated ukonan A,B & C show remarkable reticulo endothelial system (RFS) potentiating properties Ukonan A for e.g. is composed of L- arabinose, D-xylase D- Glucose L- rhamnose D-galarturonic acid is additional to small amounts of peptide.

Ukonan D, a neutral polysaccharide also shows RES potentiating activity as indicated in a carbon clearance test, it has as estimated molecular mass of 28000 and is composed of L-arabinose D- Galactose D- Glucose D-manose in the molar ratio 1:1:12:2 + small peptide fraction.

The rhizomes also contain free arbonose (1%) fructose (12%) and glucose (2%) abundant zingerberaceous starch grains, 30-60 mm long and often gelatinized are present.

Curcumin fluroscence is a broad band in acetonitrile (max= 524 nm), ethanol (549 nm) or micellar solution (557 nm) but has some structure in toluene (460,488 nm)Curcumin produced singlet oxygen upon irradiation (>400 nm) Chignell 1994.

The arabinogalactose and beta – 1-4 linked D- xylose. All of the galactose units in the backbone carry side chains composed of beta –1, 6 linked D- galactosyl residues Gonda 1993.

Four Curcuminoids were ineffective when applied independently but nematocidal activity increased remarkably when they were mixed, suggesting a syneric action Kiuchi 1993.

Ukonan D, MW 28,000 is composewd of L-arabinose : D-galactose: D-galactan type and alpha –4, 6-branched type structural units Gonda 1992.
Turmerin a water soluble 5-kD 40 residue peptide is stable to trypsin and pepsin, heat and UV. Ames assay indicates it is noncytotoxic up to milligram concentration Srinivas 1992.

Capillary GC –MS identified 9 sesquiterpenoids (Curcumene, artutmerone, xanthorrhzol, germacrone, sesquiphellandrene, curcuzerrenone, turmerone) 3 cucurminoids and a monoterpenoid (camphor) Uehara 1992.

Germacrone from Curcuma xanthorrhiza is anti-inflammatory in rats Ozaki 1990.

Curcumin photodecomposition products and half lives Tonnesen 1986.

Many chemical constituents were obtained and characterized by IR, NMR and mass spectral studies. Some chemical constituents are –

Alpha turmeric Alpha pinene, alpha Curcumin, 2 hydroxy 0 methyl anthraquinone, 4 hydroxy – cinnamoyl – methane, Rh, 4 – methoxy bisabola Beta, Sitosterol, Beta turmenone, Borneol, Caffeic acid, Camphor Rh 60, cholesterol Rh, Cineol Rh, 2.92%, Curcumene, Curcumenol, Rh, Rt. Eugenol, Rh 0.21%, 34.5, para cymene Terpineol . Rh. Eo 0.05% Turmerin Ta Turmeronol A, Rh 282, Turmerone, Rt, Rh e0 27%, Vinillic acid, Ef unsaturated fatty acids Rh, zedorohdiol, Rh 35, Zingiberene, Rh E0 8.14% Rt.

**Curcumin molecule**

![Curcumin molecule](image)

R₁ R₂ — Compound

R₁ OCH₃  R₂ OCH₃ Curcumin

R₁ OCH₃  R₂ H Demethoxycurcumin

R₁ H  R₂ H Bis-demethoxycurcumin
**Adverse Effects and Toxicity:**

0.2 or 1% of turmeric ethanol extract for 14 days to mice showed hepatotoxicity. Mice are more vulnerable to turmeric-induced hepatotoxicity than rats Deshapnde 1988.

Dietary turmeric (0.2%, 1.0%, 5.0%) or ethanol extract (0.05%, 0.25%) for 14 days, at doses reported to be cancer preventive, were found to be hepatotoxic in mice coagulative necrosis and a zone of regenerating parenchymal cells Kandarkar 1998.

Occupational allergic contact dermatitis due to curcumin food colour in a pasta factory worker Kiec-swierczynska 1998.

Liver enzyme effects by 2% curcumin in the diet of female maize for activities double while EROD (preferentially catalyzed by P450 1A1 decreased Singh 1998)

Allergic contact dermatitis from Curcumin (turmeric) Hata 1997.

Acute dosages of 0.5, 1.0 and 3g/kg body weight and chronic dosages of 100 MG/KG/DAY OF ETHANOLIC EXTRACTS OF THE curcuma longa rhizomes caused poor weight gain, changes in heart and lungs weights fall in the WBC and RBC levels Qureshi 1992.

Allergic contact dermatitis to curcuma longa (turmeric) Goh 1987.

Turmeric oleoresin to pigs at 60, 296 and 1551 mg/kg 102-109 days increased weight of the liver and thyroid at all doses. The highest dose group had poor weight gain, pericholangitis, thyroid hyperplasia and epithelial changes in the kidney and bladder Bille 1985.

Adverse events reports at the FDA’s Centre for Food Safety and Applied Nutrition (FDA)

Toxicity of rhizome in rats: guinea pigs and monkeys were reported. Ether extract of rhizome was highly cytotoxic, toxicity higher than those from known curcuminoids LD 50 value of aqueous suspensions of volatile oil was found to be 3.25 ml/kg Cytotoxic effect of curcuminoids have been observed in cell culture.

Turmeric or its alcoholic effect administered in 2.5 gm/kg and 3000 mg/kg respectively on different species of animals proved nontoxic.
1] Structure of Curcumin

Activity
Anti – HIV
Anti Inflammatory
Anti Oxidant
Anti – tumor

2] Structure of Ar – tumerone:

Activity
Smake bite

3] Methyl Curcumin

Activity
L.

4] Demethoxy Curcumin

Activity
Anti oxidant
Experimental Study and Clinical Trials:

Clinical Studies –

1) Abortifacient effect:

Hot water of dried root taken orally by pregnant human was inactive. A mixture of the following was given in the form of a decoction to a number of pregnant women. No toxic effects were noted. Dosing was 3 times daily for three days. The mixture contained Angelica sinesis (root), Ligustium wallichii (root), Prunus persica (seed), Larhamus tinctorius (flower), paeonia obvata (root), Acharthes bidentata (root), Leonurus sibiricus Caerial parts), Lycopus Lucidus var, Hirta (Leaf) and curcuma longa (root), and campsis grandiflora (flowers)(CS-I)

2) Allergenic Activity –

Commercial sample of rhizome powder was active on human adults. Reaction to patch tests occurred most commonly in patients who were regularly exposed to the substance, or who already had dermatitis on the finger tips. Previously unexpected patients had few reactions (i.e. root irritant reactions) (CS-II)

3) Antiasthmatic Activity

Dried rhizome taken orally by human adults at a dose of 250.0 mg person was active. Administration of 26 (11 male and 15 female) patients with Bronchial asthma once daily for three weeks. No side effects were observed. The preparation also contained Glycyrrhiza glabra. A dose of 6-12 gm / person daily for 15-20 days was active. One hundred seven patients with tamak swasa vatapradhan (chronic Bronchitis or Asthma) ages 31-50 had fair good response. (CS-3)

Experimental studies (pharmacological studies) –

Extracts of turmeric in petroleum ther, alcohol water significant anti-inflammatory activity in both exudative and proliferative inflammation.

All the 3 extracts showed presence of steroids and it is likely that they are responsible for anti inflammatory properties.
1) In Carragenin induced oedema 80 mg/kg of water extract almost completely suppressed inflammations ES²

2) In Granuloma pouch method the water extract was most potent and its activity was similar to hydrocortisone.

3) In cotton pellet best petroleum ether extract was most potent and activity similar to indomethacin. ES¹

4) Garg –et-al (1974) reported that petroleum ether and aq. Extracts showed 10% anti inflammatory, anti fertility activity at dose of 200 mg/body weight in rats.

5) Alcoholic extracts of rhizomes showed anti protozoal activity against Entamoeba histolytica.

6) Oil extract shows anti microbial effects on some bacteria and fungi including plant and human pathogen

7) Adrenal Hypertrophy Effect : water extract of dried rhizome together with a mixture of Levisticum officinate Artemisia capillaris and Chrusanthemum indicum was active when administered to mice. ES¹

8) Anti Bacterial Activity : Chloroform ethanol (95%) water and petroleum ether extracts of dried rhizome at a concentration of 250.0 mg/ml on agar plate were active on Bacillus Subtilis Escherichia coli, Pseudomonas aeruginosa and Staphylococcus aureus ES² Ethanol (95%) extract at a concentration of 10.0 mg/ml was inactive on Cornebacterium diptheriae, Diplococcus pneumoniae. Staphylococcus aureus, Streptococcus viridans and Sterptococcus pyogens.

   Water extracts at a concentration of 70.0 mg/ml was inactive on Corynebacterium diptherige and Diplococcus pneumoniae and produced weak activity on Staphylococcus aureus, Streptococcus viridans and Sterptococcus pyogenes. ES³ Essential oil of rhizome on agar plate was inactive on Bacillus cereus, Escherichia coli, Pseudomonos aeruginosa and Staphylocosuus aureus ES⁴

   Ethanol (95%) extract of rhizome in both culture was active on Lactobacillus acidophilus and staphylococcus aureus; equivocal on Escherichia coli and inactive.
on Salmonella typhosa ES. Undiluted essential oil on agar plate was inactive on Bacillus cereus. Escherichia coli, Psedomonas geruginosa and staphylococcus aureus ES. Water and hot water extracts of dried rhizome on agar plate at a concentration of 0.5 ml/disc was inactive on Bacillus Subtilis. H-17 (REC+) and H-17 (REC). Rhizome on agar plate at variable concentration was active on Bacillus Subtilis. H-17 (REC+)

9) Anticoagulant activity- Chromatographic fraction of dried rhizome administered intra peritonally to mice at a dose of 0.08 gm/kg was active. Results significant at p<0.05 level. Ethylacetate extract of dried rhizome administered intraperitonealy to mice at a dose of 0.1 gm/kg produced strong activity. Results significant at 0.01 level. The water extract at a dose of 0.1 gm/kg was equivocal ES.

10) Antiedema activity: Methanol extract of dried rhizome administered to mice at a dose 2.0 mg/ear was active Vs 12-0-tetrade canoylphorbol -13-acetate (TPA) induced ear inflammation. Inhibition ratio (IR) was 71. ES.

11) Antihyperglyceridemia effect: Ethanol / Water (1:1) extract of dried rhizome administered intragastric to rats a dose of 30.0 mg/gm (dry weight of plant every six hours for 48 hours was active Vs triton induced hypercholesterolemia ES.

12) Antihyperlipidemic activity: Ethanol / Water (1:1) extract of dried rhizome administered intragastric to rats at a dose of 30.0 mg/gm (dry weight of plant) every six hours for 48 hours was active Vs triton induced hypercholesterolemia ES. Ether and ethanol (95%) extract of rhizome, administered by gastric intubulation of rabbits at a dose of 1.0 gm/animal were inactive Vs cholesterol loaded animals ES.

13) Anti-ischemic effect: Rhizome administered intragastric to rats at a dose of 5.0 gm/kg was active on the heart. The dose also contained nicotinic acid. The dose was given daily for seven days during the last two of which isoproterenol was also given. Isoproterenol induced ischemic, effects on the heart were prevented ES.
14) Platelet aggregation inhibition: Ether extract of dried tuber at a concentration of 100.0 mg/ml was inactive vs collagen and ADP induced aggregation and used as ionophore vs calcium ionophore induced aggregation. A concentration of 50.0 mg/ml was active vs arachidonic acid induced aggregation. ES* Water extract of dried rhizome was active on the platelets of dried rhizome was active on the platelets of human adults and rabbits. The dose consist of a mixture of Levisticum officinale Artemisia capillaris. Cucurma longa and Chrysanthemum indicum ES

<table>
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<tr>
<th>Sr.No.</th>
<th>Vaidic literature</th>
<th>Total Plants</th>
<th>Synonym of Haridra</th>
<th>Synonym of Daruharidra</th>
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<tr>
<td>1</td>
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<td>67</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
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<td>Yajurveda</td>
<td>82</td>
<td>x</td>
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<td>Haridrava</td>
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<td>Yaskkrit Nirukta</td>
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</table>

Table No.8:- Compilation of Haridra and Daruharidra in Vedic literature.
REFERANCES OF CLINICAL & EXPERIMENTAL STUDIES OF
HARIDRA

CS –1
Li, F.K. Problems concerning artificial abortion through oral administration of

CS –2
Seetharam K.A. and J.S. Pasricha condiments and contact dermatitis of the

CS –3
Shankara, M.R, N.S.N Murthy and L.N.Shastry, method of manufacture and
clinical efficiency of ramsamanikya mishrana in tamakshwasa (bronchial asthama).
Indian J.Pharm Sci. 1979 : 41 :267 B

CS –4
Jain, J.P.; L.S. Bhatnager and M.R. Parsai Clinical trials of haridra (Curcuma
longa) in cases of tamak Swasa and kasa.. J Res Indian med Yoga Homeopathy 1979
: 14(2) : 110 –119.

CS –5
of osteoarthritis with a herbomineral formulation a double blind, placebo – conrolled,

ES –1
Liu, T.G. Hypolipemics and blood platelet aggregation inhibitors comprising

ES –2
Sankaranarayanan, J.and C.I. Jolly Phytochemical, antibacterial and
pharmalogical investigations on momordira charantia linn. Emblica officinalis
ES-3


ES-4


ES-5


ES-6


ES-7


ES-8


ES-9

ES –10


ES –11


ES –12

Gupta S.S.; D. Chandra and N. Mishra anti inflammatory and anti-hyaluronidase activity of volatile oil of curcuma long. Indian J. physical Pharmacol 1972; 16 :263A

ES –13


ES –14


Liu, Y.G. Hypolipemics and blood platelet aggregation inhabitants comprising fish oil and plant extracts. Patent –Us-4-842,859 : 1989; 6PP.
हरिद्रा - आवाप्रकाशोक प्रकार

हरिद्रा
हरिद्रा कांचनी पीता जिसका उज्ज्वल झढ़ी हुआ होता है।
कृतिज्जीबाबू दोविद्यिया हट्टे बिलास्निती है।
हरिद्रा कटुका लिखा कष्टकर्ण एक विशिष्ट विचरनुपूर्वक।
वर्णवाच्य तत्त्वविशेषलेखकानुमानानुसार ज्ञात नहीं।

कपूर हरिद्रा
कर्मालोकान्त आज्ञालेखा च नृपस्वरूप वर्णाश्रम च।
कमुलसुधा पद्माप्रक्ष्या ्व्यासचुन्नी प्रस्तुतात्मक।
आज्ञानिपर्वत हरिद्रा च नृपस्वरूप वर्णाश्रम ।
विशेषज्ज्ञानुसार - लिखा वर्णकण्ठाद्विविनाशी।

वन हरिद्रा
अपरणाहलोकेवः कुछवाता ज्ञातानां।

हरिद्रा

राजप्रकाश - विपल्लादिवर्ण
पदार्थ
हरिद्रा हरिद्राभरो स्वरापितार्ण नूतनं जिसमा वाणिज्यी शीर्षकाना।
हरिद्रा च पीता सहारो-च नृपस्वरूप चाली चाली पवित्रम् ॥ १ ॥
हरिद्रा संज्ञानेज्जीविपर्वती वाणिज्यी।
विनः वर्णराच्छै मृदुल्लमण ज्ञाता च नृपस्वरूप ॥ २ ॥
वर्णराच्छै भवं भिन्ता स्वतं भोजन्मुक्ता।
श्यामाजज्ज स्वतं क्रिस्मं जीवितानन्दी। ॥ ३ ॥

गुण
हरिद्रा कुटुर्यादेशोपाध्याय किंवतवातापूर्वक।
जेहकुटुर्यादेशोपाध्याय हरिद्रा देखवारविषालिनि। ॥ ४ ॥

केन्द्रदेव विपल्लाद - अोपसिधिर्वर्ण
पदार्थ
हरिद्रा वाणिज्यी नृपस्वरूप पीता नामानुसारिकोः।
विनः हरिद्रा वेश्या कालीस्वरूप नृपस्वरूप ॥ ५ ॥
विनः भोजन नृपस्वरूप चाली वाणिज्यी।
कृतिज्जीविपल्ला वाणिज्यी विनः स्वानुसारिकोः। ॥ ७ ॥
गुण
जित्रा तिक्रा कटु कुशा वण्योऽणा कठकयितवा ।
घण्डु मण्यायणी में हुमे वेंक पिलिवा नोक्षितनं। ॥१५॥

शोकलिपिप्रणत् - गुणवादविवर्ण
पर्याय
विज्ञासां ब्रजरी नौरी च ब्रह्मचर्यानि।
भगवता जुमाल्लन्न हरिंका हरिता तथाः ॥३॥
विषयानि च वदवली च पिलाव वार्कसान्निती।
वेदविनी धीर्यसाणा च जैव वर्ष प्रवाहिता ॥३४॥

धवलकृतिप्रणः - गुणवादविवर्ण
पर्याय
हर्षविलासिका विज्ञा नौरी नन्मिति दिखा।
नौरी वर्धवति नौरा हरिता वर्धविःपन्नि। ॥५.३॥
हलविज्ञा भर्गवता जोधा वर्धविलासिति।
विषयानि च वदवली च धीर्यसाणा तु वर्त्तविनी। ॥५.३॥

गुण
हर्षविलासिका वर्धवते तिक्रा क्रोणच्या विष्णुहतुर।
कहृतुकुशनाथप्रजा वेदविलासिति। ॥५.५॥
विशृष्टिनी कृत्तिका ब्रजाचलविलासिति।

मवनपाल प्रणः - ओऽवधी वर्ग
पर्याय
हरिन्द्रा ब्रजली नौरी ब्रह्मचर्यानि।
विषया नौरा वर्धवति विज्ञा वर्धा किलासिति। ॥

गुण
हरिन्द्रा कटुका तिक्रा क्रोणच्या वेदविःपन्नि।
स्वर्णया लक्ष्मणेनांशोभणापाणुवाणप्पपा। ॥

शालिभाषाल प्रणः - अत्तर्कः
पर्याय
हरियन्द्राभावाः भद्राः वर्धवनी विकारसिति।
कारावेड़कावाणी जोधानां ब्रह्मचर्यानि। ॥

गुण
हरियन्द्राकृतितात्रस्वर्णनाथप्रकाशवालावलु।
स्वर्णनिवर्धमेनांशोभणापाणुवाणप्पपा। ॥

45
अभियान गंगाजी

ओरी हरिद्वार महत्त्वपूर्ण पीठ घुणा व घुणा ये लाभ हो और काफी च।

श्रीसुन्दर कृष्णात्मको प्रथमा विशालत्वार्थ च रजिततीति।

भगवानु : रामाकर

हरिद्वार गुण

हरिद्वार कल्याणका वेदवर्णविवरणिका।
उपयोग कन्या स्थानी ने कन्याय विशुद्धवण गता।
करण वांग राजगोपदे कुंज करणू प्रमोहन।
प्रारूपों च प्रयों नरकान नायकानुम नक्षत्रव विषय।

पीलग चारविनियुक्त प्रतियुक्त वामवथे।

राजमहं

हरिद्वार कारकविरहितो कारणूको धर्मार्थिती।
पाण्डुशोभायी वैय जेहुकुब्रुणयपह।

हरिद्वार पवार-1

पवार

कृष्णात्मक - कृष्णात्मक (भ.प.)
मेहसती - मेहसह चोले विश्वेश्वर प्रभास। मेहसहसती इति विधं प्रष्ट।
विषयमें - विषय हरितीति।

वर्णविवरणीती - वर्ण विपुलविवरणित प्रकृतित विषयीति।
हरिद्वार - हरी हरि वर्ण हरिनकालाभ मार्गत अनुसारवतीति, 'वा कुमारावां गति.'

तिलकाबुदा - तिलक राजसाथि वामाक्षित्वाहत।

लोकात्मक - लोकं गृहसाता।

विघुणकिनिता - विघुणकिन घुणकिन कारण घुणकिन (भ.प.)

गृहसाता - गृहसाता (भ.प.)
कृष्णात्मक - कृष्णात्मकास्त विघुणकिन "घुणकिन." दत्त च यपति।

पिता - पीतावर्ण कल्यान पीत गृहस; च।

कौंते - कौंतेय- (भ.प.)

मांगलय - मांगलस्तनिकाल्पनें कुरुचे विद्वानाचार्यात।

हरिद्वारकिनिता - (भ.प.)

हरिपाणां कीमार्यविवरणिका विश्वार्थिती।
'वैश्या' (कै.प.) इत्यादि गृहसाताचार्य: कुशालवत।

लोकात्मक - (भ.प.)

लोकात्मक कीमान नियाय, औपचरिकमें प्रयोजनता।

वर्ण - (भ.प.)

यमावर्ण वर्णार्थिति, वस्त्रार्थिक गते पुदीत्वार्थि।

46
उपयोग

चक -प्रमेह
लोक्यं तुक्षाकाव्यं हरिक्षम्।
पितेश्वर वसोजाताः की फलताः॥

चुरुल - कुक्कुल
पीता मांसं वा फलस्या हरिक्षम्।
गुप्तरागाः पापहोनस्य नयनेतृ॥

वामपट - कणोदयुग तुष्णा
अर्थ पितेश्वर वज्जया वा नियुक्तं सहीकर्ष्यं॥

चक - श्रीपद
चक्षु शुद्धं तुतृतृतृतृतं नोगूरः विभेजय।
शोकाल विचिन्द्रु - कंठु।
हरिक्षालक्ष्म तुतृतृतृ लोगूरवन्य पचस्यं।
विभेजय: कांगवायं कंठु: भावालेन॥

सप्तिन्द्र
हरिक्षा तु हितेन्द्रायां ताम्भयो भास्तिन्द्र: कचित्।
अवकम्पू विघटानां। ब्रम्हायां प्रयोजित॥

बंगलेन - जेपुरशर्करस्वयं
त: पितेश्वर शबदी बशोकु मनोहराः तुष्णाय।
तरस्यं विचक्षण, उषा वाध्यं जेपुराष्ट्रस॥

श्रीतलिखितयाज
विश्वास्तेन सहितां चार्यमी प्रियम्याः।
से श्रीतलिखित सहितां सम्म विक्रय्यत॥
तेन्त्रें अभिज्ञर तदहि श्रीतलिखित शरीरे।
कार्य विवरः प्रमणजो तुष्णायाः वाततस॥

वैयानाधिपता - वातमालाबाधिन
तुल्य सिद्ध: वात्मनावोपशाखाय।
वायुपुर्वत: भारेतीरुलुमुर्वता॥
सिद्धोशोधकत्र तेसर्षः हंसेन्द्राणवानश्वर्तः तापतजन्तिवतश्च॥

रूसरकरस्वयं - गणेश: व्याख्यातयाज
- श्रीरथीक्षरसं पिठे बर्तीनाभि: वन्तः
कृतदीयं विजयायाः श्यातिकाण वाणनो:। द्वितारण॥
BERBERIS ARISTATA D. C.
Daruharidra - Berberis aristata, D.C.

Barkwood - Berberis aristata, D.C.
REVIEW OF DARUHARIDRA

History of the Plant:

Ancient man believed that plants are gods gift to humanities, for the preservation of health, prevention and treatment of disease. Thus history of plant is needed to study. Man’s struggle against disease pain and premature death and can be traced to the remote past.

The majority of medicinal plants known today are documented over 3000 years back in literature of Ayurveda. Vedas the oldest existing literature of Hindus gives us mixture of information regarding morphological description and therapeutic uses of the plants.

In Yajurved one of the synonym of Daruahridra i.e. “Pita daru” is found. The description about “Pita daru” is again denoted in “Snatpath Brahman” grantha is, its stem is having perfume and it is “Tejas” so burns fast. It is useful to make “Paridhi” of Yagya. Sayana, in his commentary on Atheraveda knotted that pitadaru is “Udumbar Vishlesha” Kashara in his sutra denotes that Haridra and Daruahridra pariksha should be used on Khalitya (Allopecia areta). (see for ref.table no.8)

References from the classical books of Ayurved reveal that “Daruahridra” is an important drug used in Netrarog, Kamarog and Twakrog. In charak Samhita Daruahridra is one of the ingredients in Mandurvatak, Punerneva Mandur for the treatment of Pandu, Kushtha, speenomegaly etc. In Sushruta samhita Daruahridra is classified and described under Tikta Varga. Sushruta has described Daruahridra in many formulations used mainly in the treatment of Netrarog in the form of Rasanjan (Bhadrodayanjan, Gutikanjan, Manshilanjan etc.)

Vagbhata in his Ashtang Hridaya has used Daruahridra in various conditions like Nasyavidhi, in Garbhavyapad or in Arsha, Atisar, Grahani, Prameha etc. diseases.

After samhita period Nighantus have tried to explain the drug in details along with its synonyms, uses etc. but generally properties and actions are described as Haridra.
In modern are the writer like P. V. Sharma, Bapalal Vaidya etc. have explained Daru haridra in detail and classified by using binomial nomenclature.

**Contravancies about Daru haridra**

Daru haridra is an important drug used in Ayurveda, the true identity of which has become obscure over a long period in the History of Ayurveda. Many plants have been used as substitute in its place. Daru haridra being a controversial drug, we find many plants used as Daru haridra in different parts of India.

Berberis is official in many countries of the world. About 40 species of Berberis are used in the world. Out of which 12-13 species found in India. Three to four species are found in Himalaya. In Nilgiri ranges also some species of Berberis are found.

All species seem to have similar therapeutic properties. Their barks and stem are bitter tonics. The fruits of Berberis Vulgaris is official in France.

"By Indian medicinal plant there are 3 well defined medicinal groups.

1) The rocky mountain group, including B-aquifolium.

2) The Asiatic group which includes B-aristatata DC.

3) The European group which includes B-Valgaris Linn.

The species which are used as Daru haridra in India are as follows:

1) Berberis petiolaes (B-vulgaris var vulgaris proper Hoof and Th. In Hook) Local name – chachar, which is used in Punjab and found in Western Himalaya from Kashmir to Nepal.

2) Berberis lycicum Royle - It is found in western Himalaya (Gadhawal to Masuri) local name is Kashmal.

3) Berberis asiatica, Roxb ex. DC local name e-Kilmore, Kinora chitra found in the vallies of Bhutan, Gadhwal, Bihar, Pasasnath.

It is used mainly for preparation of Rasanjan and its fruits are selled in local market called “Zarishka”.
4) **Berberis aristata** DC Local name Kushmoi. This species is mainly found in all ranges of Himalaya on 6000 to 10500 ft. height also in ranges of Nilgiri. It is mildly used in the treatment and its fruits are also called “Zarishk” and its bark and stem is very hard and lemon yellow in colour and very bitter in taste.

5) **Coscinium Fenestratum** : Local name – Maramanjalu

In Ceylon and South India Coscinium fenestratum (Menispermacea) is used as Berberis or Daruhaldi. It is used in South India and called “Malbari Daruhaldi.”

It is climbing shrub. Branches covered with fuscous cinerous bark, striate, when young yellow to mentose Petiole 10-12 cms long, Lmina – Shining and smooth above, minutely to mentose beneath.

Inflorescence – Supra axillary, finely to mentose,

Bisexual – Female flowers staminode 6, ovaries 3-6 style subtutate, Drups – globose 1-3 about 2.25 cm in diameter.

Names – Jhadhaldi, Jarki haldi (Deccan), Ceylon calcumba root (Eng) Marmanjalu (Tamil), ValliHaridra (Sanskrit).

**Conclusion on the identity of Daruharidra**

**The Nighantu Daruharidra**

With the scanty information available, Berberis aristata could be suggested a possible source. This is with reference its being a yellow coloured small tree having throns. (Pitadaru, Kantkateri and Kutajvat Patrayi Bhavprakash)

**Bappalal Vaidya and P.V. Sharma** :

Menispermaceae and Berberidacea are neighbor families so therapeutically both many resemble each other. But the real Daruharidra has a very hard wood, intensely yellow colour and a small tree. White coscinium is a soft woody climber with very little yellow colour.

This reference should be clearly borne in mind. The vaidyas of Kerala and other parts of south India are using the coscinium. Fenestratum as Daruharidra. Rosot
or Rasawanti the extract from the Berberis species is used in medicine. Species is used in medicine. In Rasot prepared by B.aristata and B.asiatatica Berberine percentage is very high. But extract of Coscinium (Mar-Manjalu) havn’t so much and it is medicinally less useful.

Hence it is concluded that B-aristata should be used as Daruharidra by morphologically therapeutically and phytochemistry.

**Classification (Vargeekaran):**

The classification of “Daruharidra” according to different Samhitas has been complied in table

**Table No.9: Classification According to Samhita Grantha**

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<th>Sr.No.</th>
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<tbody>
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<td>1</td>
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<td>Ashtang Sangrah</td>
<td>Shirovirechan, Arshoghna, Kandughna, Haridradi, Mustadi</td>
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<td>Sushruta Samhita</td>
<td>Haridradi, Mustadi, Lakshadi, Tiktavarga</td>
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<td>4</td>
<td>Ashtang Hridaya</td>
<td>Tiktagnana, Haridradi, Mustadi, Arshoghna, Kandughna</td>
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</table>

**Table No.10: Classification According to Nighantus:**

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<th>Sr.No.</th>
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<td>Pippalyadi</td>
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<td>3</td>
<td>Dhanvantari</td>
<td>Guduchyadi</td>
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<td>4</td>
<td>Kaiyadeva</td>
<td>Oshadhi varga</td>
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<tr>
<td>5</td>
<td>Sodhal</td>
<td>Guduchyadi</td>
</tr>
<tr>
<td>6</td>
<td>Madanpal</td>
<td>Oshadhi varga</td>
</tr>
<tr>
<td>7</td>
<td>Shaligram</td>
<td>Ashtavarga</td>
</tr>
</tbody>
</table>
**Taxonomical Classification**

1. **Family: Berbidaeae**

**Morphological Characters:**

Habitat : Clabrous herbs or shrubs, sometimes climbing

Leaves : Simple or compound rarely stipulate.

Flowers : Hermaphrodite or rarely diclinous, regular, axillary, solitary or in simple or compound racemes usually yellow or white.

Sepals : Often petaloid, 3-9 in 1-3 whorls.

Petals : Equal in number to the Sepals twice as many and like them caducous.

Stamens : 4-8 usually 6 opposite to petals.

Filaments : Free or connate.

Anthers : Bursting by two apical valves or longitudinally.

Carpels : 1-3, rarely more, distinct.

Stigma : Usually peltate.

Fruit : Berries or capsules usually indehiscent, ovoid to oblong ovoid, bright red, slightly pruniose blue styllose.

**Genera 12, species 200 N temperate regions, tropical mountains, S.America**

**Character :** Rot and bark generally purgative berk bitter, tonic and antiperiodic or depurative antiscorbutic.

**Chemical contents :**

The following are among the products isolated 1) alkaloids – berbamine, berberrine, oxyacanthine, 2) resins – Podophyloresin, podophyllotoxine, 3) Colouring matter podophyllaqueretin, 4) Acids – malic, citric
2. *Berberis (Tourn) Linn*

**Morphological Characters:**

- **Habitat:** Excrete shrubs with yellow wood.
- **Leaves:** Simple, alternate or fascicled in the axis of 3-5-7 partite or rarely simple spines entire or more often spiny toothed.
- **Flowers:** Small, yellow, solitary fascicled or in bracteate simple or compound racemes.
- **Sepals:** 6 petaloid, imbricate in two whorls.
- **Petals:** 6 imppricate in two whorls usually with 2 glands inside at the base.
- **Stamens:** 6, free dehiscing by assending valves.
- **Carpel:** 1.
- **Fruit:** a berry, blue or red.
- **Species:** 190 N.hemisphere, S. America

About forty species of Berberis are used medicinally and they all seem to have similar therapetical properties. Their stems and barks are bitter tonics and mild laxatives.

The following deserve special mention in Europe B-aetensis r and S: B-Vulgaris Linn, in North America B-aquifolium purs, Bnervosa Linn in Central America – B trifolitus Moric

Berberine has been isolated from B.aetnessis, B.aquifolium, B.aristata,, B.glauc, B.nervosa, B.valgaris, Beramine and oxyaccanthine are contain the root barks of B.aquifolium and B. Vulgaris

3. *Mahonia Nutt*

*Berberis nepalensis* Spreg Sysy II ; 120
**Morphological Characters:**

**Habitat**: An evergreen shrub, 1.2-3 m high with sparingly branched erect stems up to 20 cm diameter.

**Bark**: Pale brown, rough and corky

**Leaves**: Pinnate, 18-45 cm long, approximate at the ends of the stout twigs.

**Leaflets**: Usual 7-17, 3.8-10 cm long, ovate or lanceolate, acuminate, base of lateral pairs very oblique, Margin coarsely and sharply spinous toothed, coriaceous, glabrous shining above with 3-5 basal nerves prominent beneath.

**Flowers**: Yellow, 5-5.6 mm long sweet scented, in dense erect racemes 5-12.5 cm long fascicled at the tips of the branches.

**Pedicles**: 1.25-4 mm long berries 5-10 mm long ovoid blue-black glaucous style distinct.

**Habitat / Distribution**:

Temperate Himalaya, 4000-8000 ft from Garhwal of Bhutan, Khaisa Hills, 4000-5000 ft. Mergui, Nilgiri Mts 5000-8000 ft.

**Use**: The berries are considered as diuretic and demulcent in Dysentry.

**4: Berberis Asiatica**

**Morphological Characters**:

**Habitat**: An evergreen shrub, 1.2-18 m high and stem up to 10 cm diam.

**Bark**: Rough, furrowed and somewhat corky.

**Twigs**: Glabrous or shortly pubescent, pale yellowish.

**Leaves**: 2.5-6.3 by 1.3-3.8 cm oblong, elliptic or broadly obovate, usually with large distant spinous teeth, sometimes entire, very coriaceous, dark green with very prominent primary and secondary pale reticulate venation above, glaucous beneath.
Petiole : O or distinct upto 10 mm.

Inflorescence : A simple raceme upto 3 cm long often with a few long stalked flowers at the base.

Pedicels : 4-10 mm long, slender, often glacous.

Fruit : 7-10 mm long, ovoid, blue black with glacous bloom, style distinct.

5. **Podophyllum emodi**:

**Morphological Characters**:

Habitat : A smooth, succulent erect herb, 4 root stock creeping.

Flowering stem : 15-45 cm high, leafy on the upper portion.

Leaves : 1-3 usually 2, alternate, long stalked, often purple spotted, round, 15-25 cm, diameter deeply divided to the middle or base into 3-5 lobes, which are sharply toothed and often with deep incisions.

Flowers : 3.8-5 cm diam. Solitary rarely 2. cup shaped white sometimes pink, appearing at the same time as leaves.

Sepals : 3 petals like soon falling off.

Petals and Stamens – 6

Fruit : A large scarlet pulpy berry, 2.5-6.3 cm in length, containing many seeds.

Uses : It acts as heptic stimulant and chologogue purgative.

6. **Berberis aristata DC**

This species is selected for present study.

**Synonyms**:

1) Berberis aristata, D.C. syst. Nat II ; 8 1821, Hook F and Thoms

2) B. floribunda, Hook, F.and Th in Hook L.
3) **B. Sikkimensisi** (Schneid) Ahrendt in *J. Bot Lond* 80 (suppl), Banergee in Sharma et. Al FL. India 1:383, 1993, type Sikkim


5) **B. micrantha** (Hook and Thoms) Ahrendt in Gard. Illus 64:426 et in J. Linn Soc.


7) **B. Ceratophylla** G. Den syst Gard 1:15, 1831, Ahrendt in J. Asiatic, Soc. Beng (sci)

**Morphological Characters:**

**Habit**: A large deciduous shrub usually 1 to 3 m high but attaining 4.5 m with stem 20 cm dia.

**Stem**: (Twigs) Whitish or pale yellow, terete, internode 3-6 cm long, thorny

**Spines**: Fairly stout, 1-3 cm, solitary towards apex of stem, 2-3 side at base.

**Bark**: Plae yellow brown, closely and rather deeply sorrowed rough, width, 5-7.5 mm, internally bright yellow with coarse reticulate fibre.

**Leaves**: 3.8-10 cm X 1.5-3.3 cm, obovato to obovate – elliptic, mucronate, at apex Sub acute to acute, base cuneate, margin entire or spinous – toothed, promonent reticulate, veined, glossy dark green above, glossy pale green but not glaceous beneath. Shortly petiolate. Petiole: 3-4 mm long, 'O' or distinct

**Inflorescence**: a simple drooping raceme, 8-25 fid., 4-10 cm long, including peduncle 1-5 cm long.

**Pedicles**: 5-10 mm long.

**Bracts**: 3 X 0.8 mm.

**Flowers**: 11-14 mm in dia.
Outer sepals : 1.5 – 3 X 1 mm, ovate.

Median Sepals : 5-6 X 2-3 mm, narrowly ovate to elliptic.

Inner Sepals : Obovate 5-8 X 3-5 mm.

Petals : 6-7 X 3-4 mm, obvate to oblong – obvate, entire, base cunate glands 1.2 X 0.4 mm, non marginal.

Stamens : 4.5 – 5.5 mm, shortly apiculate.

Ovules : 4-5.

Fruits : Berries, 7-11 X 6-7 mm, ovoid to oblong – ovoid, bright red, slightly pruinose blue, stylose.

Style : 1 mm long.

Distribution

In India Jammu and Kashmir, Himachal Pradesh, Uttar Pradesh, Sikkim, Madhya Pradesh, Tamil Nadu. In Nepal and in Bhutan.

Synonyms of Daruharidra (i.e. B.aristata D.C.)

Synonyms of Daruharidra are self explanatory regarding its morphological structure, pharmacological properties and therapeutic uses. Synonyms of Daruharidra as given by different texts as listed in the table no. 11.

Etymological analysis and interpretation of the synonyms :

a) Synonyms denoting properties of Daruharidra :

1) Krimihara : Its action is antihelmentic. It has Prakritivighatak action.
2) Panchampacha : After intake of this powder, it improves digestion and enhance liver function.
3) Vishodhani : It cleans and purifies the body from inside.
4) Parjani : Who protects from diseases.
5) Darvi : Who purifies the doshas.
6) Kaleyak : Who expels and the vitiated doshas.
7) Kushtharajani : It acts on skin disorders
b) **Synonyms describing the whole plant:**

1) Haridruvaha: The yellow coloured tree.

2) Pitachandan: The yellow tree is like as sandal tree.

3) Daru: Woody tree.

4) Parjanya: Tree has fruiting after 1st rain.

c) **Synonyms describing morphological characters:**

(1) Habit: Pitadaru, Haridra, Pitakduma – Yellow coloured tree.

(2) Leaves: 1) Kantkini – The leaves have throns or spines.

2) Kantkini: The leaves are spiny at their margins.

(3) Flowers: 1) Supushpa: having beautiful flowers

2) Kusumbhak: Flowers like Kusumbha

(d) Stem: 1) Daruwaridra: Daru means stem having yellow colour like turmeric

2) Pitadru (Pitadaru): Yellow coloured stem.

3) Kashtharajani: Stem giving colour to naything.

4) Darvi: The part of plant used as medicine is the bark.

5) Darunisha: The stem has bright yellow colour.

e) Colour: Pita, Pitika, Hemakanti, Sthirranga, Nisha, Swarnavarna – These synonyms describes the yellow colour of stem, bark, root of Daruwaridra.
Table no.11: Synonyms of Daruharidra:

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Vernacular names:

- **English**: Indian barberry, tree turmeric
- **Hindi**: Daruhaldi, Kashmal, Daruhalad.
- **Marathi**: Daruhalad.
- **Gujarathi**: Daruhalad.
- **Bengali**: Daruhalidi. *Baruhradra*
- **Punjabi**: Sumlu, Chitra.
- **Telegu**: Kasturi paspu. *Hanupascalpa*
- **Kannada**: Doddamarad arisina. *Manjariscasina*
- **Malyalam**: Marmanjal, Kasturi Manjal.
- **Nepal**: Chitra.
- **Bomaby**: Jarki hald, Zarishk.

Varieties of Daruhalidra

We find five varieties of Daruhalidra mentioned only by commentaiton on Bhavprakash Nighantu and two varieties by Nighantu Adarsh Bappalal Vaidya in a compendium of Indian medicinal plants by Arya Vaidya shala, they mentioned only Coscinium fenestratum (Maumanjalu) should be used as “Daruhalidra” (Indian medicinal plants.)

**Table no.12: varieties of Daruhalidra**

<table>
<thead>
<tr>
<th>Varieties (Species)</th>
<th>Bhavprakash N. Chunekar</th>
<th>Bappalal Vaidya</th>
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Rasapanchak:

1) **Guna**: Ruksha and Laghu

2) **Rasa**: Tikta and Katu

3) **Virya**: Ushna

4) **Vipak**: Katu

5) **Action on Doshas**: Daruharidra pacifies Kapha and Pitta dosha but not vitiates vata dosha because of its ushna virya in normal doses.

6) **Action on Dhatus**: Many Nighantus describes its Raktashodhak action only Charakacharya mentioned its lekhan karma on Dhatus.

7) **Action on Malas Purish**: Because of Tikta rasa it stimulates liver function hence pittasarak. But because of ushna vrya Malasangrahi Sweda: Increases production of Sweda because of its Kledaghna property.

**Part used**: Stem, bark, roots and fruit

**Fruit** (Zarishka)

**Rasa**: Madhura amla

**Virya**: Shita

**Action of Doshas**: pacifies pitta
### Table no. 13: Properties of Daruharidra

<table>
<thead>
<tr>
<th>Guna→Nighatu</th>
<th>Guna</th>
<th>Rasa</th>
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### Table no. 14: Uses of Daruharidra:

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### Formulations and Preparations:

Rasanjana, Daruyadi Kvatha(Bh.R), Dravyadi leha(Sh.S.), Darvyadi taila(Bh.R), Dashmoola taila, Marichayadi taila, Nagarjuna anjana, Piyushvalli rasa, Sudarshan Churna(Y.R.).
Cultivation, Conservation & Ecology

Protocol for in vitro propagation of B. buxifolia native shrub of Patagonia with emphasis on the rooting stage described. Arena 2000

From the Oligocene Los Ahuehuetes locality, Mexico, five new plant species were described from fossil material that relate it to Berberidaceae based on their leaf architecture. Ramirez 2000

On the knowledge of the Rumanian acclimatized plant Berberis crataegina DC [Article in German] Petcu 1968

Rust resistant berberis, mahoberberis and mahonia plants at Cereal Disease Laboratory

Berberis canadensis (American barberry) info at PLANTS National Database

All Berberis species at PLANTS National Database

Cultivation details

Prefers a warm moist loamy soil and light shade but it is by no means fastidious, succeeding in thin, dry and shallow soils.Grows well in heavy clay soils. Plants are very hardy, they survived the severe winters without problems. Plants can be pruned back quite severely and resprout well from the base. The fruits are sometimes sold in local markets in India. Hybridizes freely with other members of this genus. Most plants cultivated under this name are B. chitria., B. coriaria., B. aristata and, more commonly, B. floribunda.

Propagation

Seed - best sown as soon as it is ripe in a cold frame, it should germinate in late winter or early spring. Seed from over-ripe fruit will take longer to germinate. Stored seed may require cold stratification and should be sown in a cold frame as early in the year as possible. When they are large enough to handle, prick the seedlings out into individual pots and grow them on in the greenhouse or cold frame for at least their first winter. Once they are at least 20cm tall, plant them out into their permanent positions in late spring or early summer, after the last expected frosts. The seedlings
are subject to damping off, so be careful not to overwater them and keep them well ventilated. Cuttings of half-ripe wood, July/August in a frame. Very difficult, if not impossible. Cuttings of mature wood of the current season’s growth, preferably with a heel, October/November in a frame.

**Trade and commerce:**

- **Retail market price:**
  - Bark: Rs. 45 per kg.
  - Powder: Rs. 120 per kg.

**Substitutes and adulterations:**

Cucurma longa (Haridra) is sometimes used as substitute. Berberis lucium Royle and B.aciatica Roxb.ex.DC are also used in medicine as Daruvaridra. In south India and Srilanka coscinium fenestratum Colebr is known used as Daruvaridra.

Daruvaridra is adulterated with shoots of plants like Samudra Shosha after boiling them in decoction of turmeric. The shoots of real Daruvaridra are elastic and donot loose their yellow colour even after boiling.

**Storage**

Should be stored after Varsha Ritu or rainy season. Bark, wood, stem or roots should be stored in the form of powder or ghanasara at low tempreture and in closed container. The Rasanjana and Zarishka should be stored in narrow glass vessel and should be kept in dry and cool place.

**Shelf Life**

- Powder: 6 months to 1 year.
- Rasanjana: Many years.
- Fruit (Zarishka): 1 year.

**Dosage**

- Juice of root: 10 – 20 mls
- Decoction: 50 to 100 ml
- Rasanjana: ¼ to ½ gms
- Fruit: 1 gm in malaria (total) 5 to 10 mg as a stimulant for heart but large dose can lead to cardiac depression.
Churna powder : 3-6 gms

Tincture : ½ - 2 drops

Berberin alkaloid : 1-5 grain

ACTION AND USES OF DARUHARIDRA:

1) Tonic, stomachic, astringent, antiperiodic, diaphoretic, antipyrutic and alternative.

2) Root is purgative

3) Fruit is adish

External Uses:

Shathaghna or reduces inflammation or odema removes pain, cures and heals ulcers, also used for eye diseases. Hence in severe conjunctivitis, the paste is applied around the eyes and 250 mg rasanjana mixed with 20 ml of rose water is used as eye drops (or can be applied in eyelids). This fluid can be applied in the ears in earache or ear discharge. In disease of the mouth and throat. Rsasanjana is used for gargling, Rasanjan can be used to wash wounds or its paste can be applied on ulcers. Chaneroid ulcers, goiter, fistula, crysipelas and other diseases are treated are treated by the application of the paste. Vaginal discharges are treated by the douche prepared from berberis of Rasanjan on perianal wounds.

Internal Uses:

Digestive System:

Appetiser, liver stimulant, cholagogue but astringent. A larger dose is laxative. The fruit is very tasty antidipsetic. Because of these actions, daruharidra is a superior medicine in agnimandya, dysentery jaundice and other liver disorders. The tablet of rasanjan prepared in reddish juice is useful in bleeding piles. It is also effective in diarrhea. Daruharidra formulation gives excellent results in cholera.
Circulatory System:

Blood purifier and haemostatic agent. Daruharidra decoction is given in syphilis and other sexual diseases. Hematemesis, malena and menorrhagia respond only to rasanjan or coagulant preparations + rasanjan. Reduces pus. Daruharidra acts on rakta dhatu. Because of Daruharidra, the inactive forms of malarial parasite in the spleen come out in the blood stream and this is very useful for the diagnostic test for malarial parasite by peripheral smear.

Respiratory System:

It is useful in the treatment of cough being kaphaghna.

Reproductive System:

Useful in uterine inflammations and vaginal discharges.

Skin:

Diaphoretic, useful in diseases of the skin like pruritus boils etc. Daruharidra decoction is useful in cleansing wounds.

Rasanjan + honey is used extremely in puerperal diseases. It is used extremely in puerperal diseases. It is specially useful in ulcer. The decoction is used for gargling in stematitis.

Tempreture:

Febrifuge and diaphoretic and is a prophylactic treatment for thyroid hence it is useful in common fever and also in chronic fever. It causes sweating is antipyretic and is useful in recurrent malaria. When malaria parasites hide in the liver, guinine is not effective but daruharidra is very effective. After administering a mild laxative 1 gm of rasanjan is dissolved in water and given 3 to 4 times in a day. Then the patient is covered with thick clothes and blankets and made to sleep. After a while the patient will feel thirsty but should not be given water. After an hour there will be sweating. The sweat should be wiped clean. Warm milk or sage kheer should be given orally. This preserves and increases the patient’s strength. The fever is reduced and there is reduction in hepatosplenomegaly.

Another Actions:

1) Moderately hot and moist.
2) For enlarged spleen and jaundice, emmenagogue.
3) Useful in mucous dysentery and colitis.
Use in Anemia and Jaundice:

1) Darvighrita

2) One suffering from jaundice should take cooled decoction of triphala, guduchi, darvi and nimba mixed with honey in morning.

Use in Kushtha:

1) Darvi or Rasanjana taken with cow’s urine (Gomutra) cures Kushtha. Similarly acts as haritaki taken with trikatu jaggary and oils a month.

2) Skin disorders (Kushtha)
   - Darvi + Rasanjana
   - Nimba + Patola
   - Khadora (heartwood)
   - Argvadha and Kutaja
   - Triphala
   - Saptapana
   - Tinisa
   - Karvira: these eight decoction should be used in various ways such as bath intake paste rubbing, dusting and proussing of oil ghee for alleviation of Kushtha.

Use in Erysipelas:

In wounds oil medicated with darvi, vidanga and kampillka is useful and in the cases where predominance of kapha and pittaghrita with durva juice is effective.

Use In Dysuria:

In dysuria caused by pitta, Darvi with Amalaki juice mixed with honey should be taken.
Use in Diseases of mouth:

The extract of darvi (rasanjana) with honey effective in the diseases of mouth, disorders of blood and sinus.

Use in Coryza:

smoking should be used with stickes made of Darvi, ingudi, danti, kinti and tulsi is effective in coryza.

Use in Eye diseases:

1) Milk boiled with Daruharidra cooled and mixed with a little rock salt should be used for washing eyes or Santhi rubbed with breast milk and mixed with ghee should used as collyrium.

2) Decoction made of Darvi 40 gm with water 640 ml reduced to one eigth is mixed with honey and used for washing. It is useful in inflammation of eyes caused by all doshas.

3) Eye drop should be used of the decoction of Darvi and Prapaundrika

4) Rasanjana combined with trikatu is made into pills which are rubbed and applied as paste. It destroys anjanmika (stye) associated with itching and inflammation.

5) In night blindness, stick maded up of rasanjana haridra, daruharidra, leaves of jati and hnimba mixed with liquid cowdung is used (as collyrium)

Use in Pradara:

Darvyadi kwatha is useful Shweta and Rakta Pradara.

Use in Poison:

In cases of poisoning paste of Haridra and Daruharidra is used.
Use in Prameha:

Darvi and amalaki juice with honey alleviate.

Some Amayek Prayogas:

Following are few useful formulas:

a)

1) Indian barberry 5 parts Rasvanti (Berberry – extract) 5 parts
2) Cyperus rotandus 3 parts
3) Semecarpus anacardium 2 parts
4) Bael fruit 5 parts.
5) Adhathoda vasika 5 parts
6) Chiretta – 5 parts

Mix and make decoction in the useful way when ready and honey 4 parts.

Dose: ½ to 1 drachm

Useful Leukonhoea, Menorrhagia etc.

b) Indian berberry 5 parts

Honey 3 parts

Oxalis corniculata 4

Mix and make pills

Useful in painful micturation acid urine etc.

c) Rasavanti, Aconitum, Heterophyllum bark of Holarrhena Antidysenterica

Each one part and flowers of wood fordia floribunda 3 parts.

Mix and make powder

• Dose 1 drachm

• Useful in bilious diarrhoea, indigestion etc.
d) Take of extract of Berberry 2 opium 2, Rock said 4 Alum 3 and chebulic Murobalan 2 parts.

Mix and make paste.

Applied locally to inflammatory swelling and as a collyrium for the eyes in conjunctivitis.

e) Take of Rasavanti 5 grains
Kernel of nim seed grains
Raisins 2 grains

Beat all together into a mass and make it into 3 pills

Dose: One pill to be taken at bed time in case of Piles.

f) Take Berberry root 6 ounces and water 2 pints. Boil down to 1 pint. Dose 2 ounces three times a day as a diaphoretic and bitter tonic.

g) The fruits of Berberis (barriers) are given as a mild laxative to children. It is used as an anti periodic and alternative in remittent types of fever.

h) The dried extract of the root known as “Rasaut” or “Rasanjan” used as a purgative for children as a blood purifier and as an external application in conjunctives in combination with opium. As a local application It is used for indolent ulcers.

i) Sharangdhara recommends a simple decoction of Berberis to be given with honey in jaundice. In painful micturation from billus or acrid urine a decction of Daruharidra and amalaki should be given with honey.

j) A decoction of root bark is used as a wash for unhealthy ulcers. (that is said to improve their apperance and promote cautrization). Rasout mixed honey is an usefull application to aphthous sores.

k) A decoction of the root bark in doses of one to two cunces was given to several patients for Malarial fever and was found to be beneficial, the effect at beginning was very slow.
Uses of Daruharidra in Ancient Samhitas

- Charak Samhita:
  1. Ingredient of lepa (pack) prescribed for promotion of complexion and anti-pruritic effect. (Ch.S./Su.St/3-3)
  2. It includes in shirovirechaniya dravyas (Ch.S./Su.ST/2-3)
  3. Ingredient of Kushthadi churna and Haidradi lepa in the treatment of Kushtha (Ch.S./Su.St/3-10,3-14)
  4. In Chikitsasthan Daruharidra mentioned in the treatments of Prameha, Jwara, Kushtha Rajyakshma, Arsha and Manasrog like unmad and Apsmar.

Some examples

Phaltrikadi Kwath for Prameha (Ch. S./Ch.ST/6-40); Triphaladi Churna for loss of sensation in Kushtha (Ch.S./ch.St/7-68) KanaKarishta in the treatment of Arsha (Ch.S./Ch.ST/14-160)

- Sushrut Samhita:
  1. Daruharidra is mainly used by sushrutacharya in the form of Anjana and for the treatment in the form of Anjana and for the treatment of Netrarog.
  2. Medicated ghee with Roots of sugarcane cardamon and Daruharidra etc. is useful in pittabhi syyanda (conjuctivities) by various ways like Tarpah, Shek, Nyasa etc. Daruharidra is the main ingredient of Kasmaryadi anjan, Guti Kanjan, Manhasilanjan, Bhardrodayanjan etc.
  3. It is also used in the treatment of Jwara patoladi gritam (Sh.S./U.T.St 38/229)

Atisara : Dravyadi ghritam (Sh.S./UT.St 40-105)
Visuchika – Vyoshadyanjan (Sh.S./UT.ST 56-44)
Apsmar – Panchgavya Ghrita (Sh.S./UT.ST 61-37)
• **Ashtang Hridaya:**

1. Daruharidra is mentioned along with Haridra in all their references only it includes separately in shodhanadigana i.e. ‘Shirovirechana gana’ (Ash/SU./15/4)

2. Vagbhatcharya mentioned Daruharidra is a one of the ingredient of Asisthaulyanashak yoga which is also useful in the treatment of Hridrog, Kamala and Buddhivardhak. (Ash/Su.ST/14-29)

3. It is one of the ingredient of pittajgrahaninashak churma which is also useful in Pandu and Aruchi (Ash/Ch.St./10-36)

4. It is useful in the treatment of Prameha in combination with Deodaru ad Amalaki (Ash./Ch.S/12-6&7)

   It is one of the main ingredient of Manduvatak for Pandu (Ash/Ch.ST/16-7) MahatiKtaghrita for pittaj MahaKushtha (Ash/Ch. ST/19-8-10), Balataila for Vatajvyadhi (Ash/Ch.ST/22-87) etc.

5. It is also mentioned in the treatment of unmad Apasmar like mental disorders, vartmarog, Akshiorg (eye diseases) Karnarog, Mukhorg (E.N.T) and anti-poisonous treatment.

**Sharangdhara Samhita:**

In Sharangdhara Samhita many Kalpas of Dariharidra are mentioned in madhyam and uttarakhand. Some of them are as follows.

1. **Kwathadi Kalpana (Madhyam Khanda –2)**

   1. Dravyadi Kwath : used in the treatment of leucorrhoea and mehorragia (Shweta and Rakta) Pradar nashak) (2-110)

   2. Phaltrikadi Kwath – For the treatment of Prameha (2-109)

   3. Vasadi Kwath – for Netrarog, Shwas, Pinus etc. (2-146)

2. **Churna Kalpana**

   Sudarshah Churna – In the treatment of Jwara trishna Sarvangashool etc. (6-27)
3. Vatak Kalpaha:
   - Chandraprabha vati: useful for Prameha Ashmari Mutrakricha, Arsha, Kushtha etc. (7-40)
   - Yograj Guggul – Decoction of Darvi used as a Anupah (vehicle) with yograj Guggul is useful in the prameha (7-56).

4. Ghrit Taila Kalpana
   - Maha panchtikta Ghrrita – for Kushtha, Rakarsha Pandu etc. (9-50).
   - Vacha tail (Gandamalanashak (9194)
   - Triphala Ghrita – for eye diseases (9-66)
   - Pathadi taila – for Nasya (9-178)

5. Asavarishta Kalpana
   - Kumari Asava : useful in Prameha, parinam shool, udar etc. (10-20)
   - Khadirarishta – useful in mahakushtha, Pandu Grahadi, Arsha (10-62)

Bhavprakash Samhita:
1. Useful in Tandrik Sannipat (jwara) with Ajamutra by Nasya marga (Bh.P/1-645)
2. Useful in decoction with honey in the treatment of kamala (Bh.P/8-40)
3. One of the ingredients of Mahanarayan taila which is useful in all vata – vyadhi (Bh.P.24-29)
4. Main ingredient of vyoshadhi Saktu which is useful in obesity (Sthoulya) 7 prameha, Pandu etc. (Bh.P. 39/53)
5. Also its external application with other Triphala, Haridra etc. in the treatment of obesity, Pandu is useful (Bh. P.39-58)
6. Externally applied on netra for the treatment of Netraroga (Bh. P. 63/16)
**Traditional and Folk Use**

Berberis aristata is a well known medicinal plant in Iran and has also been used as food. Antihistaminic pA2=4.5 for fruit extract vs. 9.4 for dextchlorpheniramine. Anticholinergic pA2=4.4 for fruit extract vs. 9 for atropine in guinea pig ileum Shamsa 1999

Berberis aristata is widely used in Chinese folk-medicine as leukogenics, anti-arrhythmics and anti-hypertensives. Berbermine suppressed delayed type hypersensitivity reaction, mixed lymphocyte reaction and prolonged allograft survival Luo 1998

Berberidaceae roots have been used in European folk medicine for inflammation. Total ethanol extract inhibited induced paw edema. Berberine suppressed a delayed type hypersensitivity more than oxyacanthine Ivanovska 1996

Use Berberis in place of endangered Golden Seal. Article at Henriette’s Herbal

**Methods of preparation**

**Contemporary Standardized Methods**

Effects of storage time on the berberine content in Berberis amurensis Rupr [Article in Chinese] Dong 1987

Arthritis treatment with Guaiacum, Berberis, Harpagophytum, Rhus-tox, Bryonia and boron; US Patent 4,440,760

Oxyacanthine or Berberis extract in a hair growth promoter; US Patent 5,663,160

**Folk Methods**

Contemporary Formulas -Folk Blends

- Amoebic liver abscess treatment in hamsters by an India folk mixture of Boerhavia diffusa, Tinospora cordifolia, Berberis aristata, Terminalia chebula and Zingiber officinale had a cure rate of 73% at 800 mg/kg/d Sohni 1996
Entamoeba histolytica treatment with an India folk mixture of Boerhavia, Berberis aristata, Tinospora, Terminalia and Zingiber had MIC=1000 micrograms/ml vs. 10 micrograms/ml for metronidazole Sohni 1995

Pharmacognostical & Phytochemical Study:

Morphological, anatomical, and phytochemical aspects were carried out to identify the diagnostic features of B. asiatica root. Some of the diagnostic features of the root drug noted from the anatomical study are patches of pericyclic fiber, pitted sclerids, and berberine-containing cells and heterocyclic medullary rays.

Phytochemistry:

The chief constituent of the roots and stem bark of Berberis aristata is an alkaloid berberine which is reported to be responsible for hepatoprotective activity of Berberis aristata Other constituents including berbamine, aromoline, palmatine, oxyacanthine and oxyberberine are also isolated. Alcoholic extract of the bark of Berberis aristata yielded berberine, berberine chloride and palmatine chloride (yield of the alkaloids as salts, is 4% of dry wt of bark).

Physicochemical studies revealed the presence of total ash 2.650%; acid insoluble ash 0.266%; alcohol soluble extractive 11.833%; water soluble extractive 15.333%; tannins 1.723%; sugar 0.332%; starch 16.444%; and alkaloidal content (berberine) 2.4%. A comparative high performance thin layer chromatography (HPTLC) analysis with B. aristata showed a similar profile. Berberine was identified as the major constituent, with a slightly lower percentage (2.4%) in the former. The R\textsubscript{f} value of other bands was also calculated.

Microscopy of Daruharidra:

1) Powder shows tubular cork cells
2) Sclerenchymatous elements
3) Small yellow fibers with funnel shaped pits a narrow lumen in stands 2 or 3 fibers wide each fiber about 160 um by 15 um few small pitted sclercids.
4) Few isolated starch grains spherical 25-30 um in diagram
Few fragments of xylem vessels, reticulate thick and some with small bordered pits from incompletely removed wood.

**Analytical Chemistry - BERBERIN MOLECULE:**

Berberine is a plant alkaloid with a long history of medicinal use in both Ayurvedic and Chinese medicine. It is present in Hydrastis canadensis (goldenseal), Coptis chinensis (Coptis or goldenthread), Berberis aquifolium (Oregon grape), Berberis vulgaris (barberry), and Berberis aristata (tree turmeric). The berberine alkaloid can be found in the roots, rhizomes, and stem bark of the plants. Berberine extracts and decoctions have demonstrated significant antimicrobial activity against a variety of organisms including bacteria, viruses, fungi, protozoans, helminths, and chlamydia. Currently, the predominant clinical uses of berberine include bacterial diarrhea, intestinal parasite infections, and ocular trachoma infections.

**Pharmacology of Berberine:**

The pharmacologic actions of berberine include metabolic inhibition of certain organisms, inhibition of bacterial enterotoxin formation, inhibition of intestinal fluid accumulation and ion secretion, inhibition of smooth muscle contraction, reduction of inflammation, platelet aggregation inhibition, platelet count elevation in certain types of thrombocytopenia, stimulation of bile and bilirubin secretion, and inhibition of entricular tachyarrhythmias.

8-substituted derivatives of the protoberberine alkaloids palmatine and berberine were prepared and investigated by 1D and 2D NMR spectroscopy. Marek 2003
The contents of carbohydrates, organic acids, some vitamin, poliphenolic compounds, pectin tannin, mineral elements, in berberis aristata are listed and utilization of berberis in traditional medicine and dietet are also outlined-Pozniakovskii 2003

First enantioselective total synthesis of (-)-tejedine a secoisbenzyltetrahydroisoquinoline isolated from Berberis vulgaris reported. Wang 2002

Berberine can be monitored selectively and sensitively with capillary zone electrophoresis at 254 nm within 14 min in the plant extract in a concentration range of 0.1-50 micrograms/ml Liebich 1998

Isoquinoline alkaloid detection by capillary electrophoresis - mass spectrometry in Hydrastis, Eschscholzia, Berberis, Jateorhiza and Chelidonium Sturm 1998

Automated TLC of alcohol extracts of Aesculus, Artemisia, Baptisia, Berberis, Carduus, Cinchona, Echinacea, Lycopus, Paulinia, Thuja Gocan 1996

Biosynthetic pathway of the bisbenzylisoquinoline alkaloid berbamunine in barberry, Berberis stolonifera starting from tyrosine Kutchan 1996

A new method for the quantitative determination of the alkaloids of Berberis aristata(author’s transl)[Article in German] Pitea 1975

Isolation of magnophlorine and columbamine from Berberis aristata [Article in Polish] Domagalina 1971

Quantitative determination of berberine in Berberis aristata, L Tses’ko 1971

Pharmacodynamics:

- The antimicrobial activity of Berberis leaves, stems and root aqueous extracts was studied in vitro on Gram-positive and Gram-negative bacteria and fungi and Candida sp. Freile 2003

- Localization of the two branch point enzymes of isoquinoline biosynthesis, berberine bridge enzyme (BBE) and (S)-tetrahydropseudoberberine oxidase (STOX), were demonstrated from different Berberis species. Bock 2002
- Synthesized 9-O-acyl-and 9-O-alkyl-substituents of berberrubine showed antimicrobial activity against Gram-positive bacteria and fungi and alkyl analogs had more antimicrobial activity than acyl analogs. Kim 2002

- Berberis crataegina DC. root exhibited potent anti-inflammatory, analgesic and febrifuge effects in mice and rats and through bioassay-guided fractionation berberine was isolated as the main active ingredient. Yesilada 2002

- Berberine individually was effective against most of the fungi except Helminthosporium spp. When Santonin was added the mixtures, wherein santonin is kept constant & berberine concentration is varied, were effective against all the fungi. Singh 2001

- The in vitro effect of berberine sulphate salt on Trichomonas vaginalis was comparable to efficacy of metronidazole as regards to potency with the advantage of being more safe. Soffar 2001

- Active inhibitors 5'-methoxyhydnocarpin-D (multi-drug resistance (MDR) pump inhibitor) from leaves of Berberis trifoliolata and pheophorbide a from Berberis fendleri could be considered as plant defense agents against natural pathogens. Stermitz 2001

- Cell membrane stabilizing property of indigenous drugs and prevention of the toxic effect of bile salts in various hepatic disorders including Berberis aristata was demonstrated by hydraulic permeability of water in presence of bile salt through a transport cell model. Upadhyay 2001

- Berberine extracts and decoctions have demonstrated significant antimicrobial activity against variety of organisms including bacteria, viruses, fungi, protozoans, helminths, and chlamydia with a long history of medicinal use in both Ayurvedic and Chinese medicine. [No authors listed] 2000

- Two compounds, flavonolignan 2 and the porphyrin 3 that are themselves devoid of multidrug resistant pump (MDR) inhibition in Staphylococcus aureus, but that form potent synergistic couples with a subinhibitory concentration of berberine were identified from Berberis species. Stermitz 2000a
• 53 bisbenzylisoquinoline alkaloids isolated from several plants including Berberis valdiviana exhibited a wide range of biological potencies in antiplasmodial assays, and the majority also exhibited some degree of cytotoxicity against human cultured mammalian cells (KB cells). Angerhofer 1999

• Berberine is shown to inhibit Activator protein-1 (AP-1) a transcription factor which plays critical role in inflammation and carcinogenesis in a dose- and time-dependent manner at concentrations higher than 0.3 microM. Fukuda 1999b

• Berberine is the component effective against Mycobacterium tuberculosis while canadine, 8-oxotetrahydrothalifendine, beta-hydrastine and two new quinic acid feruloyl esters are inactive. Gentry 1998

• SAR of berberines shows that antibacterial activity against Bacillus subtilis and Salmonella enteritidis increased as the length of the C-13 aliphatic side chain increased. Iwasa 1998

• Protoberberines are dual poisons of topoisomerases I and II. 17 analogs were cytotoxic to cancer cells. Sanders 1998

• Depression of atria by 7-O-demethylisothalicberine from Berberis chilensis is reversed by calcium. Morales 1993

• Berberis rariflora and Chenopodium multifidum are antibacterial against Gram(+) bacteria. They contain sterols. Ruggeri 1991

• 7-O-demethylisothalicberine at 10(-4)M blocked the action potential of transitional pacemaker cells. Effect is similar to verapamil. Morales 1989

• In vivo antibacterial activity of Berberis asiatica. Hashmi 1986

• Experimental research on Berberis vulgaris [Article in Bulgarian]. Manolov 1985

Berberine an antidiarrheal medication inhibited by approximately 70% the secretory responses of the heat-labile enterotoxins of Vibrio cholerae and Escherichia coli in the rabbit ligated intestinal loop model and was effective when given either before or after enterotoxin binding. Sack 1982
• A review on the chemical and pharmacological aspects of genus Berberis Ikram 1975

Study of the hypotensive action of berbamine, an alkaloid isolated from berberis lycium. Khan 1969

• On some antihelminthic properties of preparation of barberry (Berberis heterobotrys Wolf) [Article in Russian] Nuraliev 1968

• [On some antihelminthic properties of preparation of barberry (Berberis heterobotrys Wolf)].[Article in Russian]. Nuraliev 1968

• [hypotensive action of berberis spp.] [article in chinese]. Chu 1962

• [Treatment of chronic cholecystitis with a tincture of Berberis vulgaris leaves].[Article in Russian]. Lomakina 1961

**Pharmacokinetics (ADME):**

Pharmacokinetic studies of berberine in rats revealed berberine displays a linear pharmacokinetics in the dosage of 10 to 20 mg kg\(^{-1}\); processed through hepatobiliary excretion against a concentration gradient ;& berberine efflux might be affected by P-glycoprotein & organic cation transport. Tsai 2004

**Genetics & Molecular Biology:**

Two cytochrome P450 (P450) cDNAs involved in the biosynthesis of berberine isolated from cultured Coptis japonica cells and characterized. Ikezawa 2003

A spectrophotometric method was developed for the estimation of total alkaloids precipitated by Dragendorff’s reagent (DR) in plant materials and the alkaloidal percentage of some plant materials including Berberis aristata, Solanum nigrum, and Piper longum were determined. Sreevidya 2003

The N-acetyltransferase activity and 2-aminofluorene -DNA adduct formation in human leukemia cells were inhibited by berberine in a dose-dependent manner and it also decreased the apparent values of Km and Vmax from human leukemia cells in both cytosol and intact cells. Chung 2000
Berberine effectively inhibits activity of enzyme cyclooxygenase (COX-2) abundantly expressed in colon cancer cells and plays a key role in colon tumorigenesis in a dose- and time-dependent manner at concentrations higher than 0.3 microM. Fukuda 1999a

Berberis aristata, contains active principle(s) that cause(s) a selective inotropic effect, involving-in the form of the modulatory effect on actin myosin cooperativity-a novel mechanism of action. Gilani 1999

T- Berberine strongly inhibited in vitro the proliferative response of mouse spleen cells to T-dependent mitogens concanavalin A and phytochemagglutinin. Ivanovska 1999

Two alleles encoding the enzyme (S)-N-methylcoclaurine 3'-hydroxylase (CYP80B1) responsible for 3'-hydroxylation of (S)-N-methylcoclaurine penultimate step in biosynthesis of central alkaloidal intermediate (S)-reticuline were isolated. Pauli 1998

Berbamunine synthase (CYP80A1) from B. stolonifera in insect cell culture obtained different products than berbamunine synthase in the absence of plant reductase Rosco 1997

Berberine and sanguinarine intercalate DNA and inhibit DNA synthesis and reverse transcriptase Schmeller 1997

Berbamunine synthase (EC 1.1.3.34; CYP80), an oxidase that catalyzes stereoselective formation of a C—O phenol couple in bisbenzylisoquinoline alkaloid biosynthesis. The cDNA from Berberis stolonifera was expressed in baculovirus Kraus 1995

Leishmania is inhibited by berberine by interacting with DNA Ghosh 1985

**Adverse Effects & Toxicity:**

Adverse effect reports on Berberis and Mahonia at the FDA’s Center for Food Safety and Applied Nutrition FDA
**Interactions:**

Leaf extracts of Berberis aetnensis C showed the presence of pheophorbide which improved activity of ciprofloxacin and proves presence of Cytological multidrug resistant efflux pumps inhibitors. Musumeci 2003

**Clinical Trials:**

Pyrimethamine effect on chloroquine-resistant malaria was increased more by berberine (74%) than by tetracycline (67%) or cotrimoxazole (48%) in a randomized clinical trial with 215 patients Sheng 1997

Case history of 57-year-old depressive and alcohol-dependent man who became ill after ingested 40 ml of podophyllin tincture for treating warts, made from resin from the roots and rhizomes of various Berberis plants. Leitner 2002

Treatment of stomatitis with Berberis poiretii compositus [Article in Chinese] Xu 1985

**Animal Studies:**

Berberine, an alkaloid isolated from Berberis aristata, inhibits the carcinogenesis induced by 20-methylcholanthrene (200 microg/ 0.1 mL/mouse) or N-nitrosodipropylamine. Morphology of liver tissue & levels of marker enzymes indicated that berberine offered protection against chemical carcinogenesis. Anis 2001

Pretreatment of animals with berberine (4 mg/kg; orally twice daily for 2 days) prevented the acetaminophen- or CCl4-induced rise in serum levels of alkaline phosphatase (ALP) & aminotransaminases (AST & ALT), suggestive of hepatoprotection. Janbaz 2000

Berberis vulgaris is a well known medicinal plant in Iran and has also been used as food. Antihistaminic pA2 = 4.5 for fruit extract vs. 9.4 for dextchlorpheniramine. Anticholinergic pA2 = 4.4 for fruit extract vs. 9 for atropine in guinea pig ileum Shamsa 1999

Berberine, syringin, limonin and mangiferin inhibit parathyroid hormone (PTH)-stimulated bone resorption in neonatal mouse bone Li 1998
Prevention of acetaminophen-induced liver damage by Berberis aristata leaves.

Gilani 1992

E. coli in rats is inhibited by berberine from Berberis aristata at 0.1 mg Khin-Maung 1992

Palmitine hydroxide from Berberis chitria roots orally to dogs at 30 mg/kg/d for 60 days reduces primary and secondary spermatocytes and elongated spermatids by 60, 68 and 58%, respectively and decreased Leydig cells Gupta 1989
आवाप्रकाश नियंत्रणी

पर्याय

tरवी ब्रह्मचारिणी च पर्नियाद्वा पर्नवीताति च

कालकृत्यांकपीताचा च महावेन्द्र प्रचन्दाचा \.

नेव वालीकोकिर कोकतस्तत्त ब्रह्मचारीनिना

पीतानुभूति हरिभवचित्तादिवसां च पीतानु

मुणकर्त्रं — वार्षिक विशारदाना विनयानु लेखनान्तरकथान्तरतु।

वारुहिक्रम

पृष्ठीय

केवलदेश नियंत्रणी — ओषधि वर्ष

पर्याय

कटुकृत्यांकपीताचा वार्षिक विशालित्रा लिखाना

पीतानु ब्रह्मचारिणी स्वात्तु पीतानु पीतानु

... के.लि. १९१६

गुण

पर्यायचा हेमकार्तिको पीतानुक — कटुपकाठी

(नवेब कार्तीयिको गोस्वामित्वाचा कालेको।परि च)

तहत वार्षिी विशेषचरण कामिंग्जास्वयम्भतितु।

... के.लि. १९१६

श्रीदेव लिखित्यु — मुणकर्त्रं वर्ष

पर्याय — कालत्रुतु वडस्पूपार्वा सुधिक्षा पीतानु

कांवजी करस्तेकिनी तत्च कालेकर्त्ता स्वरुपादु।

... लो.लि. १३५

विशालोधि कृत्स्निभिः हेमकार्तिता कुकुसुमाला

ब्रह्मचारिणीवल्ली च पीतानु पीतानु बलावादु।

... लो.लि. १३५

शासनियुक्तु — पिन्नवाड़ि वर्ष

पर्याय

अत्यन्ति ब्रह्मचारिणी च वार्षिक पीतानु वा पीतानु

कालेकर्त्ता पीतानुस्वरूपार्वा च कामिली।

... वा.लि. २००

कटुकृत्यांकपीताचा पीताचा वारुहिक्रम स्वरुपारवा

कारीको काजलाती शक्तिविद्या प्रचन्दाचा।

स्वातः कटुकृति जेथा प्रोश्चनका साधकशाखाम।

... वा.लि. २००
गुण
तिक्ता बकङ्कित्र्वा तु कट्टरस्म व्रजोहस्तु।
करण्णकिल्लन्तिविकर्ष्यन्तिवि किस्माषिधिलोकोह। || ...शा.शि. २०२

शालिशाख निपण्डु - अष्टवर्ग
पर्याय
वार्षिककारहित्राच विलीजालोकाचीरतशस्त्र। ... शा.शि.

गुण
तितकावाकरक्षित्र्वा तु कट्टरस्म व्रजोहस्तु।
करण्णकिल्लन्तिविकर्ष्यन्तिवि किस्माषिधिलोकोह। || ...शा.शि.
वार्षिकहितोक्षघ्न्वा कालिन्दिव्यवाचिलिक। ... शासकाश

धवन्तरी विषण्डु - मुदुस्यादि वर्ग
पर्याय
अत्या बकङ्कित्र्वा च पीतारुः पीताचलवस्त्र।
विगित्वा कारकित्री सा च कारकित्री स्थुत।
कालीकर वर्णित्वा वार्षिक पीतावस्त्रीतक।
करण्णकिल्लन्तिविकर्ष्यन्तिवि किस्माषिधिलोकोह।
हेमावर्षती पीता हेमाकालसं कुलस्यतक। ... श्र.शि. ५४, ५५, ५६

गुण
तितका वार्षिककर्त्र स्यादू कषोषणा अवलोक्षितित।
करण्णकिल्लन्तिविकर्ष्यन्तिवि करण्णकिल्लन्तिविकर्ष्यन्तिवि || ... श्र.शि. ५९

जननय्याम निपण्डु - ओषधि वर्ग
पर्याय
बार्षी बकङ्कित्र्वा अव्या पीताचल स्यादू।
करण्णकिल्लन्तिविकर्ष्यन्तिवि करण्णकिल्लन्तिविकर्ष्यन्तिवि || ... ज.शि. २३०

वार्षिकर्त्रा - रसांजन
पर्याय
शाक्षातान्तरामृतीन्दारस्मानान्तराधिराज्जु। ... शा.शि.

बनस्यादि विद्या
बार्षीकार्मिकत्वनियांताविद्यारस्मानान्तराधिराज्जु। ... शा.शि.
तत्तवसाध तात्त्वज्ञातस्येव परमहितम् ॥

गुण

रसाज्ञेयं तत्त्वं नलेजविज्ञेयं तवकालं तु।
धर्मं रसावं तत्त्वं छेकं ग्रामोऽवहतु॥

रसाज्ञेयं तत्त्वं छेकं धिकारं विवेकावज्ञ।
कर्तिकप्रकाशालयोज्यितं सायु लावद्ये तु।

दस्ति दशोऽर्थं तीर्थं कद्रमर्शस्ववज्ञ।
छेकं च भले धीमं परम्पर्यं कस्माश्शलं तु॥

तुध्यं विव श्वशंस छहसीं धिका विवक्षश्लं तु।
श्वासण्डुधुखशोभनं चुवराचारं विकारितम् ॥

... आ.प्र.

शोधन विधी

तोब्जुधे कथोपपरिचितवदृढः दुःसंज्ञान।
श्वासण्डुधुकहस्मं तोब्जुधेयत॥

पथ्यों वित्तमस्ति जरोऽपरुपुष्करणेऽतु।
शिष्यावादश्चत्तथा अवरुपमिता॥

86
STHOULYA : AYURVEDIC REVIEW

REVIEW OF MEDA:

Being a dushyha dominant disorder meda, plays a major role in pathogenesis of Sthoulya Meda is an important dhatu among Sapta dhatu. Which has received equal importance as dosha. (Sha. Pu. 7/61 . Su. Su.24/9). It is so called because it smoothen (Snihyati) the body.

"Medhyati Snihayati Anen Iti Medah ",

Literaly, the word Meda is derived from root “ Jhimida Snehane ” which stand for Sneha, Fat, Oil etc. (Vachasptyam)

It means Sneha or the fatty substance of body, which is also known as fat. Adipose tissue of the body which serves as an energy reserve is known as fat.

SYNONYMS OF MEDA:

Mamsi and Mamsatej:

Meda dhatu is formed from Mamsa dhatu by Mamsagnipaka; so it is known as Mamsaj or Mamsatej.

Asthikruta:

Formation of Asthi is done from Meda so it is known as Asthikruta.

Vasa and Vapa:

The fatty substance which locates in Mamsa is called as Vasa and when its depot in abdomen, it is termed as Vapa.

Majja:

“ Asthi Madhyagata Sneha ”, is known as Majja.

Godha:

Mashtishkgata Sneha ” is known as Godha or Mastulunga.
There are two types of Medadhatu. One is poshaka and second is Poshya. Among these two, Poshaka Meda dhatu is mobile in nature (Gatiyukta); which is circulated in the whole body along with the Gatiyukta Rasa – Rakta dhatu; to give the nutrition of poshya medadhatu. Although by investigation we have seen that cholesterol and lipids are present in the circulating blood.

Second poshya meda dhatu is immobile in nature (Gativivrajita); which is stored in medodharakala. The site of Medodharakala is Abdomen (Udara) and small bones (Anu asthi), Udara, Sphik, Stana, Gala are also depots of poshya meda (Cha Su. 21/8). It is also found in Mamsa as Vasa. Practically we have also seen that main fat depots are at subcutaneous tissue and omentum.

Meda dhatu is also considered as a Sneha dominant drava dhatu, (Which Guru and snigdhagunayakta. Teja and Bhrajishnutayukta, and is dominated mainly by Jala Mahabhuta) Meda dhatu is dominated by Prithvi & Apa Mahabhutas (Cha.Chi. 15/29/-32). As a result of Mamasagnipaka it can be distinguished in the form of Sukshmabhaga (minute part) which is responsible for the further transformation of the Meda dhatu.

**PRAMANA OF MEDA DHATU:**

The total quantity of Meda is two Anjalies and the Vasa (Muscle’s fat) is three anjalies. Thus, total Meda content of the body is enumerated as 5 Anjali, and total measurable body elements are counted as 561/2 Anjali, from this equation total Meda content of body can be roughly derived, which may 11 to 12%. This equation may vary with person to person and extract measurement of body humorals is not possible due to unpredictable and ever changing nature of body (Su. Su.15/44). In sthaulya, this proportion may found raised.

**KARMA OF MEDA DHATU (FUNCTIONS):**

According to sushruta, Sneha, Sveda, Drudhatva and Asthi pushti are the functions of Meda dhatu (Sh.S.Su. 15/6). Netra and Gatra snigdhata are the additional functions of Meda mentioned by Ashtanga samgraha (A.S.Su. 19/4). Snehana is the main function of Meda dhatu and with sneha property it helps to keep luster of skin,
hair, eye etc. Snigdha gatrata symptom of sthaulya may arise through increased snehana function of Meda. It also helps in formation of cell membrane and binding of body tissue; thus modern physiology also collaborates snehana karma of it.

Another function of Meda is nourishment of further dhatu Asthi and Upadhatu snayu and Sandhi. Snayu and Sandhi both are directly related with the Asthi dhatu. In Charka Samahita snayu and Sandhi are mentioned as Updhatu of Meda (Cha.ch. 15/17).

Snayu provides support to the Asthi and Sandhi helps in joint formation. In sthoulya over nourishment of only Meda dhatu cause malnourishment of all other dhatus including asthi dhatu also.

Another function of meda is creation of Sveda and Sveda is mentioned as mala of Meda in Cha. Chi 15/18. As per sharangdhara Samahita, Sveda is upadhatu of Meda.

One more function attributed to Meda is Drdhatva, which is possible through help of snayu the Updhatu of Meda. It Drudhatva is taken as energy that other two nutrients; Carbohydrate and Protein moreover, extra energy preserves in the body in form of Meda. Meda provides supports to various organs and helps in binding of important organs.

MEDOVAHA STROTAS:

The channels which give nutrition to the medodhatu or the vessels carrying the nutritive material upto the site of Medodhatu can be considered as Medovaha Strotas.

Dr. C. Dwarakanath that the channels through which nutrition to the adipose tissue is transported are to be termed as the Medovaha Srotasas.

Dr. Ghanekar B.G. considered the medovaha srotasas as the capillaries of the perinephric tissue & omentum (Su. Su. 6)

The fat cells are held together mainly by the network of capillary blood vessels which are distributed to them. (Gray’s Anotomy)
**Moola of medovaha srotas:**

According to the three Acharyas it may be enumerated as under:

<table>
<thead>
<tr>
<th>Vrika</th>
<th>Charaka</th>
<th>Vapavahana</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vrika</td>
<td>Sushruta</td>
<td>Kati</td>
</tr>
<tr>
<td>Vrika</td>
<td>Vagbhat</td>
<td>Mamsa</td>
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</table>

The three Acharyas have considered unanimously Vrikka as one of the moola of medavaha Srotas but Vapavahana, Kati and Mamsa are mentioned as second moola separately. More Anatomically preference is given than the physiological point of view by Sushruta and Vagbhat in considering Kati and Mamsa as “Moola” of the Medovaha Srotas while Charaka’s consideration was a physiological one.

The definition given by Dr. Ghanekar for Medovaha srotas seems to be acceptable to certain extent. At the same time it represents the Boundries of the channel i.e. in his view every srotas should have its own channel and “Moola” The definition thus raises the question as to whether the prevalence of Medas and Medovaha srotas should be restricted to the perinephric tissue and the Omentum only or sometime different. The fact there are various other places than the above mentioned where the presence of fat is observed deposited similarly. The skin has some fat beneath it therefore the existence of Medo dhatu should be accepted spread up through out the body while the mentioned part or organs are only the places of conversion.

On seeing embryology, anatomy and physiology of vrikka of a glance it is difficult to name Vrikka as Kidneys. Sharangadhara says that they nourish the medodhatu in side the stomach area and by Charaka they are considered “Moola” so these structures must be directly related with fat metabolism. But in the extent of modern science as well Ayurvedic science no such function is seen being performed by them. If we take into the consideration of two structures situated above the two kidneys i.e. Supra-renal glands as Vrikka that fulfils the all aspects of fat metabolism.

**Vapavahana:**

The 2nd moola of the Medovaha srotas has been stated to be Vapavahana Chakrapani has interpreted it as Tailavartika Dr. Ghanekar has considered it as omentum.
Kati:

Shuustruta has clearly pointed out the exact site of the kati but normally the kati is the place where the fat accumulates.

Mamsa:

Vagbhat consideration of Mamsa as the moola of Medovaha strotas is not easy to explain correctly. But we might have consider the vasa (Mamsagata Sneha) below the skin and as such the entire skin may be considered as the moola of Medovaha Strotas.

UTTPATTI OF MEDODHATU:

There are different views and opinions about the origin of Medadhatu

• According to Charak the Rakta dhatu is combined with Tej, Apa and is made solid by the agni so that it gets converted into Mamsa, that again being digested by its own agni, "Medadhatvagni" and stirred up by the agni and getting combined with the quality of Apa and unctuous substances and finally gets converted into the Medadhatu. (Cha. 15/35) Meda is dominated by Prithvi and Apa Mahabhutas (Cha. Sa.7/16).

• When the Mamsagni acts upon the poshaka Mamsa Dhatu and due to this Mamsagnipaka it is divided into the following three parts.

1. The sthulabhaga (Gross part)
   Which is responsible for the formation of sthayi Mamsa Dhatu.

2. The Sukkshmabhaga (Minute part)
   Which is responsible for the further transformation of Meda Dhatu.

3. The Kattibahaga (Excreta part)
   Which is known as Khamala.

Again Meda dhatvagni acts upon the sukhmabhaga (Poshaka Medodhatu); and converts it into the sthayi or poshya Medadhatu as a sthulabhaga, Sveda as a Malabhaga and Poshaka Asthi as a Sukhsmabhaga.
In this way this process runs and Meda dhatu gets nourishment by Annarasa which has fatty substances.

The formation of dhatus (Dhatu parinamana siddhanta) is explained through three nyayas.

1) Kedar Kulya Nyaya 2) Khale Kapot Nyaya
3) Kshir dadhi Nyaya.

Out of these three maxims, commentators have favoured one or other but we have accept that at one time or another all these hypothesis work in the body. Therefore a comprehensive view is being presented here as follows:

- The Ahara Rasa which is absorbed from pakavashaya reaches in the heart by the conduction of Samana (Sa. Pu. 6/9)

The Rasa is again being thrown into circulation from the heart to first through large channels and then through smaller and smaller ones by the help of Vyanavayu. (Cha. Chi 15/36)

The Rasa ultimately reaches to the Sthayidhatus, as extracellular fluid (Kedarikulya Nyaya). This contains nutrition for all but cell of specific tissue have selective permeability for specific material only (Khalekapota Nyaya). The nutrition thus accepted is acted upon by Dhatvagni to convert into Sthayi dhatu, just like conversion of Kshira into Dadhi (Kshiradadhi Nyaya)

Thus in any of above mentioned way when Meda poshakansha is reached in Medovaha strotas, Meda dhatu is formed. Because of according to one belief each and every dhatu is produced from its own strotas. So Meda dhatu is produced in the form of Parinam Apadhyamana Dhatu from the Medovaha strotas.

According to another belief, in Medodharakala the Medodhatu is produced.

Thus, nourishment of Meda is depends upon quantity and quality of Ahara Rasa which in turn affects proportion of Meda in body (Cha. Su. 28/4)

Excessive intake of Guru, Snigdha, Madhura drvya may cause over nutrition on meda leading to Medovriddhi and its lower intake may cause under nutrition leading to Medakshaya.
**ROLE OF AGNI:**

The role of agni, in digestion and metabolism of food is very important, as all the dhatu and body components depend on this for proper formation. Sushruta has mentioned that obesity and asthma depend chiefly upon Rasa, indicating that the Rasa, which is the source of nourishment for the entire body, is also the source of Medodhatu. According to Ayurvedic classics, digestion and metabolism of food, is carried out by Agni at three levels.

1) Jataragni level.  
2) Bhutagni level  
3) Dhatwagni level

Food ingested undergoes Jataragnipaka at the outset, resulting in distintegration of the bigger molecules of the food for absorption. The functions of digestion and absorption both have been attributed to Jataragnipaka. Entire phenomenon of digestion has been described under the heading of Awasthapaka, which includes salivary (Madhurapaka) gastric (Amlapaka) and intestinal (Katupaka) digestion. Bhutagni and Dhatwagni further act upon the food thus produced by Jataragni.

Annarasa absorbed from the Mahasrota goes in circulation. The circulating ‘Rasa’ is made available to different tissues for nutrition in 3 ways known Trividha Nyayas.

1) Kedarkulya Nyaya  
2) Khale Kapota Nyaya  
3) Kshira Dadhi Nyaya

There is likely hood of the presence of two types of Agnis operating at Dhatu level by taking part in Anabolism and Catabolism. The first type is the Agni performing the functions of anabolism and regeneration have been referred by the name of Rasa Dhatwagni, Rakta Dhatwagni and so on. When there is hypo functioning of any of these Agnis, it results in increase of other Dhatus. In Medoroga, the Dhatwagni mandata is at the level of Rakta and Mamsa Dhatus. Hence, the formation of Rakta and Mamsa is reduced and the entire nutrition is channelised to nourish Meda. Medagni, being normal or hyper functioning the turn over of the nutrients (Madhura Rasa) into fat is quickly achieved. Jatharagni also governs this aspect of Dhatwagni. In Medoroga though there is relative hyper functioning of Medo Dhatwagni even then it is associated with hyper functioning of Jatharagni.
Second type of Dhathwagni i.e., Agni situated at the Dhatu level has been named as Pachaka-Ansha or Kayagne-Ansha, which is similar in function as Jathara or Pachaka Agni. Hyper functioning of this Dhathwagni will lead to the consumption (Kshaya) of Dhatus and its hypo functioning will result in growth and regeneration (Vridhi).

In this process Agni plays crucial role particularly Jatharagni, Apya–Parthiv Bhutagni and Rasa – meda dhatvagni. The general principle of Dhathvagni Dhatuvriddhi due to Dhatvagnimandhya and Dhatukshya by Dhatvagni vruddhi are described in (As. SU. 19/16, AH. SU. 11/34)

When excess supply of nutrients homologous to Dhatu, May first lead to illumination of Dhatvagni, so that the excess load metamorphosed properly. But when such a situation persists for a long time, the Dhatvagni may be diminuted leading to excessive accumulation of Dhatu. But here, the dhatu is not normal. It is rather in the form of some intermediate stage of metamorphosis i.e. Ama Dhatu. In Sthaulya due to excessive supply of Snigdha Madhura, Guru etc. types of nutrition the Ahararasa excessive nutrition homologous to medas. Due to the persistant over load, the Medogni is dimunated leading to excessive accumulation of Medas in Ama form.

**STHOULYA:**

**ETYMOLOGY**

Etymology of Word “Sthoulya”

The word Sthoulya is delivered from root “Sthu” with suffix “Ach” which stands probably for thick or solid or strong or big or bulky.


According to Vachaspatyam, the word Sthaulya means heaviness of the body.

According to Amarakosha, it stands for excessive growth of the body.
As per “Hemachandra” it indicates the state of over nutrition of body or dullness of intellect.

According to Kautilya, the world “Sthulata” means largeness or bigness or bulkiness or stoutness of body.

Anekarthavat Dhatunam:

Kuta, means highest position or degree.

Nispragna means folly or silliness.

Sthulya is used as one of the names of Bhagawan Vishnu, one type of Kanda, Priyangu, Rakta Lasuna, Ikshu etc. Shabdalkalpadrum suggest Sthula as showing fattiness.

Nirukti:

A person having and bulkiness of the body due to extensive growth especially in Udaradi region is termed as “Sthula” and the state (Bhava) of Sthulata is called “Sthoulya”

Synonyms:

Other synonyms mentioned by various Ayurvedic texts have been given below:
Table No.15: Synonyms of Sthoulya-

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</table>
DEFINATION OF STOULYA:

Atisthula has been defined as a person, “who on account of the onordinate increase of fat & flesh, is dis figured with pendulous, buttocks, belly & breasts & whose increase bulk is not mached by a corresponding increase in energy.”

VARGIKARANA (TYPES) OF STHOULYA:

Hina sthoulya: (BMI 25-30 kg/m2)

Mild degree of over weight, without any complication or secondary disease with less than four undesirable effects & with duration of less than one year can be considered as Hina Sthoulya.

Madhyam Sthoulya: (BMI 30-40 kg/m2)

Moderatedegree of over weight, with least complications without secondary disease, with less than 8 undesirable effects & with more than 5 years duration can be considered as Madhyam Sthoulya.

Adhika Sthoulya: (BMI > 40kg/m2)

Excessive state of over weight with complication and or secoandary diseases with all 8 undesirable effects & with more than 5 years duration can be considered as Adhika Sthoulya.

NIDAN OF STHOULYA:

The etiology of Medovruddhi mentioned in the Ayurvedic classics can be broadly clarified as three types:

1) Sahaja (congenital) causes
2) Viharaja (behaviour) causes
3) Aharaja (Dietic) causes

1) Sahaja Causes:

Sahaja or genetic is caused due to osha (defect) in shukra and or Arthva. According to Charaka, the excessive intake of madhura rasa by the parents, modifies
bija causing dosha and the obesity results in the next generation. Constitution of Sahaja type of person is such that even with normal or subnormal diet, there is a tendency of adiposity. Sushruta also agrees with this concept but he has explained this disease as an idopathic one.

2. Viharaja Causes:

Table No.16: Viharaja Causes of Medoroga

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<th>Sr.No</th>
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# Table No. 17: Aharaja Causes

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Hetu

The aetiological factors of medo-roga, mentioned by Ayurvedic texts are as follows (Ref. : Ch. Suo 21/4; Su.Su. 15/37, AS. Suo 24/22; A.S. Sh. 2/61).

1) Ahara

Atisampurana/Adhyasana (overeating), Guru ahara (heavy food), Shita (cold), Madhura (sweet), snigdha (fatty) ahara. Kapha vardhak ahara or shleshmala ahara.

2) Vihara

Avyayama (lack of exercise), Divasvapana (excessive sleep), Avyaya (lack of sexual intercourse).

3) Manas

Achintana (lack of mental exercise), Harshanatitaya (cheerfulness).

4) Garbhopghatkarbhava

Madhura ahara sevana by mother, during pregnancy.

5) Bijasvabhava

defect at the level of chromosome or gene.

6) Anya Karana

Santarpana, Brihana (M. Ni. 34/1).

Indulgence in above mentioned food habits, life style leads to kapha vridhi. The ahara-rasa formed will be excessively madhura and snigdha, while circulating in the body, obstructs the channals and causes medo-virdhi.

AMA

Ama is considered as undigested, unripened and unmetabolised things. It refers to undigested or incompletely digested food at the jatharagni level, at dhatvagnilevel, refers to incompletely metabolised dhatu. Ama formation is because of the mandagni (A. H. Su. 13/25).
When this ama gets mixed with the doshas, dhatu, mala etc. it is termed as ‘sama’ (A. H. Su. 13/27). The settlement of sama dosha, dhatu in the body at one place depends upon the defect or ‘khavaigunya’ in the strotas (Su. Suo 24/19).

**ROLE OF AHARA IN STHAULAYA**

Vagbhatta refers to the ‘tulyatva’ and ‘vishishtatva’ of Aharadravyas to dhatuvridhi and ksaya respectively (A. S. Su. 19/14). According to him, dravyas are either tulya (homologus, similar or identical) which increases the dhatu having similar properties and dravyas which are vishishta (heterologus, dissimilar or not identical) lead to dhatu kshaya of dissimilar properties (A. S. Suo 19/18). The substances which are meant to nourish the various structural or functional constituents of the body should be tulya or identical in following respects.

1. **Dravya Samanya or identical substances**
   
   eg: Mamsa nourishes mamsa dhatu in body.

2. **Gunasamanya or substances having similar properties to the dhatu**
   
   eg: milk, ghee increases shukradhatu.

3. **Karma samanya or identical actions**
   
   eg: Sleeping during day time increases and promote the kapha in the body.

   One or the other or all the three aspects of the ahara dravyas contribute, to rapid increase of dhatu of the similar properties.

In Medo-roga medo-dhatu is markedly increase than the other dhatus. In other words while medo-dhatu is increasing in the body in excessive amounts and all the other dhatus undergo wasting on the account of lack of nourishment for them. Charaka has considered medo-roga or Atishaulya as ‘Nanatmaja’ vikara of kapha. So’ the dravyas that are tulya or homologus to kapha, will also increase the medo-dhatu (C. Su. 20/17).

According to Sushuruta, ahara-rasa formed in atishthaulaya is madhura (sweet) and snigdha (increase fat contents) which is responsible for increase in medo dhatu (Su. Suo 15/37).
CHARAKA HAS CONSIDERED THAT WHEN MOTHER TAKES EXCESSIVE MADHURA AHARA DURING PREGNANCY, THE OFFSPRING WILL BE ‘STHULA’ (C. SH., 8/21).

VAGBHATTA HAS NAMED THIS TYPE OF DISEASE AS ‘GARBHAJA VYADHI’ AND SUBDIVIDED IT AGAIN INTO ANARASAJA TYPE (A.S. SU. 22/3).

BIJASVABHAVA

The role of bijasvabhava or genetic abnormality in pathogenesis of Medoroga is stated by Charaka (C. SU. 21/4).

It can be explained that when parents or grand parents are indulged in madhura, snigdha ahara they may develop bijadosha, which give rise to a constitution that there is tendency for the formation of meda even with, normal or subnormal diet.

ANYA KARANA

The other reason for atisthaulya are excessive santarpana ie. overeating of madhura, guru, picchila, nava ahara, meat of animals living near water, mansa, dahi ghee etc. (C. SU. 23/6).

By brihana or roborant therapy in excess, also is responsible for atisthaulya (C. SU. 22/38).

DHATU KSAYA-VRIDHI

The ksaya (decrease) or vridhi (increase) of the sthula dhatu depends on the following factors.

1. AHARA-VIHARA

The food and the activities, samana or homologus or with similar properties that of body elements will tend to increase the particular element (A.S. SU. 19/14).

For eg. Madhura snigdha guru ahara which is having the properties homologus to the kapha, increases the kapha in the body similarly divasvapna, avyayama will also increase the kapha.
In case of medo-roga the ahara-vihara homologus to the properties of the medo-dhatu increases the meda in the body.

2. AHARA-RASA

The fire in the wood gets more aggravated, when a big tree burns and the fire seems to be extinguished, when a small bush burns. Similarly when the ahara-rasa of specific quality, in good amount and constantly reaches the dhatu with similar properties, will increase the dhatu formation (dhatu vridhi) and when the ahara-rasa fails to reach the dhatu, there will be dhatu-ksaya or wasting of the dhatu (A. S.Su. 19/17).

In medo-roga the ahara-rasa formed by the madhura, snigdha ahara sevna is not only madhur but madhuratatsya and atisnigdha which circulates allover the body. This ahara-rasa is having the properties similar to medo-dhatu and ahara-rasa is in excess quantity also, so this ahara-rasa is capable for the medo-dhatu vridhi and is not compatible to the other dhatu and the other dhatus remain undernourished.

3. DHATVAGNI AND PACHAKANSHA

When the dhatvagni or the agni acting on the asthayi dhatu increases or decreases, the dhatu formation also increases or decreases.

Pachakansha act on the sthula dhatu or sthayi dhatu, when the action of this agni increases there is dhatu kshaya and when its action decreases, there is dhatu vridhi (AS. Suo 19/16).

In medo-roga the medodhatvagni is normal or hyperfunctioning leading to the medodhatu vridhi. Also the action of pachakansha on the sthula medo-dhatu is decrease, further leading to medo dhatu vridhi.

4. BIJASVABHAVA

The dhatu vridhi-ksaya also depends upon bijasvabhava i.e. when the mother or father or both are sthula, the offspring will also be 'sthula’ as there is inheritance of atisthaulaya by the bijasvabhIwa. There is tendency of the children to grow fat even with normal or subnormal diet.
SAMPRAPTI

By the nidana sevana i.e. excessive intake of madhura snigdha, guru ahara and activities like divasvapna, avyayama etc, kapha gets aggrevated. The jatharagni status is hyperfunctioning which digests all the ingested food and because of the aggrevation of kapha and the madhura-snigdha ahara, the ahara-rasa formed after the digestion of this food is maduratarasya and atisnigdha i.e. excessive sweet and increased fat contents. This ahara-rasa is homologus to the properties of medo-dhatu so it goes constantly towards it increasing the medo-dhatu formation.

Also the previous dhatvagnis i.e. rasagni, raktagni may be normal or subnormal or hypofunctioning as compared to the medodhatvagni. The hyperfunctioning medo dhatvagni and there is decreased action of the pachakansha on the sthula medodhatu which causes the medo dhatu vridhi.

Bijasvabhava also plays an important role in medovridhi, causing medodhatu vridhi even with normal or subnormal diet.

In this way one or the other or all the factors are responsible for the medodhatu vridhi. The meda gets accumulated in sphika, udara, stana leading to the enlargement of these organs to the extent that they cause pendulous movements, when the person moves, also there is ayatha upacaya utasaha. This stage is termed as “Atisthaulaya”.

Further increase in meda causes the obstruction of the channals, manifesting the ill effects of atisthaulaya, Kshudrashvasa, kshudha, trishna etc. (Su. Suo 15/37).

By the obstruction of the channals of the meda, vayu circulates mainly in kostha, further stimulating the appetite, again resulting in overeating. If the appetite is not satisfied, the vayu will cause drastic complications due to lack of proper amount of food, it will burn the body just like the fire burns the forest (M.Ni. 34/5, 6, 7).
SAMPRAPTI OF MEDOROGA

Madhura, Snigdha ahara + Bija swabhava + Divasvapna Avayam

\[ \downarrow \]

↑ Kapha

\[ \downarrow \]

Atimadhura - Atisnigdha ahara rasa

\[ \downarrow \]

Circulated all over the body

\[ \downarrow \]

↑ Medodhatvagni and ↓ Vpacakansa action

\[ \downarrow \]

↑ Medo dhatu

Accumulation in sphika, udara, stana

\[ \downarrow \]

Atisthaulaya

\[ \downarrow \]

Overeating ← ↑ jatharagni ← ↑ vayu ← obstruction of channals by meda

\[ \downarrow \]

Upadrava of Atisthaulaya

The samprapti of atisthaulya can also be explained on the basis of 'shatkriyakala'

Sanchaya

Because of excessive madhura, snigdha, guru ahara intake and divasvapna avyayam etc. and also due to bijasvabhava there is sanchaya of kapha.
**Prakopa**

The kapha increases in quantity and quality and is responsible for the formation of the atimadhura and atisnigdha ahara rasa.

**Prasara**

Atimadhura and atisnigdha ahara rasa formed circulates all over the body through the channals.

**Sthanasanshrya**

The circulating ahara rasa gets collected in the medo dhatu because of defect or khavaigunya in medo vahasrotas and the medodhatu increases and gets accumulated in sphika, udara, stana.

**Vyakti**

The increased quantity of medodhatu accumulated in body organs make them increase in size and pendulous movements when the person moves. The channels get blocked by the meda causing the ill effects of the sthaulaya.

**Bheda**

The manifestation of the upadrava or the complications of obesity such as prameha, prameha pidika, bhagandara etc. can be considered as the bheda stage of atisthaulaya.

**PURVARUPA OF STHOULYA:**

Knowledge of Purvarupa is important for differential Diagnosis and to treat the disease in its beginning (Cha. Ni. 1/36) So that disease can be controlled easily and can not develop further.

Purvarupa of sthaulya has not been described by any Ayurvedic text. As per directions given in Vata Vyadhi (Cha. Chi. 28/19), Urahkshata (Cha. Chi.11/12) and Trishna (Cha. Chi.22/8) chapters the initial manifestation of Sthaulya related symptoms can be considered as premonitory symptoms or purvrupa of Sthaulya.
There is presence of symptoms itself in mild form mean marginal over weight since childhood can also be taken as Purvarupa of Sthaulya.

More over, Medovaha Strotodushti Lakshana which are also described as Purvarupa of Prameha can be considered as Purvarupa of Sthaulya (Cha. Su.28/15). Bahudrava Shlesma and Abaddha Meda are the two morbid components involved in pathogenesis of Prameha (Cha. Ni. 4/8), which are found vitiated in Sthaulya. So Shlesma Sanchaya and Medodusti lakshana related purvarupa of prameha and Medovaha srotodusti Lakshna described by Acharyas can be considered as Purvarupa of Sthaulya. The symptoms related with Meda Dushti Like Atinidra, Tandra, Alasya, Visra Sharira Gandha, Anga Gaurava, Shaithilya etc. can be considered as Purvarupa of Sthaulya.

**CLINICAL FEATURES**

Charaka has mentioned the features of sthaulaya which are as follows (C. su. 21/9).

1. Meda, mansa ativridhi (increase fat and muscular tissue).
2. Chala sphika, udara, stana (enlargement of buttocks, belly and breasts to the extent that they become pendlllous when the person moves).
3. Ayatha upacaya-utsaha (improper metabolism and lack of vigour and vitality).

**DISABILITIES WITH STHAULAYA**

Caraka has stated eight fold disabilities or ill effects due to sthaulya (C. Suo 21/4).

1) Ayushorhasa (shortening of lifespan)
2) Javoparadha (swiftness in movement)
3) Kricchavyavaya (difficulty in sexual intercourse)
4) Daurbalyam (weakness)
5) Swedabadha (excessive sweating and its unpleasant effects)
6) Daurgandhya (bad odour).

7) Kshudhatimatrama (increased appetite)

8) Pipasatiyoga (excessive thirst)

**AYUSHORHASA**

Because of the conversion of complete food taken into meda and also due to obstruction of the channels by meda, the rest of the dhatus are not properly nourished and the person is weak. His body immunity will also decrease and will get diseases easy, decreasing the life span.

**JAVOPARADHA**

As the person does not have much strength and also the fat is heavy, there is swiftness in movements or hampered movements in atisthaulaya.

**KRICCHAVYAVAYA**

Difficulty in performing sexual intercourse is mainly due to paucity of semen and obstruction of the genital passage by meda and kapha.

**DAURBALYAMA**

Only medodhatu is in excess quantity and the rest of the dhatus are imbalanced or state causing generalised debility. Due to the margavrodha by meda other dhatus remain undernourished.

**SWEDABADHA AND DAURGANDHYA**

There is excessive production of sweat, due to admixture of meda with kapha and because of excessive sweating and inability of the person to bear strain of exercise, there is distress or unpleasant effects.

Daurgandhya is because of excessive sweating and also due to the over production of other malas of the body.
**KSHUDHATIMATRAM**

Due to obstruction of the channels by the meda, vayu circulates mostly in the abdomen causing ‘tikshnagni’. Due to which food taken is very quickly digested and again there is feeling of hunger. If food intake is reduced, Vayu burns the body just, like the fire in the forest destroys the forest.

**RUPA:**

Disease is known by its interrogation; observation and inference. Rupa serves as an instrument in all these three. On the completion of Dosha – Dushyasammurchhana the feature of the manifested disease is Rupa.

Different Acharyas have presented the symptoms of Sthaulya in their texts. Charaka has enlisted the following symptoms as cordinal pr Pratyatma Lakshana of Sthaulya. Medomamsa Ativriddhi Chala Spik, Chala Udara Stana, Ayatha Upachya, Anutshaha ( Cha. Su. 21/9 ) Besides these cardinal symptoms, eight disabilities of Sthoulya i.e. Ayushohrasa ( diminution of life spana ), Javoporodha (lack of agility), Kriccha Vyavaya ( difficulty in sexual act), Daurbalya (deability), Daurgandhya ( Foul smelling of body ), Svedbadha ( distressful sweating ), Kshudha Atimatrata (excessive hunger ), and pipasa Atiyoga (excessive thurst ) are the most prominent clinical features of Sthaulya narrated by Charak (Cha. Su. 21/4) Susrut ( Su. Su. 15/32) and Vagbhat ( AS 24/23-26 ).

All the symptoms of Sthaulya described in various Ayurvedic texts have been summarized in the following table.
Table No18: Rupa of Sthoulya-

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<tr>
<th>Sr No</th>
<th>RUPA</th>
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<th>SH.</th>
<th>AS</th>
<th>AH</th>
<th>MN</th>
<th>BP</th>
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<td>Chala Sfick (Pendulous Buttock)</td>
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<td>Anusaha (Lack of Enthusiasm)</td>
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<td>Alpa Vyavaya (Lack of sexual life)</td>
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<td>Swedabadha (Excessive sweating)</td>
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</table>

Elaborated pathogenesis of occurrence of Ashta Dosha of Sthoulya has been mentioned in (Chi. Su.21/4), which are as follows:

- Due to excessive formation of Medodhatu formation of other dhatus is inhibited so due to lack of nourishment it causes Ayushohrasa.
- Due to Shaithilya, Saukumarya and Guru properties of Medodhatu it causes Javoparodha.
- Due to obstruction in genital passage by medodhatu and paucity of semen it results in Kriccha Vyavaya.
- Owing to imbalance of Sapta Dhjatu produced by Sanga type of obstruction of Medodhatu it results in general debility (Daurbalya).
- Due to excessive sweating, innate quality of Medodhatu and morbid nature of vitiated Meda it causes Daurgandhya.
- By the admixture of Kapha with Meda, Vishyandi, Bahutva and Guru properties of Meda and its inability to bear the strain of exercise it results in Svedabaddha.
- Due to increased Agni Koshta and initiation of Vata by obstruction of Meda it results in Kshuhatimatrata and Pipasatiyoga.

**DIAGNOSIS (NIDAN)**

Early diagnosis of a disease is very important by treatment viewpoint. Sthaulya is a slow growing disorder. If it can be diagnosed in initial stage and preventable measures can be taken in early stage it can be prevented. The methods described in Ayurvedic texts for diagnosis of a disease, mostly are subjective. But, diagnosis of Sthaulya is easy and very apparent. It has been mentioned as chakshu Indriya vijlIeya bhava in Shusruta Samhita (Su, Su 10/5) by following words:

चबुतित्व सिक्कलोः क्रमीत्तत्त्वावलोः

According to this overnutrition condition Sthaulya or undernutrition condition karsya can be diagnosed by inspection only, Apart from this Pratyaksha pariksha, Anuman pariksha and Aiptopdesha are another useful diagnostic methods, which can be applied to diagnose Sthaulya and its related symptoms, (Cha. Vi. 4/3). According to Ashtavidha Pariksha, Sthaulya can be determined by Akriti Pariksha (Rogapariksha Cha. 1)

Ayurvedic pramana pariksha and samhanana pariksha can be correlated with objective criteria of diagnosis like measurements of body weight, height, various girth measurements and skinfold thickness measurements.

Charaka samhita is pioneer to describe anthropometry. Pramana paridlsa is described elaborately, under the caption of dashavidha pariksha (Cha. Vi. 8/117), Pramna pariksha has been also narrated in detail in Shusruta Samhita (Su, Su, 35/12
Here, an attempt has been made to correlate Ayurvedic Praman Pariksha with modern anthropometry. It may provide a relative measurement and objective criteria for patients of sthaulya,

The measurements described in Charaka Samhita and Shusruta Samhita for different anatomical parts and sites (Anga-pratyanga) are summarized and illustrated in table.

### Pramana pariksa described in ayurvedic texts

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Praman in Angula</th>
<th>Inch</th>
<th>Cm</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cha.</td>
<td>Su.</td>
<td>63-90</td>
</tr>
<tr>
<td>Ayam</td>
<td>84</td>
<td>120</td>
<td></td>
</tr>
<tr>
<td>Vistar</td>
<td>84</td>
<td>----</td>
<td>63</td>
</tr>
<tr>
<td>Shir parinah</td>
<td>32</td>
<td>----</td>
<td>24</td>
</tr>
<tr>
<td>Shir ayam</td>
<td>16</td>
<td>----</td>
<td>12</td>
</tr>
<tr>
<td>Griva parinah</td>
<td>22</td>
<td>20</td>
<td>15-16.5</td>
</tr>
<tr>
<td>Griva ayam</td>
<td>4</td>
<td>----</td>
<td>3</td>
</tr>
<tr>
<td>Vaksha parinah</td>
<td>48</td>
<td>----</td>
<td>36</td>
</tr>
<tr>
<td>Bhuja parinah</td>
<td>16</td>
<td>16</td>
<td>12</td>
</tr>
<tr>
<td>Bahu parinah</td>
<td>12</td>
<td>----</td>
<td>9</td>
</tr>
<tr>
<td>Jangha parinah</td>
<td>52</td>
<td>----</td>
<td>39</td>
</tr>
<tr>
<td>Uru parinah</td>
<td>30</td>
<td>32</td>
<td>22.5-24</td>
</tr>
<tr>
<td>Jangha madhya parinah</td>
<td>16</td>
<td>18</td>
<td>12-23.5</td>
</tr>
</tbody>
</table>

### Skinfold thickness measurement

According to ayurvedic texts skin consists six (Cha. Sa. 7/4) to seven (Su. Sa. 4/4) layers. Specific measurement of each skin layer is not given by Charak Samhita. Bur Shusruta Samhita has given particular measurement of each layers in following fashion.
Further, while describing operative treatment of jalodara, Shusruta has mentioned pramana (Su. Chi. 14/18), which can be taken for fleshy regions and measurements of six yava correlates with 1.0404 cm.

<table>
<thead>
<tr>
<th>Skin layer</th>
<th>Praman</th>
<th>Measurement (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st layer</td>
<td>1/8 yava</td>
<td>0.013</td>
</tr>
<tr>
<td>2nd layer</td>
<td>1/16 yava</td>
<td>0.014</td>
</tr>
<tr>
<td>3rd layer</td>
<td>1/12 yava</td>
<td>0.019</td>
</tr>
<tr>
<td>4th layer</td>
<td>1/8 yava</td>
<td>0.029</td>
</tr>
<tr>
<td>5th layer</td>
<td>1/5 yava</td>
<td>0.046</td>
</tr>
<tr>
<td>6th layer</td>
<td>1 yava</td>
<td>0.234</td>
</tr>
<tr>
<td>7th layer</td>
<td>2 yava</td>
<td>0.468</td>
</tr>
</tbody>
</table>

**UPADRAVA:**

Due to chronic consistence of Sthaulya some bizzare complications ensures as the result of deformity of several systems, organs and thus ultimately leading to Ojo dusti and death. High intensity and severity of Sthaulya, due to Ati Kshudha and Ati Pipasa and manifestation of severe complication and even death due to its ignorance have been mentioned with example of Davanala in (Cha. Su. 21/7-10). Due to ignorance, if kala Vyatikrama occurs then various type of complications manifests with dominance of Vata and Pitta dosha. Here, Kala Vyatikrama has been explained as later stage of life by Gangadhara.

Whereas, it has been interpreted as lapse of mealtime by Chakrapani. Both these explanations seem to be true as elder obese are noted with more complications and even death with VLCD (Very Low Calory Diet) and therapeutic starvation another no specific Upadravas have been mentioned by Acharya Charak, but other treatises have listed a few. These Upadravas have been presented in the table.
Table No. 19: Upadrava of Sthoulya

<table>
<thead>
<tr>
<th>Sr. No.</th>
<th>UPADRAVA</th>
<th>SH</th>
<th>AS</th>
<th>AH</th>
<th>MN</th>
<th>BP</th>
<th>YR</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>Ama Roga</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>Apachi</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>3</td>
<td>Arsha</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>4</td>
<td>Atisara</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>5</td>
<td>Bhagandara</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>6</td>
<td>Jwara</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>7</td>
<td>Jantavaha</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
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<td>8</td>
<td>Kamala</td>
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<td>9</td>
<td>Kasa</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>10</td>
<td>Kustha</td>
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<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>11</td>
<td>Mutra Kricchra</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>12</td>
<td>Prameha</td>
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<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>13</td>
<td>Pramehapidika</td>
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<td>+</td>
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<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>14</td>
<td>Shleepada</td>
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<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>15</td>
<td>Sanyas</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>16</td>
<td>Udararoga</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>17</td>
<td>Urusthambha</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>18</td>
<td>Vatavikara</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>19</td>
<td>Visarpa</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>20</td>
<td>Vriddhi</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

According to Su. Su. 15/32 complications occur due to grave obstruction of various channels, particularly Medovaha strotas. Here Dalhana has specified only obstruction of Vata by Meda. Further more, disorders of Sleshma, Rakta and Mamsa along with symptoms, complications and secondary diseases. Sthoulya are stated as the result of morbid Meda (AS. Su.19/7) and inability to bear the strain of disease as well as therapy is mention as ill effect of the disease ( AS. Su. 23/7). Madhava Nidana has described complication of Sthoulya in brief by* “Vikaran darunan kritwa nasrayati asshu jivitam” Srikanthadutta has explained above complication under this heading. According to Bhavprakash excessive perspiration and foetid odour caused by Meda is the main pathology in genesis of Krimi.
After thorough and conventional analysis these Updravas can be subdivided into following pattern.

**Updravas due to Agni Vikriti:**

Updrava like Ajirna, Atisara, Arsa, Udararoga etc. can emerge due to malfunctioning of Agni and formation of Ama.

**Updravas due to Meda Vikriti:**

Apachi, Vrdradhi, Slipada, Pramehapidika, Visarpa etc. Upadrava may result due to Meda particularly Baddha Meda. Besides these Granthi, Arbuda, Galganda, Vriddhi, etc. disorders resulting due to vitiation of Meda can be taken as Upadrava of Sthaulya, Granthi, Apachi, Galganda are the diseases of vitiated Medodhuta results due to Sthansamshraya of three dosha & Rakta in Medo dhatu (Su. Su.21/33) & Granthi & Vrana are narrated as symptoms of Medogata Vata. (Su. Ni.1/26).

**SADHYASADHYATA ( Prognosis )**

Kriccha sadhya nature of Sthaulya has been described by most of the Ayurvedic classics Bad prognosis of Sthaulya has been described by Charak, because if they are not duly managed. They are prone to death due to excessive hunger, thirst and complications. Moreover, lack of immune power ( Vyadhi Asahatva ) is mentioned as a common feature as well as serious draw back of Sthaulya ( Cha. Su. 28/6 ) so they are frequently prone to secondary disease.

Bad prognosis for sahaja ( hereditary ) diseases is described in Cha. Chi . 6/57. Hence sahaja sthaulya can be considered as Asadhya.

It has been mentioned that Medogata disease are curable only in uncompleted patients with more Bala and less choricity ( AH. Chi. 22/51-52 ).So Vagbhat has describe Sthaulya as Asadhya disorder due to its relasping and challenging nature. He has quoted that there is no remedy for patients of Sthaulya ( AH.Su. 14/27 ).

The treatment of Sthaulya is tedious work in comparison with Krusha person. The aim of treating “Sthaulya” is at reducing Vata, Agni and Meda. Neither santaparna nor apaterpana mode of treatment is efficacious for correcting Sthaulya; because
tarpana Chikista pacifies Vyaya & Agni; but at the same time raises Meda dhatu. On the other side Apatarpan Chikitsa reduces Meda on one hand but elevates the status of Angi and Vayu in the body.

From above, it can be concluded that Apatarpa Dravyas Gurugunas can produce the desired results. Also there is a very limited choice of drugs and diets for Sthula person and there is a greater probability of getting affected by complications so Vagbhat has considered Sthaulya as Dushchikista Vyadhi. It has been mentioned as incurable due to innate tendency (Svabhava) (BP. Ma 39/11).

Sudden drastic weight reduction orgain is described as Arista (Bad Prognosis) (AH.Sa. 5/12). Yogaratnakar that sudden weight loss has further elaborated it or gain could be fatal within six months. Therefore Sadhyasaddhyata despicted in Ayurvedic classics (Cha. Ni. 8/33-35) which are as below.

**Sukha Sadhya :**

Jatotara Hina Sthaulya having duration of 1 to 5 years, without any complications or secondary disease, can be considered as Sukha Sadhya.

**Kruccha Sadhya :**

Jatotara Madhya Sthaulya having duration of 5-10 years, with least complication but without secondary disease can be considered as Kriccha Sadhya.

**Asadhya :**

Sahaja Sthaulya is Asadhya. Jatotara Adhika Sthaulya having duration of more than 10 years, in the presence of complication and secondary disease can be considered as Asadhya.

**CHIKITSA:**

In Ayurveda, general principal of management of any disorder is

चेन्नाम् कंसेजन्त विकासन्त्र व दव्यल्लेख क ... च. वि. 9/30

Thus in any disorder management is divided into 3 parts.

1) Nidan Parivarjan

2) Samshodhana

3) Samsamana
1) **Nidan Parivarjan**

The root of Samprapti process, Nidana must be avoided for best management of the disease. In Sthaulya the factors i.e. Aharatmaka Viharatmak, manas and others which are mentioned in Nidan’s chapter and which are responsible for the disease should be avoided.

2) **Samshodhan Therapy**

Meaning of Shodhana is apakarshana of dosha. Therapies in which the aggravated doshas or the excretory product of digestion are eliminated after mobilising them from their respective sites, by Urdhva or Adhah marga from the body is known as Shodhana Therapy. It is also termed as Aparkarshan. There are two main parts shodhana therapy.

**Shodhana Therapy**

```
<table>
<thead>
<tr>
<th>Bahya Samshodhana</th>
<th>Abhyantar Samshodhana</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Udavartana</td>
<td>1. Varmana</td>
</tr>
<tr>
<td>2. Avagah</td>
<td>2. Virechana</td>
</tr>
<tr>
<td>3. Parisheka</td>
<td>3. Nasya</td>
</tr>
<tr>
<td>4. Lepana</td>
<td>4. Niruha</td>
</tr>
</tbody>
</table>
```

Many Acharyas have mentioned external purification therapy for management of Sthaulya. In all classical texts, Udavartan was mentioned as part of Dincharya and for Sthaulya Raksha Udacartan was narrated by Acharyas. The benefits of Udavartan was also mentioned i.e. Kaphahara, Medasah Pravilyanm, Sthirikaranam Anganam, Tvaka Prashadakar (AH. U. 2/15). In Stjaulya Udavartan posses removes the foetid odour, restricts the process of excessive sweating, alleviates the aggravated doshas by function.
Abhyantar Samshodhana

Being a syndromic entity (बह्यत्कृष्णन्तः) Samshodhan therapy is highly recommended for Sthaulya management by Charak. According to Vagbhat, patients with Ati Sthaulya, Adhika Dosha and Adhik Bala should be treated with Samshodhana therapy including Varmana, Virechana, Ruksha Niruha, Raktamokshana and Shirovirechana Charak has recommended Varmana and Virechana Karmas for all Santarapanajanit Vyadhies. As Sthaulya also comes under the same category, Vamana Virechana therapies can be tried Sthaulya.

Though higher exclamation of Samshodhana therapy, Purvakarma like Snehana, Svedana and Pradhana Karma including Vamana, Virechana Anuvasan. Basti has been contraindicated to Atishula Patient by most of the Ayurvedic texts. Snehana Karma is always restricted for the patients of Sthula (Cha. Cu. 13/53). However on exigency usage of Taila is recommended (Cha. Su. 13/44-46) Lekhan and Medahara properties (Su. Su. 45/112) and Sthulatyahara action (Su. Chi. 31/16, AS. Su. 6/100) of Taila are described in Ayurveda and advised to use particularly Tail of Sharsapa (Su.Su. 45/11-7) Tuvarak and Bhallataka (Su.Su. 45/112) and Yavatitka (Su.Su. 45/125). Among them Sharsapa Taila can be used for internal as well as external snehana.

Another important Purva Karma Svedana is also restricted for the patient of Sthaulya by most of the Ayurvedic texts except Sushruta Samhita (Chi. 32/15), Kashyapa Samhita (Su. 23/19-20) and Sharangdhar (U. 2/11-12). Although on exigency Mridu Sveda is advised for thaulya patients (BP. Pu. 34/11-12). Especially Nirangni Sveda Viz. Guru pravarana, Bahupana, Kushdha Nigraha. Atap sevan, Vyayam, Ahava, Krodha, Bhaya and Upanaha (Cha. Su. 14/64, Su. Chi. 32/15) are advocated Vagbhat has indicated the use of pinds sveda on extreme requirement (AS. Su. 26/8).

Most of the texts prohibit the use of Varmana Virechana Karma due to inability to bear the potency of medicine and therapy causing Pranaparodha (Life threatening Condition (Cha. I. 2/8-9, 11-12). Moreover, Ati Bruhmana and Meda Mamsa Ativruddhi are mentioned as a predisposing factors for Vamana and Virechana Vyapada (Ka. Shi. 3) However Vamana is indicated for treatment of Medo Roga (Su. Chi. 33/18). Further more, similar line treatment is advised for disorders of Shleshma and Meda (AS. Su. 9/13, YR- Dosha Samana-3) and Vamana is considered as the
best for alleviation of Kapha dosha (Cha. Su. 25/40). Hence, Vamana can be used for the treatment of Sthaulya, but only in uncomplicated patients.

Though Virechana has been forbidden for patients of sthaulya (Cha. Si. 2/11-12, Sh.S Chi. 33/29-3/5 Ka. Si. 7), mild Verechana is indicated for Pittadhikya condition. In Sushruta Samhita, while description of Virechana Karma, Staula Pramehi is considered as Durvirechya due to aggravation and dominance of Meda Tikshna Sodhana (Virechana) is insisted (Su. Chi. 12/6). Line of treatment for Atinidra has been described by Charak has been included for the management of sthaulya by Bhava Mishra (BP. Ma. 39/11-26) and Virechana karma is indicated for treatment of Atinidra and thereby for Atisthaulya. Among Virechaka dravyas mentioned in Ayurvedic Materia medica, Haritaki, Katuki, Aragvadha, Trivruta, Danti – Drayanti, Snubji etc. are attributed to have additional medanasaka property. Hence Virechana Karma could be applied to the patients to the patients of Sthaulya with suitable drugs among these. Practically, also Vamana and Virechana karma seems to be fruitful for the management of Sthaulya.

Bruhumana Karma of Anuvasan is well established and so application of it is Unanimously contraindicated for patients of Sthaulya (Cha. Si. 1/36, Su. Su.Chi. 35/21). It is considered as one of the etiological factor of Sthaulya and always restricted for the patient of Sthaulya. But Asthapan Basti particularly Lekhan Basti is highly recommended for management of Sthaulya by ancient Aurvedic physicians (Cha. Su. 21/17-18, Su. Su. 15/38, Chi. 35/22). Tikshna Basti is considered as most suitable and complicated disorder like Sthaulya (Cha. 10/17). Apart from that instant nourishing and non nourishing property of Basti is also well known (Cha. Si. 10/5). The role of ruksha, Ushna and Tikshna Basti in management of Sthaulya is very well explained by Gangadhara (Cha. Su. 21) (Jalpakapataru comme). As per his explanation Basti itself is the complete treatment for derangement of vata and further admixture of Ruksha, Ushna, Tikshna dravyas with Basti contributes to alleviate Kapha and Meda. Thus this type of Basti provides complete elimination of Samprati and considered as the treatment of Sthaulya Sushruta also recommended Basti therapy and emphasizes to consider patients Agni, Bala etc. while administering Lekhan dravya is combination of Vata and Teja Mahabhuta dominance (Sh. Su. 41/10). Further characteristics of Lekhan dravyas and Lekhan Basti are given by Sharangdhara (S.S.Pu. 4/10 & U 6/23). According to him, the substance which can cause absorption
or extirpation of Dosha, Dhatu, Mala and emiciation of body is called Lekhan and the Basti prepared with Triphala Kwath is termed as Lekhan Basti. Lekhan or Karshana Basti helps to remove abstraction of Meda, Kapha and Kleda from Srotas by its veerya and helps to alleviate ata and normalize the function of Agni and Vayu.

Basti prepared with Taila, Gomutra, Kanji and Saindhava (Cha. Si. 10/13/14), Erandmuladi Nirula (Cha. Si. 3/41), kaphanasaka Basti (Cha. Si. 10/23-24), Lekhana Basti (A.H. Ka. 4/7-10, Sha.U. 6/23), Madhutailik Basti (S.S. U. 6/3/-33, BP/29-30) etc. can be practiced for management of Sthaulya.

Raktamokshna along with Urdhva and Adhah Samshodhana is mentioned as one of the best therapy for Sthalulya patients especially for Medasvi Dhatri (KuDhatri Chikitsa Adhikar). Bhavaprakasha has also indicated Raktamokshana as oline of treatment for Sthaulya (Bh.P.Ma. 39/12). Rakta Mokshana may be applied for patients of Sthaulya, more specifically in the patient with Rakta Gat Medo Vruddhi.

Avapida Shirovirechana is mentioned as line of treatment for Abhisyanna Meda Vyapta Shrira especially Sirah i.e. excessive accumulation of Meda in body especially in upper body (S.S. Chi. 40/44). Moreover Karshana Nasya is indicated in the disorders of Kapha Dosha (Ka. Si. 2) and Sthaluya is enumerated as one of the twenty kapha nanatmaja disorder. So Shirovirechana Nasya can be applied to patients of Sthaulya. Besides that, Triphaladya tail has been indicated for Nysya Karma in patients of sthaulya (BP. Ma. 39/55-56, YR – Medoroga Chi.) Shirovirechana can be used in patients of Sthaulya to alleviate of kapha and its symptoms like Maha Glani, Jadya, Alasya, Krathana, Gadgadtva, Atinidra etc.

3. Samshamana Therapy:

Among Shad Upakarma (Six type of therapies), Langhana and Rukshana can be administered for Samshaman purpose in Sthaulya (Cha. Su. 22/4) Saman Chikista can be implemented through seven different ways.

Deepan, Pachana, Kshudha Nigroha, Trisha Nigroha, Vyayama, Atapaseven, Marutseven.

Above mentioned all seven procedure can be counted under single title i.e. “Samana Langhana Chikista”. Langhana is administrated in Santarpanajanya Vyadhi, in Amashyothavikara, in Shleshmika Vikara, in Rasaja Vikara and it is best remedy advised in Sama Roga.
According to general principle of management (ध्युयम हङ्गम, ज्रुम्दयग्नि हङ्गम) for the management of Sthaulya of Vata, Pitta and Kapha especially Samana Vayu, Pachaka Pitta and Kledaka Kapha along with depletion of Mdhatu increasing Medo dhatvagni is main aim of treatment in Sthaulya.

- Deepan and Panchana – The Deepan dravyas are dominated by Agni and Vayu Mahabhutas which are anti to the constitution of Meda and Kapha i.e.Jala and Prithvi Mahabhuta, Deepan therapy is efficacious in Sthaulya because apart from digesting Ama and Apakva Meda, it has the additional property to potentiate Jatharagni, Bhutagni and Dhatavagni.

- Kshudha Nigraha - Exercising control over one’s appetite is one of the age old therapies practiced among the Indian masses. Fasting is one type of “Nidana Parivarjan Chikista”, because the food is the main source of Nutrition of Md dhatu fasting controls the over production of Meda dhatu. Fasting first of all halts the nutrition of Ama and thus activate the Agni and Digests the Ama. As a result, Agni functions are restored the Dhatavagni gets stimulated which resists further nourishment of Medodhatu.

The digestive power absorption of the food, both are stimulated in Shool Purusha. So, the person digest food quickly, but if he is deprived of the food, at the time of his need, the Margavarodhajanya aggravated Vayu and Agni may lead to serious complications. Looking to this possibility it is advisable to give small quantity of Laghu and Ruksha Aahara in place of complete or severe starvation, so that the Tikshagni gets fuel in the form of food and ultimately checks the over production of Meda.

- Trisha Nigraha – Excessive thirst is a symptom and also a cause of Sthaulya. Drinking of cold water in large quantities depresses the Jatharagni by adding to the liquidity of Pitta. It also increases the metabolic by product, Kleda. O a Sthula Purusha should take luke warm water in small quantities. Thus he should control his thirst, which aids in the phenomenon of deepan and panchana. From the word control of thirst we can arrive the conclusion that obese person should avoid sweet and soft drinks, nutritious fruit juice and cold water.
• Vyayam – Importance of Vyayam or physical exercise has been described since centuries in Sthaulya. Most of the Acharyas described Avyayam is cause of obesity and in the management of Sthaulya all have given more importance to Vyayam. So, Vyayam works as Nidana Parivarjan as well as it melts the excessive fat deposited in the fat depots.

In all classical texts, Vyayam was also mentioned as a Routine (Dincharya) the detail description of Vyayam was mentioned by various Achryas. “Sharir Ayasajanka Karma” is Vyayam. It is the definition of Vyayam benefits of Vyayam was also mentioned i.e. Laghava, Karmasamarthya, Agnidipti, Medkshya, Vibhaka Ghangatratva. Indication and contraindication and quality and quality of Vyayam was also mentioned. “Sharir Artha Sakya” Vyayam in routine is beneficial for good health. They also mentioned vyayam is the best remedy for obesity.

Atapa Sevan: Atapa Sevan enhances Ushma in the body. This raised body heat increase heat reduces Meda by Vibhajana and Vilayan. It potentiates Kleda Vilayana.

Marut Sevan: The word Maruta is the synonyms of Vata. By the Vata sevana, then Ruksha Guna of Vata reduces Kleda and Kapha by Shoshana and Stimulation Jatharagni.

Treatment According to Charak Samhita:

Charak Samhita has been given treatment in following words कृषि वायुवियन्ति वेश्यः स्थूलात्मा कर्षण प्रवति। वातात्वि वृत्तात्झ श्वेताश्चैरहस्ति च। \( ... \ C. २४/३०-२५. \)
Administration of articles which possess additional Vata, Slesma and Mad NaShaka properties is considered as an ideal for Samasaman therapy. Chakrapani has explained that Guru property is sufficient to alleviate vitiated agni and thereby Atikhudha and Apatarpana property provides non nourishment and thus leads to depletion of Meda. For example Madhu possess Guru and Ruksh properties, hence it is ideal for management for Sthaulya. Gangadhar has interpreted it that Guru property is suitable to alleviate Tikshnagni and vitiated vata especially Koshthagata vata and thereby Ati Kshudha, and apatarpan property is that which can not provide Tarpana (Trupti) and cause reduction of Meda due to under nourishment (Aposhakatvat).
Some Samsamana yoga like Guduchi – Bhadra Musta, Triphala, Takrarista, Makshika, Vidangadi Lauha, Bilvadi panchmula and Silajatu with Agnimanth svarasa are advised to practise for prolonged period as prayoga due to chronic consistence of the disease (Cha. Su. 21/21-24). Guduchi and Triphala are advocated due to its Tridoshahara properties, which indicates that all the tridosha hara Dravya with additional Medanasaka properties can be tried for Sthaulya management. Vidangadi Lauha acts through its specific action and lack of Guru property (Gaurava Nirapeksh). Ruksh Udarvartana is advised to use for the external treatment.

As earlier mentioned Samtarpana and Apatarpana are two opposite actions and its resultant disorder Sthaulya and Krisata are two opposite conditions. So, etiological factors of Karsya can be taken as line of treatment of Sthaulya Ruksh Annapana Prayoga, Langhana, Pramitasana, Kriya Atiyoga, Shoka, Nidra Vega Vinigraha, Ruksh Snana, Ruksh Udvartan, Abhyas, Krodha etc. are the causative factors of Karsya which can be practiced as line of treatment for Sthaulya (Cha. Su. 21/10-12). Here, Gangadhara and chakrapani have explained Pramitasana as stokasya Asana. i.e. Matra Alpasya Asana which can be taken as intake of low calory and low quantity diet. (LCD)

Apart from that varied treatment of Sthaulya is depicted as different places in Charaka. Drugs and preparation like Karsana Yavagu of Gavedhuka (Su.2/25), Lekhaniya Mahakashay (su. 4/3(3)), Venuyava (Su. 27/20), Bibhitaka (su.27/148) Dhana and Virudha Dhana (Su. 27/266) and Madhudaka (Su. 27/323) are advocated as Medanasaka and Lekhan. Akasa and Vyavya Mahabhuta dominant Dravya are attributed to have laghavakara action (Su.26/11), So Akasa and Vayavya Mahabhuta dominant articles can be useful for management of Sthaulya. Katu and Kashay Rasa are attributed to have Karsana and Upachyahara action and Tikta Rasa is attributed to have Lekhan and Meda Upashoshan Karma (Su.26/44); hence, Katu, Tikta and Kashay Rasa can be used for treatment of sthaulya and Dasavidha Langhna therapy is mentioned for the same. (Su.23/8, 25). Administration of Viruksana and Chhedaniya Dravya especially Silajatu, guggulu, Gomutra, Triphala, Loha Raja, Rasanjana and Madhu in proper dose and duration is advised by Sushruta Samhita (Su. 15/38). Here Dalhana has explained that Viruksaha property helps to reduce Meda and Chhedaniya property helps to remove obstruction from body channel. Particularly from Medovaha srotas by its Sroto Vishodhana property.

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2) Treatment according to Sushruta

Besides that, Sushruta has included regional treatment and narrated paschima maruta i.e. wind from western direction as Meda Vishosana (Su.20/28). In 38th chapter of Sutrasthana various groups of drugs like Varunadi Gana, Salasaradi Gana, Rodhradi Gana, Arkadi Gana, Muskadi Gana, Trayusnadi Gana, etc. are described as Medanasaka (Su. 44/70). So, these drugs can be used for the treatment of Sthaulya. Haritaki is advised for the treatment of Santarpanajannya Roga (su.44/69) and Amalaki is mentioned as Medapaham (Su. 44/70). So Haritaki and Amalki can be used for treatment of Sthaulya. Karpura (su.46/203), Kansya (Su.46/330), Trapusisa (su.46/331), different Mani (Su.46/332) are described as Lekhan and Medahara, which can be used for treatment of Sthaulya.

Further more, line of treatment of Maha Kustha is advised to practice as line of treatment of Sthaulya (Su.Chi. 10/3) and in same chapter different type of ayakriti are mentioned as Medanasaka. Besides that different methods of Aptarpana mentioned in (Su.chi. 11/4) can be used for management of Sthaulya.

3) Treatment according to Ashtang Sangrah:

Madanphaladi Churna, Kutajadi, Hingvadi Churna and Vidangadi Mantha are added in line of treatment in line of treatment (Su.24/37-46) and Krisna Kauha (Su.12/1748), Sankha and Samudraphena (Su. 12/22), Tuttha (Su.12/23), Manahsila (Su.12/24), Anjan (Su. 12/25) and Silajatu (Su.16/17) are additional Dhatu described as Lekhan and Medanasaka in Astanga Samgraha, which can be included for treatment of Sthaulya. Rasanjana is mentioned as the best for the treatment of Sthaulya and Guggulu is mentioned as the best for the disorders of Meda and Vata (AS. Su. 13/3), so Guggula can be used for the treatment of Meda Avrutavata condition. Further in the 16th chapter of Sutraasthana, Asanadi, Sursadi, Vatsakadi and Vachadi, Haridrani Gana are mentioned as Medanasaka and lekhana, which can be used for Sthaulya management.

4) Treatment according to Ashtang Hridaya

Gumutrapaki Haritaki (Chi. 8/55-56), Rodhrasava (Chi.12/28), Navaka Guggulu (Chi.21/50) Amruta Guggulu (U.28/38), Vardhamana Bhallataka Rasayana (U.39/66-71) etc. are the remedies added by Vagbhat in Sthaulya management is Ashtang Hridaya.
5) Treatment according to Sharangdhara

Vacha, Yava, Ushnodaka and Kshaudra are mentioned as Lekhana Dravya by Shaangdhara (Pu.4/10). Sakhotaka Valkala Kwatha (Ma. 2/126), Brihat Manjisthadi Kwatha (Ma. 2/139-144). And Yogaraj Guggulu (Ma. 7/69) are additional remedies indicated by Shrangadhara for Sthaulya management.

6) Bhavprakasha – Dhumapana and Upasana added in line of treatment of Bhavprakasha (Ma. 39/13). In modern medicine, thermogenic activity of smoking is well established, hence Dhumpana with Meda Kapha Nasaka drugs may be useful for treatment of Sthaulya.

Chavyadi Saktu (39/15), Triphaladya Churna (39/16), Muli Churna (39/18), Erandapatra Kshara (39/23), Badaripatra Peya (39/25), Amritadi Guggula (39/27), Dasanga Guggula (39/28), Trasusnadi Guggulu (39/29), Lauha Rasayana (39/30-40), Lauharist (39/41-48) etc. are the remedies mentioned by Bhava Prakasha for Savanga Medaharana. Besides that Sinhanda Guggulu (Ma.29/227-230), Pancha nimba Avaleha (Ma.54/56-66) Piplyadi Gana Kwath (Ma. 71/143-146) etc are mentioned and Meda Nasaka.

Patol Patradi Kwath (39/19), Ati Mukta Churna (39/23), Chitraka Mula Churna (39/23) Erandamula Churna (39/23) are the drugs used for Udaragata Medovriddhi (Android obesity). Silajitvadi Udavartana is advocated for the external treatment in (Ma. 39/26).

Triphaladya Tail earlier used by Vagbat reapplied by Bhavaprakash for internal use with Sursadi Gana Kwath and for external use in Gandusa, Nasya. Abhyanga and Basti (Ma. 39/55-56)

Bhavaprakash as elaborated symptomatic treatment of Sweda dhikya and Gatra Daurgandhya symptoms by giving preparation like Mahasugandhi Tail (Ma. 39/58-65) Vasa patra and Bilva Patra lepa (39/66), Alambusha churna (39/67), Bilvadi and Putikaranja Lepa (39/63), Chinchapatra lepa and Dugdha Haridra Lepa (39/69), Sirisadi Lepa and Tejapatradi Lepa (39/70), Samudraphena Churna Lepa (39/74), Haritaki Udavartan (39/72), Haritakyadi Pralepa Jambukwata snana etc. Many other Udarvartana and lepas and also mentioned oral Medicines like Panchatikta Grita Guggulu in (39/81).
7) Treatment according to Yogratnakar

In Yogratnakar, many new combinations are described i.e. Navaka Guggulu, Trayusnadi Lauha, Talpatra Kshara, Rasa Bhasma, Trimurti Rasa, Vadvangi Rasa for treatment of Sthaulya, and also described Udarvartana and lepa of different drugs for symptomatic treatment for Svedabhadha and Gatra Daurandhya.

DOSE DURATION AND METHODS OF TREATMENT:

Prag Bhakta i.e. intake of Medicine before meal is instead for Krishkaran purpose (AS. Su., 23/14) It has been further elaborated by Sharandhara and advised to take Lekhan drug on empty stomach in early morning and before a meal (Pu. 4/10) So for treatment of Sthaulya medicine should be administered before meal and ideally in morning and on empty stomach. The use of Avisadakara, Mridu and Sukhakara Aushada in gradual increasing dose with caution is advised for Sthaulya management (AS. Su. 23/7). Further it has been emphasized to consider Agni Bala, Deha Bala, Dosha Bala, and Vyadhi Bala Prior to fixation of dose and duration of treatment for Sthaulya. It has been advised in Charak Samhiita to follow constant and prolonged therapeutic intervention for management of Sthaulya. (Gangadhara on Cha. Su. 21/16).

PATHYA - APATHYA

Practicing appropriate Pathya, Apathya along with treatment of disease is one of the unique characteristics. Ayurvedic science, classical texts emphasize that success or failure of the treatment depends to a large extent on the practice of Pathya and Apathya. The food articles, drugs regiment which do not affect the body and mind adversely are regarded as Pathya and in the same way which adversely affect the body are considered to be Apathya.

Acharya Lolimb Raj has highlighted the importance of Pathya apathya by registering that “What is the need of the medicine, if the person is following the Pathya – Apathya rules and there is no effect of medicine for the one who is not following the Pathya Apathya rules. Keeping view the pathological factors, the ancient Acharyas have listed numerous Pathya, Apathya for Sthaulya.

For better understanding, the Pathya- Apathya described by various Acharyas is given below in table “A” and it is being further summarized in table “B”
Table No.20: Pathya Pathya for Sthoulya- A

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<td>Godhum Naveen Dhanya (Shali)</td>
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<td>Kanda Shaka, Madhura, Rasatmak</td>
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<tr>
<td>4. Phala Varga (Fruits)</td>
<td>Kapittha, Jambu, Amalki, Ela, Bibhitaki, Haritaki, Maricha, Pippali, Erand Karkati, Ankola, Narang, Bilvaphala,</td>
<td>Madura Phala</td>
</tr>
<tr>
<td>5. Drava Varga</td>
<td>Honey, Takra, Ushnajala, Tila &amp; Sarshapa Tail, Ashava Arista, Jeema Madhya</td>
<td>Milk preparations, (Dugdha, Dhadhi, Sarpi) Ikshuvikara</td>
</tr>
<tr>
<td>6. Mamsa Varga</td>
<td>Rohita Mastya</td>
<td>Aanupa, Audaka, Gramy Mamsa Sevena</td>
</tr>
</tbody>
</table>

### Table ‘C’ Pathya – Apathya Vihar (Physical Regimen)

<table>
<thead>
<tr>
<th>Pathya</th>
<th>Apathya</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chinta</td>
<td>Nitya Harsha</td>
</tr>
<tr>
<td>Shoka</td>
<td>Achintana</td>
</tr>
<tr>
<td>Krodha</td>
<td>Manso Nivrutti</td>
</tr>
</tbody>
</table>

### Table ‘D’ Pathya – Apathya Vihar (Mental Regimen)

<table>
<thead>
<tr>
<th>Pathya</th>
<th>Apathya</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shrama</td>
<td>Shhetal Jala Sevan</td>
</tr>
<tr>
<td>Jagarana</td>
<td>Diwaswapa</td>
</tr>
<tr>
<td>Nitya Bhramana</td>
<td>Aavyayam</td>
</tr>
<tr>
<td>Ashwarohana</td>
<td>Aavyayam</td>
</tr>
<tr>
<td>Hastavyarohana</td>
<td>Ati Ashana</td>
</tr>
<tr>
<td>Vyavaya</td>
<td>Sukha Shaiya</td>
</tr>
</tbody>
</table>
Acharyas have widely enumerated the Laghu Anna and excessive physical activity patients of Sthaulya. It is clear from the discussion on pathophysiology that the less calorie requirement is needed to these patients. Patients require to take more and more physical activity so that accumulated fat can be burnt. However while planning the treatment always it should be kept in mind that less calorie consumption (Langhana Apatarpan) and more calorie expenditure (Langhana – Apatarpan) are Vata provocative factors hence whenever Ahara Kalpa is to be derived it should be anti vata along with anti kapha for the patients of Sthaulya.

The average daily requirement of calorie is being presented here in tabular form recommended by ICMR.

In previous chapters calorie value and protein contents various diet articles have already been presented. This all should be considered while planning the diet and physical regime for the patients of Sthaulya.

Reference Shlokas

- दह खृत्रु नाशनस्थियिवयो अही पुकार विनविनिता अक्षिति, तयया अतिदीर्घत, अतिकृत्य, अतिशुल्कन, अतिशोभान, अतिशम, अतिकृत्य, अतिशोभान, अतिशुल्कन, अतिकृत्य अक्षिति।

- प्रजातिस्वतानुक्रमेष्यां एकाग्रे विनविनितिविशेष अक्षिति।...

- जनतं व्याहितेतावहितस्वादुकुशी बनी।

- जनतं वास्तवात्तर्रत्नं वर्षाणीही विद्धि।

- व्यायामाय नियंत्रित स्थुलतातिपोषितेन।...

- अतचतर्निधित्वाती नवो भूल।

- वेदिको मत्तपरिन्मनुपुस्त स्थुलतातिपोषितेन।...

- कार्यमुद्र वर्षीय स्थूलवानं हि स्थूलवत्वं श्रेष्ठजन।

- वृद्धायं संविधे जातासारिजेस्यविवाहतित्र।...

- अन्तुमय।...

- जेलक्ष्यान्त संतुष्टे वाहनवातिषुवातः।

- विकालवासं वाक्षपालं वृद्ध्य नामदत्तस्याभासं जीवंतिताय।...

- जेलोरहणं खोरालं बृहीं नृत्त ब्यानांत।...

- जेलोरहणं बृहीं बृहीं कडात।

- जेलोरहणं हे तयया अक्षिति, कटी बृहीं।...

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• वया उद्वयस्था स्तिरयहस्तिकायायूणुवर्जिताः: तैरयहस्तिकायाः दृश्यत ।
  ... च.वि. ५/१२
• जेहें,दिति वस्येवायां वस्यषुद्वन्तं तद्विव आमायकेदैव देघे पुष्प।
  ... च.वि. ६/२२
• जेहें जसपुष्टिमयटमनृत्व। ... च.शा. १५/८
• ब्री जेहें। ... च.शा. १/१५
• जेहें पलिब्जाः न विचारते। ... च.शा. १५/३७
• वास्तवं स्वल्पिति वालसं तद्वितीय इति कथयते।
  तद्वीरम बुद्ध स्लिगमपपलकाविनिति बुझय वाजर! ... शा.शा. पूर्वब्यं ५/२२
• जेहार्जस्ययहस्तिकायारोषेन जात्यस्तिरयहस्तिकायाः। ... आ.शा. च.शा. १/२४
• वर्णयस्तिर्यहस्तिकायारोषेन ज्ञात्यस्तिर्यहस्तिकायाः। जेहें जेहें जेहें मात्र।
  ता भाषवता वितेष्युपुष्पं जा यम न्यायाया ते वर्णस्तिर्यहस्तिकायाः।
  ... च.वि. ८/१०६
• ... जेहें स्त्रायुःश्रवः। (स्त्रायुःश्रवः: इति या)। ... च.वि. १५/१७
• वस्येव धर्मान्य नारीवां कार्य: भवति नवग्रहत।
  बुद्ध मांश्य अव: स्लेहं ता वस्यार्चिकारितता।
  स्लेहं वस्ये वस्यलक्ष्यम केशार्जस्वामित्वज्ञ लाभम।
  इति वायुमं जेहें द्वयेस्मेस्य नाम ग्रहतः। ... शा.शा. पूर्वब्यं ५/२८
• जेहार्जस्य स्लेहर्जस्य विस्ताराज्ञायुयस्तिर्यहस्तिकायाः।
  स्लायाः तु मुहः पाक: स्त्रायुःस्त्र व तत: वायः। ... च.शा. ४/२९
• स्त्रायुःस्त्रस्य सूब्जधं प्रोक्ता वेदे मानसिनिमस्यस्याः। ... शा.शा. पूर्व १/३४
• ज्ञात्यमधस्यकृतिः: वन्द्यावलेष्व आश्चयता तता। ... च.शा. ५/३४
• महाविकोशस्त्रो तं विश्व इति वेशा। ... च.शा. ५/२९ उपाधिः
• ग्राम: स्लेहर्जस्त्र जेहें। ... च.वि. १५/१८
• विव्ह (उद्वक) उज्जायानुर्जन स्लेहर्जस्त्र: विक्रमात्त्र: लेवब्याख्यास्त्र: अवग्रहायते। (तद्वः वर्णस्तिर्यहस्तिकायाः)
  ... च.शा. ३/१७
• स्लेहं: केश (क्षेत्र) ज्ञात्याश्रुमधस्यकृतु:। ... च.शा. १५/५
• स्लेहस्त्र केशस्य कृतिः: (क्षेत्र विक्रियाप्रथमत)। ... आ.शा. १५/१५
• मधवात्त्र ज्ञात्यायां व्यक्तिवंस्त्रो वेशायायाः: प्रभायाः।
  लाभस स्त्रेष्य जीवायां ते तन्तु चायस्त्रण च। ... च.शा. १७/६६
• जेहें स्लेहं प्रभायात्त्र: ज्ञात्यायां ते तन्तुतेव जेहें प्रभायाः।
  ... च.शा. १५/१५
• जेहें स्लेहस्त्र मण्डलः: ज्ञात्यायाः: कृपायाः। ... आ.शा. १५/१६
• विशेषाधिकृत विषय क्रम: प्रथम ।

विष्णुप्रदेशीय विषय क्रम: वर्तमान ॥... जून. ५१/५२

• जेद्विक्षित विषय क्रम: प्रथम ।

ईक्ष्वाकुवर्ष साधक ॥... जून. ३४/३५

• जेद्विक्षित विषय क्रम: प्रथम ।

चंद्र जंगल विषय क्रम: प्रथम ॥... जून. २२/२२

• विद्वत्तिकृत विषय क्रम: प्रथम ।

ताप... वायु प्रमाण गाढ़ातिष्ठ। वायु गाढ़ातिष्ठ।... जून. १५/३१

• जोलियो विद्वत्तिकृत विषय क्रम: प्रथम ।

चंद्र विद्वत्तिकृत विषय क्रम: प्रथम ॥... जून. २४/२२

• विद्वत्तिकृत विषय क्रम: प्रथम ।

चंद्र विद्वत्तिकृत विषय क्रम: प्रथम ॥... जून. २२/२२

• विद्वत्तिकृत विषय क्रम: प्रथम ।

चंद्र विद्वत्तिकृत विषय क्रम: प्रथम ॥... जून. २४/२२

• विद्वत्तिकृत विषय क्रम: प्रथम ।

चंद्र विद्वत्तिकृत विषय क्रम: प्रथम ॥... जून. २२/२२

• विद्वत्तिकृत विषय क्रम: प्रथम ।

चंद्र विद्वत्तिकृत विषय क्रम: प्रथम ॥... जून. २२/२२
OBESITY : MODERN REVIEW

DEFINITION

Obesity is an increase in total body fat, a weight of 120% or above the ideal body weight.

It can also be defined as a disorder of energy balance. When food-derived energy chronically exceeds energy expenditure, the excess calories are stored at triglycerides in adipose tissue.

- Excess deposition of adipose tissue is obesity.
- A Body weight 20% or more above desirable weight for age, sex and height is regarded as obese.
- A recent National Institute of Health consensus conference defined obesity as Body Mass Index greater than 27 kg/m². Now a days obesity is defined at or greater than 25 kg/m² BMI.
- Parks has given obesity may be defined as an abnormal growth of the adipose tissue due to an enlargement of fat cell size (hypertrophic obesity) or an increase in fat cell number (hyper plastic obesity) or a combination of both.
- It can also be define as a disorder of energy balance. When food derived energy chronically exceeds energy expenditure, the excess calories are stored as triglycerides in adipose tissue.

Hence, the modern terminology obesity can be used satisfactorily for the disease sthoulya.

ETYMOLOGY OF WORD OBESITY:

Literary, the word obesity is derived from Latin word “Obesus” from “ob” by reason of and “Edo”. I eat means having eaten. The word obesity is referred to excessive states of over weight and is the clinical term used to describe this condition.
SYNONYMS OF OBESITY:

Adiposity, Over weight, Fatness, Turgidity, Hypertrophy, Stoutness, Enormity, Corpulence, Polysarca, Oily dropsy, Plumpness, Embonpoint etc. are the synonyms of Obesity mentioned by various medical texts and literature.

DIAGNOSIS

Specific Criteria for Diagnosis of Obesity:

It is very difficult to label a person as an obese; because numbers of factors are needed to be considered to arrive at a conclusion. In most cases it can be detected by visual inspection and this usually suffices for diagnosis but it also important assess the serving of obesity. Obesity is defined in a number of way; Obesity can be assessed in several ways. The direct methods of measuring body fat include under water weighing (Densitometry) estimation of total body water, estimation of fat cell mass by isotope dilution method, estimation of total body potassium, Tobec (conductivity), Bioelectric impedance. Both computed tamography and nuclear magnetic resonance imaging can be used to distinguish between the fat and lean tissue of the body. These are several laboratory methods for measuring body fat but none of them is widely available for clinical use. These methods generally divine the body into fatty compartments. In addition to the laboratory methods, there are several techniques that can be used in the field studies or the clinical setting. These generally based on anthropometric measurements of body dimensions and are less direct that the laboratory methods.

The techniques are summarized in this table:
<table>
<thead>
<tr>
<th>Method</th>
<th>Cost</th>
<th>Ease</th>
<th>Accuracy</th>
<th>Measures Regional fat</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Height and weight</td>
<td>Low</td>
<td>Easy</td>
<td>High</td>
<td>No</td>
</tr>
<tr>
<td>2. Skin folds</td>
<td>Low</td>
<td>Easy</td>
<td>Low</td>
<td>Yes</td>
</tr>
<tr>
<td>3. Circumstances</td>
<td>Low</td>
<td>Easy</td>
<td>Moderate</td>
<td>Yes</td>
</tr>
<tr>
<td>4. Ultrasound</td>
<td>Moderate</td>
<td>Moderate</td>
<td>Moderate</td>
<td>No</td>
</tr>
<tr>
<td>5. Density</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Immersion</td>
<td>Low</td>
<td>Moderate</td>
<td>High</td>
<td>No</td>
</tr>
<tr>
<td>Plethysmograph</td>
<td>High</td>
<td>Difficult</td>
<td>High</td>
<td>No</td>
</tr>
<tr>
<td>6. Heavy water</td>
<td>Moderate</td>
<td>Moderate</td>
<td>High</td>
<td>No</td>
</tr>
<tr>
<td>Triturated</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deuterium Oxide</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Or heavy O₂</td>
<td>High</td>
<td>Moderate</td>
<td>High</td>
<td>No</td>
</tr>
<tr>
<td>7. Potassium isotope (¹⁰⁰K)</td>
<td>V.high</td>
<td>Difficult</td>
<td>High</td>
<td>No</td>
</tr>
<tr>
<td>8. Total body electrical Conductivity</td>
<td>High</td>
<td>Moderate</td>
<td>High</td>
<td>No</td>
</tr>
<tr>
<td>9. Bioelectric impedance</td>
<td>Moderate</td>
<td>Easy</td>
<td>High</td>
<td>No</td>
</tr>
<tr>
<td>10. Fat Soluble gas</td>
<td>Moderate</td>
<td>Difficult</td>
<td>High</td>
<td>No</td>
</tr>
<tr>
<td>11. Absorptionmetry</td>
<td>High</td>
<td>Easy</td>
<td>High</td>
<td>No</td>
</tr>
<tr>
<td>( dual energy X-ray Abdual photon Ab )</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12. Computed tomography</td>
<td>V. High</td>
<td>Difficult</td>
<td>High</td>
<td>No</td>
</tr>
<tr>
<td>13. Magnetic resonance</td>
<td>V. High</td>
<td>Difficult</td>
<td>High</td>
<td>No</td>
</tr>
<tr>
<td>imaging</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14. Neutron activation</td>
<td>V. High</td>
<td>Difficult</td>
<td>High</td>
<td>No</td>
</tr>
</tbody>
</table>

Table No.21: Methods of Estimating Body fat and its Distribution
The indirect methods more commonly utilized in clinical and field practice and have the advantage of the use of less sophisticated equipments are as follows.

**WEIGHT AND HEIGHT MEASUREMENT**

The most common method of recording relative weight is from the standard height and weight tables which refer to body weight in relation to a reference population of desirable standard.

**WHO Concept of human physique According to BMI Rang**

Body Mass Index (BMI) = \[
\frac{\text{Weight (kg)}}{\text{Height (m)}^2}
\]

**Table No.22: B.M.I. & Risk Grades**

<table>
<thead>
<tr>
<th>BMI Range</th>
<th>Indication</th>
<th>Mortality</th>
<th>Morbidity</th>
<th>Metabolic effects of injuries</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI &lt; 16.00</td>
<td>Indicates grade 3 thinness</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI 16.0 – 16.99</td>
<td>Indicates grade 2 thinness</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI 17.0 – 18.49</td>
<td>Indicates grade 1 thinness</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI 18.5 – 24.99</td>
<td>is the normal range for An individual</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI 25 – 29.99</td>
<td>Indicates grade 1 overweight</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI 30.0 – 39.99</td>
<td>Indicates grade 2 overweight</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI ≥ 40.00</td>
<td>Indicates grade 3 overweight</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Mortality

Morbidity

Metabolic effects of injuries

Coronary Heart

NIDDM

Hypertension

Gall Bladder

Diseases

Osteoarthritis
The standard weight and height table for Indian Female and male is adapted from the Life Insurance Corporation of India Standard. These tables for ideal and desirable weight are based on acturial estimation of what consistent with normal life expectancy.

Body mass index (BMI) is a measure of body fat relative to height. It is calculated by dividing the body weight (Kg) by the square of height in meters.

**SKIN FOLD MEASUREMENT**

The width of subcutaneous skin fold in biceps, triceps, sub scapular and suprailliac is measured with calibrated calipers. Nomograms are available to convert skin fold measurements into percentage body fat.

Skin folds have also been used to measure fat distribution between the trunk and extremities. This is termed fat patterning index.

**MEASUREMENT OF WAIST AND HIP CIRCUMFERENCE**

Measurement of waist and hip circumference with a tape measure provides the same information as skinfold thickness and is also an estimation of body fat distribution. Both in men and women, high waist / hip ratio is considered a risk factor for ischaemic heart disease stroke etc. In males, the risk for the disease increases when the waist / hip ratio rises above 1.0 and in females when it rises above 0.8. Subjects with abdominal obesity (android obesity) are at greater risk for cardiovascular complications than those with gluteal obesity (gynoid obesity)

**CLASSIFICATION OF OBESITY:**

**Hina Sthaulya:** (BMI 25-30 Kg/m²) Over weight

Mild degree of over weight, without any complication or 5 secondary disease with less than four 5 undesirable effects and with duration of less than 1 year can be considered as Hina Sthaulya.

**Madhyam Sthaulya:** (BMI 30-40 Kg/m²) Obese

Moderate degree of overweight, with least complications without secondary disease, with less than 8 undesirable effects and within duration of 1 to 5 years can be considered as Madhyam Sthaulya.
Adhika Sthaulya: (BMI > 40 Kg/m²) Very Obese

Excessive state of complication and or secondary disease, with all eight undesirable effects and with more than 5 years duration can be considered as Adhika Sthaulya.

In modern texts, obesity is classified as follows:

According to Onset:
(a) Insidious    (b) Gradual    (c) Rapid

According to Clinical Condition:
(a) Enviable    (b) Regal      (c) Pitiable

According to Severity:
(a) Mild        (b) Moderate    (c) Severe

According to distribution of fat:

(a) Generalized: Generalized obesity is usual in alimentary or exogenous obesity.

(b) Central or Trunk type: Involving only the trunk and neck, it is common in Cushing syndrome or hypothyroidism.

(c) Superior or Buffalo type: Involving the face, neck, arm and upper part of trunk and is common in Cushing’s syndrome or hypothyroidism.

(d) Inferior Lypodystrophy: Involving the lower part of the trunk and legs accompanied by wasting of upper of the body.

(e) Girdle type or fatty apron: Involving the hips, buttocks and abdomen found in pituitary or hypothalamic lesions.

(f) Breeches or trochanteric type: Involving only the buttocks, found in hypogonadal syndrome.

(g) Lipomatous or multiple lipomatous: With localized deposits of fat over the body called Dercum’s disease or adiposis dolorosa when associated with tenderness and pain over the fatty lumps.
**According to Histopathology:**

(a) Hyperplastic obesity: Accompanied by increase in adipocyte's number, life long history and bad prognosis.

(b) Hypertrophic obesity: Accompanied by increase in adipocyte's size, adult onset history and good prognosis. This is more often associated with metabolic disorders such as Diabetes Mellitus, Hypertension, Coronary artery disease and Hyperlipidaemia.

**According to Etiological Factors:**

(a) Physiological: Observed temporarily during Puberty, Pregnancy and Lactation.

(b) Pathological: It can be further divided into three Viz.

(i) Exogenous

(ii) Endogenous

(iii) Idiopathic

(i) Exogenous: Called as simple obesity and accrues to sedentary habits and over eating.

(ii) Endogenous: Glandular entity can be subdivided as follows:

- Cushing's Syndrome - Hypothalamic
- Frohlick's Syndrome - Dercum's disease
- Adrenogenital syndrome - Water - salt retentional
- Hypothyroidism - Hand Schuller Christain Syndrome
- Achord their syndrome - Localized obesity
- Hypogonadal Syndrome - Hyperinsulinism
- Mesopausal - Lawrencemoon-biedle Syndrome

**ETIOLOGY OF OBESITY:**

The causes of obesity are distributed in main three groups according to modern medicine.
1. Exogenous: Where the chief cause is excessive appetite or over feeding

2. Endogenous: Where the endocrine factors are important.

3. Miscellaneous: A number of factors are known to be associated with its development, which are taken under this heading.

**CAUSES OF OBESITY**

<table>
<thead>
<tr>
<th>Exogenous</th>
<th>Endogenous</th>
<th>Miscellaneous</th>
</tr>
</thead>
<tbody>
<tr>
<td>Over eating</td>
<td>Endocrine</td>
<td>Age</td>
</tr>
<tr>
<td>Dietary habits</td>
<td>factors</td>
<td>Sex</td>
</tr>
<tr>
<td>Drinking habits</td>
<td></td>
<td>Occupation</td>
</tr>
<tr>
<td>Smoking</td>
<td></td>
<td>Socioeconomic factors</td>
</tr>
</tbody>
</table>

The most common cause of obesity is excess calorie intake couples with physical inactivity. Liray (1951) categorises obesity in four types: (a) Antroid (b) Gynoid type (c) Sponge like obesity (d) Endocrine obesity

I. **Anatomical Characteristic of Adipose tissue**

Hypertrophic obesity (enlargement of fat cells) correlates with android or truncal fat distribution and is more often associated with metabolic disorders.
II. Age at onset of obesity

Obesity which starts in early childhood tends to be more severe and generalised and obese children have a higher chance of being obese in adulthood.

Obesity which starts later in life is hypertrophic and central in type. In women pregnancy contributes to weight gain.

III. Aetiological Factors

1. Neuroendocrine disorders
   a. Hypothalamic disorder: Injury to ventromedial region of the hypothalamus results in obesity.
   b. Hypothyroidism: Thyroid deficiency in the adult presents with clinical features of myxoedema.
   c. Cushings Syndrome: The pattern of weight gain in Cushings syndrome is characteristic, with accumulation of fat in the trunk, supraclavicular fossa and dorsal cervical region, while the arms and legs are spared.
   d. Polycystic ovary syndrome (Stein- Leventhal Syndrome) consists of irregular or absent menses, hirsutism, obesity and infertility.
   e. Hyperinsulinism: Hypersecretion of insulin occurs in insulinoma, this can increase body weight.

2. Genetic Syndromes

   These rare syndromes – Lawrence – Moon – Biedl Syndrome, Praker – Will Syndrome, Asstrom Syndrome, Cohen syndrome are associated with dysmorphic features. In addition to obesity, hypogonadism is common, other features are short stature, mental retardation and polydactyly.

3. Drug-induced obesity

   Drugs such as corticosteroids, tricyclic antidepressant cyproheptadine phenothiazines and lithium can lead to weight gain.
4. Excess calorie intake and physical inactivity

Overnutrition is more responsible for the continuity of overweight by increasing the total number of fat cells in the body.

Sedentary habits are responsible for weight gain during adulthood.

Psychological factors in the development of obesity are recognised. Ingestion of excess food can result from an emotional reaction to environmental situations.

**Body Weight Regulation**

The two sides of the energy equation, intake and expenditure are finely regulated by neutral and hormonal mechanisms.

The balance is maintained by an internal set point or ‘lipostat’ that can sense the quantity of the energy stores and appropriately regulate the food intake as well as the energy expenditure.
The current understanding of neurohumoral mechanisms involved in weight control are described which are experimentally proven.

It is established that adipocytes communicate with the hypothalamic centers that control appetite and energy expenditure by secreting a polypeptide hormone called 'leptin'. Leptin acts as an antiobesity factor. Leptin mediates its effects by binding to and activating leptin receptors in the hypothalamus. In experimental animals, triggering of the leptin receptor inhibits appetite and increases energy expenditure, physical activity and production of heat. Thermogenesis is controlled, at least in part, by leptin receptor – mediated hypothalamic signals that increases the release of norepinephrine from sympathetic nerve endings in the adipose tissue. The fat cells express β3 – adrenergic receptors that when stimulated by norepinephrine cause fatty acid hydrolysis and also uncouple energy production from storage. Thus the fats are literally burned and the energy so produced is dissipated as heat. There are other catabolic effects mediated by peptide, all transduced by its hypothalamic receptor, which in turn communicates with other endocrine glands through the hypothalamic pituitary axis.

The role of the leptin – leptin receptor system in the regulation of body weight is also supported by the observation that mutant mice lacking leptin receptors are massively obese. In db/db mice obesity occurs, even leptin levels are markedly elevated, because the leptin mediated afferent signals impinging on the hypothalamus fail to regulate appetite and energy expenditure.

Prominent among the hypothalamic mediators of leptin signaling is neuropeptide Y (NPY). This polypeptide increases appetite and inhibits sympathetic activity and the production of heat, thus favoring weight gain. It is postulated that leptin deficiency causes obesity by increasing the production of NPY, and conversely, signaling through the leptin receptor inhibits synthesis of NPY.

On the basis of studies, it is highly likely that dysfunction of the leptin system plays a role in human obesity. However, obesity caused by mutational inactivation of the leptin gene has been found in only few humans. Majority of obese humans have high level plasma leptin, indicating that there is some form of leptin resistance. Such resistance may be at the level of transport of leptin into the central nervous system. Defect exists is supported by the fact that despite high levels of serum leptin
in obese individuals, the level in the cerebrospinal fluid are not proportionately increased. In addition to defective transport, there may be abnormalities in hypothalamic pathways that are normally regulated by leptin.

In addition to the role of leptin in regulating body fat, it is also required for initiation of puberty and secondary sexual characteristics. These effects are mediated via the hypothalamic-pituitary axis.

Despite the rarity of well-defined genetic syndromes of obesity in humans, there is little doubt that genetic influences play an important role in weight control. Obesity is not merely a genetic disease, but environmental factors are definitely involved.

**PATHOPHYSIOLOGY OF OBESITY:**

**GENETIC SUSCEPTIBILITY**

Genetic determinations can either play a major role in the pathogenesis of obesity or enhance susceptibility to its development. The dysmorphic forms of human obesity in which genetics play a major role include the Prader Willi Syndrome, Ahlstroms Syndrome, Laurence – Moon – Biedl Syndrome, Cohen’s Syndrome and Carpenters Syndrome.

Four genes have been cloned in which mutations cause obesity in experimental animals and some of them appear to be important in human.

1. The leptine gene defect because of the presence of a stop condon that truncates the protein at amino acid 105.
2. Splicing defects in the leptine receptor.
3. Gene defect called tub results from a defective phosphatase.
4. Additional genes, including those for beta3 adrenergic receptor, tumor necrosis factor and lipoprotein lipase have been implicated in the development of human obesity

**FAT CELL AND OBESITY**

Fat cells produce lipoprotein lipase, which is involved in hydrolising the triglycerides of very low density lipoproteins (VLDL) and chylomicrons and
complements D and C3b. The adipocyte also produces cytokines such as tumor necrosis factor α, angiotensinogen, and leptin. The fat cell generates large quantities of lactate and metabolize glucose to provide glycerol – 3 phosphate for triglyceride synthesis.

Most forms of obesity are associated with enlarged fat cells and higher rates of basal lipolysis. Fat cells also serve as reservoir for storage of fatty acids released during the clearance of chylomicrons and can in turn release these stored fatty acids by the intracellular hormones sensitive lipase.

ENVIRONMENTAL FACTORS

Environmental factors interact with genetic susceptibility in the pathogenesis of obesity. For e.g.: hypothalamus injury from trauma or surgery and destructive lesions in the region of the ventromedial or the paraventricular nucleus in the hypothalamus can produce obesity. The two major factors in hypothalamic obesity are hyperphagia and distribution in the automatic nervous system activity.

NUTRITION INTAKE AND SUBSTRATE OXIDATION

To maintain normal body fat stores nutrients must be oxidized in the body in proportion in which they occur in the diet. The daily intake of carbohydrate equals body stores of glucose, carbohydrates stores are more vulnerable to changes in dietary carbohydrate than either fat or proteins. When dietary fat increases carbohydrate oxidation decreases to preserve carbohydrate stores, and oxidation of fat increases to provide nutrient needs. If body is unable to reduce carbohydrate oxidation, the compensatory mechanism is increased food intake to provide needed carbohydrate with increasing fat storage until a point is reached at which the oxidation of fatty acids increases to meet average dietary intake.

There are three known predictors of future weight gain: a low metabolic rate, a high oxidation of food, indicating carbohydrate oxidation and the need to eat to replace carbohydrate and insulin resistance.

There are some neurotransmitters, specific modulators of the intake of one or other macronutrient. So the increase or decrease in the intake of fat or protein or carbohydrate is response to the neurotransmitters. This controls the quality in quantity of food intake.

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OBESITY & ITS ASSOCIATED RISKS (Complications):

Obesity is a type of disease which invites many major and minor disease. It significantly shortens the life span and is associated with an increased incidence of a multitude of major and minor diseases. By consideration of health, the simplest statistic is that, a middle aged man who is 10 Kg overweight can expect to die roughly 4 years earlier than one normal weight, due to severe hazards which are associated with overweight. The relationship between body weight or body fat and medical complications curvilinear.

There is no question that severe obesity is a serious threat to life. Further more, obesity is associated probable – casually with other significant factors for coronary artery disease such as HBP, hyperlipidemia, Glucose intolerance as well as certain types of cancer. These disorders and obesity is self have a strong familial component. Less well-known complications include Hepatic stenosis, Gall bladder disease, pulmonary function impairment, endocrine abnormalities obstetric complications, Trauma to the weight bearing joints, Gout, Cutaneous disease, proteinuria, increased hemoglobin concentration and possibly immunological impairment. As a result of these conditions, relative weights (Rw) of 130% are associated with an excess mortality of 35% Rw of 130% have a greater than two fold excess death rate. Patients with morbid obesity (Rw>200%) have a greater than 10 fold increase in death rates.

Due to increased coagulability of blood, obese individuals also constitute an increased surgical risk. So the mortality rate for various surgical procedures is also high in the obse individuals. General Anesthesia also carries much higher risk.

In all these above mentioned hazards, perhaps the most common complication is Diabetes which in turn may lead to kidney failure. In the presence of over weight, there is an increased tendency to develop a variety of disease which are all classified in following mentioned systems.

1. Cardio Vascular System:
   a) Hypertension  
   b) Coronary Heart Disease  
   c) Myocardial Infraction  
   d) Left ventricular Hypertrophy  
   e) Generalized Athersclerosis  
   f) Peripheral Vascular disease  
   g) Atheroma  
   h) Premature cardiovascular death.
2. **Respiratory System** :

   a) Chronic Bronchitis
   b) Hypoxemia
   c) Alveolar hypoventilation
   d) Pickwickian syndrome (Obesity hypoventilation syndrome)
   e) Pulmonary hypertension
   f) Pulmonary embolism
   g) Obstructive sleep apnoea

3. **Gastro Intestinal Systems** :

   a) Gall Stone
   b) Hiatus Hernia
   c) Cholecystitis
   d) Pancreatitis
   e) Dyspepsia
   f) Diverticulosis of colon
   g) Reflux oesophagitis

4. **Endocrine and Metabolic system** :

   a) Diabetes mellitus
   b) Hyper Lipidaemia
   c) Hypo metabolic state
   d) Hyper Cholesterolaeemia.
   e) Fatty Liver
   f) Gout
   g) Polycystic Ovarian Syndrome

5. **Musculo Skeletal** :

   a) Osteo arthritis
   b) Backache

6. **Skin** :

   a) Ulceration
   b) Acanthosis Nigricans
   c) Fungal infection
   d) Fragilitas cutis inguinalis
   e) Erythema intertigo

7. **Miscellaneous** :

   a) Varicose Veins
   b) Delayed wound healing
   c) Technical – difficulty in surgery
   d) Risk in general Anaesthesia
Obesity particularly central obesity, increases the risk for a number of conditions, including diabetes, hypertension, hypertriglyceridemia, low HDL cholesterol and possibly artery disease. Some major risk factors are discussed below.

**DIABETES MELLITUS**

Obesity is the most powerful risk factor for NIDDM, with both its magnitude and duration being important. Studies also show that there is a strong correlation between relative weight and the prevalence of diabetes. In the development of diabetes mellitus, obesity precedes the onset of diabetes by month to years.

Study in Norway also shows that persons with greater weight have higher mortality because of diabetes.

In Pima Indians the increased risk of diabetes with obesity has a strong familial tendency; when one or both parents is diabetic, 100% offspring will develop diabetes if they become sufficiently obese.

Obesity predispose to carbohydrate intolerance by increasing insulin resistance, important features of NIDDM.

**CARDIOVASCULAR DISEASE**

In addition to increased work load on heart leads to dilation and hypertrophy which predispose congestive cardiac failure. The association between obesity and heart disease is not straight forward but the associated hypertension and diabetes. Obesity is also associated with an increased risk of sudden death, probably due to cardiac arrhythmias, and with increased risk of atherosclerosis, probably a reflection of an abdominal lipid profile including decreased levels of high-density lipoprotein cholesterol (HDL) and increased levels of low-density lipoprotein cholesterol (LDL).

There is increase in myocardial oxygen demand, when the supply cannot meet the demand, it results in infarction and death.

**HYPERTENSION**

Obesity is associated with hypertension, which can be reduces improvement in weight. Increase in cardiac out put and peripheral resistance is responsible for the hypertension.
The excess of insulin, in turn, may play a role in the retention of sodium, expansion of blood volume, production of excess norepinephrine and smooth muscle proliferation that are the hallmarks of hypertension.

**PULMONARY DISEASE**

Significant alteration in pulmonary function occurs in the severely obese and is due to increased oxygen consumption associated with breathing. The extreme form of pulmonary dysfunction is the Pickwickian syndrome or obesity hyperventilation syndrome which is characterised by somnolence, obesity and hyperventilation, hypoxaemia and CO$_2$ retention along with mechanical factors are responsible for the hyperventilation. The sleep apnoea in the obese may be central obstructive or mixed. Pulmonary hypertension, Polycythaemia and cor pulmonale result from prolonged pulmonary dysfunction.

**JOINT DISEASES**

Marked adiposity predisposes to the development of degenerative joint disease. Osteoarthritis is due to trauma of increased weight bearing, but arthritis also occurs in non-weight bearing joints also suggests that additional factors are involved. The prevalence of gout is also increased and may reflect impairment in urate clearance. Ketone bodies compete at the renal tubule for reabsorption of urate, and increased production of ketones from fat metabolism may increase urate levels.

**ENDOCRINE DISORDERS**

In men, there is consistent reduction in the concentration of total serum testosterone. Obesity is associated with clinical symptoms suggestive of abnormal ovarian function, including irregular menstruation, secondary amennorhoea, hirsutism, early menarche and delayed menopause.

The serum free testosterone level has been reported to be elevated in obese women and this hyperandrogenaemia is said to be responsible for irregular menses and hirsutism.
CANCER

The incidence of endometrial and postmenopausal breast cancers in women, prostate cancer in men, and colorectal cancer in men and women is related to the degree of obesity. Visceral obesity increases the risk of postmenopausal breast independent of degree of obesity. Both in pre and postmenopausal women circulating androgens from the adrenal gland are converted to oestrogens in the peripheral tissue. The enhanced production of oestrogen may have role in high incidence of endometrial carcinomas and breast cancer in obese women.

GALL BLADDER DISEASE

Gall bladder disease increases with obesity and age, possibly related to increased total body cholesterol, increased cholesterol turnover and augmented biliary excertion of cholesterol in the bile. Disturbances in nidation factors in the bile and alterations in the level of bile acids and phospholipids may promote precipitation of cholesterol stones.

SKIN PROBLEMS

Among the skin problems associated with obesity are acanthosis nigricans, manifested by darkening of skinfolds on the neck, elbows and dorsal interphalangeal spaces. Acanthosis niogricans is also associated with insulin resistance and NIDDM. Skin turgor and friability may also be increased obesity, enhancing the risk of fungal and yeast infection in skin folds.

METABOLIC ALTERATIONS IN OBESITY

Adiposity is known to be associated with metabolic abnormalities. In most of the obese subjects metabolic alterations appear to be secondary to the obese state. Commonest abnormality is in glucose metabolism resulting from an acquired insulin resistance at the periphery. Various lipid metabolism abnormalities are also recorded. Disorders in triglycerides and free fatty acid metabolism are the most significant. Obesity also alters the cholesterol metabolism to some extent. Metabolic disturbances appear to correlate only to fat cell size, and virtually all metabolic disturbances and induced by weight gain, and are reversible after weight reduction.
ALTERATION IN GLUCOSE METABOLISM

Obesity is commonly associated with carbohydrates intolerance. The probability of developing Non-insulin dependent diabetes is correlated with duration of adiposity, time of onset on serverity of carbohydrate intolerance depends on variety of interacting genetic environmental factors.

This intolerance to glucose either improves or very often disappears after a successful weight reduction. Thus the incidence of glucose intolerance in obese state is due to increased adipose cell mass.

ALTERATION IN INSULIN SECRETION AND ACTION

In obesity there is hyperinsulinemia and presence of peripheral insulin resistance. The knowledge of this is important because its apparent relationship with the development of decreased glucose tolerance and diabetes mellitus is frequently associated with obesity. One of the consequences of this resistance to insulin appears to be feed back compensatory hyperinsulinism. The beta cells of pancreatic islet are stimulated by unknown mechanism to produce more insulin and beta cell hypertrophy results. Decreased in insulin clearance by the liver may contribute to the hyperinsulinemia of obesity particularly central obesity.

Inspite of high insulin levels, obese subjects do not become hypoglycaemic, suggesting that they may have insulin resistance. Me studies show that inotial stages of insulin action on target tissues, namely insulin binding to cells seems to be impaired in obesity, which reflects in turn, decrease in insulin receptors and insulin resistance.

Lipids are a group of heterogeneous substances metabolically active constantly moving in the circulation and existing in a state of dynamic equilibrium between peripheral tissues, gastro -intestinal tract and liver. Triglycerides, cholesterol, free fatty acids and phospolipids constitute the plasma lipids. Cholesterol and phospholipids constitute about 2/3 of the total plasma lipids, whereas FFA is metabolically most active.

The FFA is utilized for energy purposes or resynthesis of Tg in the adipose tissue while glycerol reached the liver to be utilized for endogenous Tg synthesis.
Considering the raised levels of Tg, cholesterol or both and the lipoprotein (Lp) molecule which is in excess in the circulation, hyperlipoproteinemias have been classified by Fredrickson into give major groups.

Table.No.23-

<table>
<thead>
<tr>
<th>Type</th>
<th>Chol.</th>
<th>TG</th>
<th>LDL</th>
<th>VLDL</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>N to ↑</td>
<td>↑↑↑</td>
<td>N</td>
<td>↑</td>
</tr>
<tr>
<td>II a</td>
<td>↑↑</td>
<td>N</td>
<td>↑</td>
<td>N</td>
</tr>
<tr>
<td>II b</td>
<td>↑↑</td>
<td>↑↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>III</td>
<td>↑↑</td>
<td>↑↑↑</td>
<td>↑↑</td>
<td>↑</td>
</tr>
<tr>
<td>IV</td>
<td>N to ↑</td>
<td>↑↑</td>
<td>↑↑</td>
<td>↑</td>
</tr>
<tr>
<td>V</td>
<td>↑↑↑</td>
<td>↑↑↑</td>
<td>↑</td>
<td>↑</td>
</tr>
</tbody>
</table>

The concentration of high-density lipoprotein in the blood is greater in women than in men. Jogging and other forms of exercises and a moderate intake of alcohol increase the concentration of high density lipoproteins in plasma. It has been demonstrated that patients with myocardial infarction, diabetes mellitus and other diseases depress the level of high density lipoproteins (Their approximate mean composition is shown in table No.24–
### Table 24: Composition and characteristics of the human plasma lipoproteins.

<table>
<thead>
<tr>
<th></th>
<th>Chylomicrons</th>
<th>VLDL</th>
<th>LDL</th>
<th>HDL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protein</td>
<td>2</td>
<td>7</td>
<td>20</td>
<td>50</td>
</tr>
<tr>
<td>(0% particle mass)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Triacylglycerols</td>
<td>83</td>
<td>50</td>
<td>10</td>
<td>8</td>
</tr>
<tr>
<td>(0% particle mass)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cholesterol</td>
<td>8</td>
<td>22</td>
<td>48</td>
<td>20</td>
</tr>
<tr>
<td>(0% particle mass)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(free + esterfied)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phospholipids</td>
<td>7</td>
<td>20</td>
<td>22</td>
<td>22</td>
</tr>
<tr>
<td>(0% particle mass)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Particle mass (daltons)</td>
<td>0.4-30x10^6</td>
<td>10-100x10^6</td>
<td>2-3.5x10^6</td>
<td>1.75-3.6x10^6</td>
</tr>
<tr>
<td>Density range (g/ml)</td>
<td>&gt;0.95</td>
<td>0.95-1.006</td>
<td>1.019-1.063</td>
<td>1.063-1.210</td>
</tr>
<tr>
<td>Diameter (nm)</td>
<td>&gt;70</td>
<td>30-90</td>
<td>18-22</td>
<td>5-22</td>
</tr>
<tr>
<td>Apolipoproteins</td>
<td>A1, B-48</td>
<td>B-100,E</td>
<td>B-100</td>
<td>A1,A2</td>
</tr>
<tr>
<td></td>
<td>C1,C2,C3</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
ALTERATIONS IN LIPID METABOLISM

(A) FREE FATTY ACID METABOLISM

Lipid metabolism is greatly affected in obesity. It has been found that when obese persons are starved there the rise in plasma levels of free fatty acids. Some response is observed after muscular exercise and cold exposure also. This may be due to enhanced lipolysis because of adipose tissue hyperplasia or concomitant glucose intolerance. Thus in obese patients the larger part taken by lipid in their total energy metabolism is a consequence of their increased adipose mass.

(B) TRIGLYCERIDE METABOLISM

Triglyceride, metabolism abnormalities are one of the consequences of obesity. The major function of the adipose tissue is the storage of energy in the form of triglyceroids and its release in the form of fatty acids. Some studies show that hypertriglyceridemia is associated with increased carbohydrates intake, while in some studies it was observed that circulating insulin levels are significantly associated with plasma triglyceride levels and since insulin is one of the factors involved in endogenous triglyceride rich lipoprotein secretion in liver.

(C) CHOLESTEROL METABOLISM

Disorders in cholesterol metabolism are also associated with obesity. Fat cell play an active role in cholesterol uptake from circulation and are the major storage site for cholesterol. Large fractions of cholesterol is stored in the unesterified from, in obesity the total body cholesterol is augmented. The degree of obesity is related with increase in cholesterol production rate and show elevated levels of serum cholesterol.

Raised to levels suggest an increase in VLDL and/or chylomicrons levels while an isolated rise in cholesterol indicates rise in LDL level.

Thus from metabolic alterations alone it would appear appropriate, to define obesity in term of adipose tissue cell size.
In modern medical science no premonitory symptoms of obesity have been described so, overweight and its related initial or mild symptoms can be taken as premonitory symptoms. According to modern medicine, the stage of localization can be understood by preferential deposition of adipocytes in specific depots under influence of some adipocyte enzymes adrenoreceptor endocrine secretion and protein of ob gene.

Lipo protein lipase (LPL) also plays an important role in controlling the regional deposition of fat. Women have more LPL in the gluteal and femoral adipose tissue, which has bigger fat cells than they do in abdominal adipose tissue. In contrast obese men show minimal regional variations adipose tissue LPL activity or fat cell size. Regional variations in receptor for glucocorticoids or sex hormones, steroids may play a role regional differences in LPL activity. Enhanced LPL activity in femoral and gluteal tissue tend towards lower body or gynoid adiposity in women.

In certain endocrinopathies and genetic syndromes also there is a preferential deposition of fat in different depots occurs. In the initial stage of disease by observing regional variation in fat deposition one can predict up coming disorder.

The clinical spectrum of obesity as mentioned in various text books of modern medicine are as under. But its manifestation depends upon a variety of factors. In most cases, the diagnosis will be apparent from the patient’s appearance.

- Obesity can be diagnosed from the gain in weight.
- In addition to this the development of skinfold is observed in the obese individual specially around the axilla; under the breast, perineal region and almost protruberant abdominal wall.
- The belly is sticking out due to excessive accumulation of fat in the abdominal wall so protrusion of abdomen is a more common clinical feature; which is observed in most of obese person.
- General lassitude, dyspnocia, on excretion, aches and body pain are also observed in obese person as the clinical features.
- Varicose veins and oedema of the ankles are most trouble some features of obese person.
• Gallstones are also observed as clinical feature in obese person.
• In obese person possibility of fungal infection is greater in the skinfold areas.
• Liver is also palpable due to the deposition of fat.
• There may be symptoms associated with Diabetes Mellitus, hypertension Pickwikean syndrome that are observed.
• In fatty women sterility is also observed.
• And some diseases, which are produced as the complication of obesity, are more often observed as the clinical feature in an obese person like Dyspesia, Bronchitis, Hernia etc.

PROGNOSIS:

It is easy for an obese person to lose upto 5 Kg. Weight, but to achieve further weight loss and to maintain it is very difficult. Statistical studies showed that if 12 Kg. Or more than 12 Kg weight reduction was kept as criteria for successful treatment, the result was only 12 to 28 percent, with a stricter standard (the loss of over 20 Kg.) the success amounted to only 2-8%. Short term treatment of obesity with drug is generally not warranted because obesity is a disorder that can not be expected to remit without continued treatment and many of the times even though with best efforts many obese patients obstinately remained unchanged. The death rate from secondary metabolic disorders is 4 to 5 fold higher in obese than normal weights. The expectation of life of obese individual is much shorter than normal weights. Very roughly, the expectation of life decreases by 1% below the normal for every \( \frac{1}{2} \) Kg weight above normal.

COMPLICATING FACTORS

Here the complicating factors include-

1) Elevated abdominal:gluteal ratio- In male more than 0.95 and in female more than 85
2) Medical Complications like -Diabetes Mellitus, Hypertention, Hyperlipidemia
BMI < 25  
Yes  

BMI < 25 - 30  
Yes  

BMI < 30 - 35  
Yes  

BMI < 35 - 40  
Yes  

BMI > 40  
YES  

LOW RISK

MODERATE RISK

HIGH RISK

VERY HIGH RISK
EPIDEMOLOGY (PREVALENCE OF OBESITY)—

A number of factors influence body fat including age, sex, race, socio economic class etc.

Age:

Obesity is most prevalent in middle age but can occur at any stage of life. Obesity in childhood and adolescence is likely to be followed by obesity in adult life. After the age 30 lean body mass starts to decline the specific action of growth hormone and is replaced by fatty mass. Most common form of obesity in middle age is prevailed due to this phenomenon. Fat increases in both sex. Puberty and during adult use3 rises between 30 to 40 percent of body weight. Between ages 20 to 50, fat content of men approximately doubles and that of women increase by about 50 percent.

Sthoulya can emerge at any stage of life. More indulgence of Madhura Ahara by a pregnant lady may tends towards birth of obese child (Cha. Sa. 8/21) and this type of childhood obesity may run for life long.

Sex:

Normally the women are more prone to be obese than men. The young adult women contains fat approximately 15% of body weight and it is about more than young man. In that phase of puberty and adolescent due to hormonal changes more fat accumulates in body particularly in femoral, gluteal and breast region especially in females. Which is considered as sign if Yauvana.

Female have innate tendency and large fat cells to accumulate more fat in above regions. Sex hormones also plays an important role in this type phenomena. Moreover puberty, pregnancy, menopause and cyclic hormonal changes attributes towards obesity in females.

According to Ayurvedic concepts meda is matrija bhava (Cha. Sa. 3/6) i.e. female dominant proportionate of the body, which indicates dominance of fat in females.
Race:

The native of tropical countries are susceptible to become corpulent by reason of excess water intake, lack of exercise and excessive consumption of alcohol and food.

At present Gujrat is second largest state in country regarding prevalence of obesity and heart diseases. Every 6 person out of 10 have either generalized obesity or abdominal adiposity which is thought to be more devil than previous one. Obesity in Sindhi community is also very obvious.

Ethnic tendency towards abdominal adiposity, high TG levels and low HDL-C levels in south Asians particularly in Asian Indians is very well established and well documented and is a root cause of CVD and premature mortality in this population. At present 44 million Indians fall under group of higher risk cardiovascular disease and tend to increase due to obesity but have no national agenda or any task force to prevent this premature deaths. Neither physicians nor pot belly peoples are aware of this critical condition and are ignoring their fat layers by considering it as money belt, love buckles etc.

Certain races are more prone to become fat than others, e.g. the Dutch, South Germans, South Italians, Maltese, Herbews, the native of India Ceylon and some African races.

Using BMI it is possible to compare the prevalence of obesity in several countries.
Table No. 25: Prevalence of overweight in several affluent countries

<table>
<thead>
<tr>
<th>Country</th>
<th>Prevalence of Age (yrs)</th>
<th>Percent Men</th>
<th>BMI 25-30 women</th>
<th>Percent Men</th>
<th>BMI &gt; 30 Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>North America</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>United States</td>
<td>20-74</td>
<td>31</td>
<td>24</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td>Canada</td>
<td>20-69</td>
<td>40</td>
<td>28</td>
<td>9</td>
<td>12</td>
</tr>
<tr>
<td>Europe</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Great Britain</td>
<td>16-65</td>
<td>34</td>
<td>24</td>
<td>6</td>
<td>8</td>
</tr>
<tr>
<td>Netherlands</td>
<td>&gt;20</td>
<td>34</td>
<td>24</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>Australia</td>
<td>25-64</td>
<td>34</td>
<td>24</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>Urban India</td>
<td>25-64</td>
<td>30</td>
<td>34</td>
<td>8</td>
<td>10</td>
</tr>
</tbody>
</table>

The prevalence of individual with BMI of 25 to 30 Kg/m² is almost identical in all of the population.

The prevalence of those with BMI above 30 Kg/m², however, is higher in both the United States and Canada than other countries.

By now it is well established that obesity in the United States is reaching epidemic properties. The world Health Organization (WHO) and the International Obesity Task Force (IOTF) have declared an obesity epidemic on a global scale. As the IOTF puts it, Obesity, which increase the risk of developing such potentially, fatal conditions such as DM and heart disease, “Poses one of the greatest threats to human health and well being as 21st Century approaches”

The data from the National Health survey suggests prevalence of obesity epidemic on a global scale.
Table No.26: Prevalence of obesity epidemic

<table>
<thead>
<tr>
<th>Country</th>
<th>Year</th>
<th>% Men</th>
<th>% Women</th>
<th>Year</th>
<th>% Men</th>
<th>% Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>U.K.</td>
<td>1980</td>
<td>6.0</td>
<td>15.0</td>
<td>1994</td>
<td>8.0</td>
<td>16.5</td>
</tr>
<tr>
<td>Brazil</td>
<td>1976</td>
<td>3.1</td>
<td>5.9</td>
<td>1989</td>
<td>8.2</td>
<td>13.3</td>
</tr>
<tr>
<td>Canada</td>
<td>1978</td>
<td>6.8</td>
<td>12.0</td>
<td>1992</td>
<td>9.6</td>
<td>14.0</td>
</tr>
<tr>
<td>East Germany</td>
<td>1985</td>
<td>13.7</td>
<td>20.5</td>
<td>1992</td>
<td>22.2</td>
<td>26.8</td>
</tr>
<tr>
<td>Australia</td>
<td>1980</td>
<td>9.3</td>
<td>11.5</td>
<td>1989</td>
<td>8.0</td>
<td>13.2</td>
</tr>
<tr>
<td>Western Samoa</td>
<td>1978</td>
<td>38.8</td>
<td>58.4</td>
<td>1991</td>
<td>59.1</td>
<td>76.8</td>
</tr>
<tr>
<td>Thailand</td>
<td>1985</td>
<td>2.2</td>
<td>3.0</td>
<td>1991</td>
<td>3.0</td>
<td>3.8</td>
</tr>
<tr>
<td>Mauritius</td>
<td>1987</td>
<td>3.2</td>
<td>5.3</td>
<td>1992</td>
<td>10.4</td>
<td>15.2</td>
</tr>
</tbody>
</table>

These epidemiological studies reveals influence of Race on various aspects of obesity like dietetic factors, physical activities, socioeconomic conditions, genetics etc.

**Socio Economic Status:**

It is believed that obesity is a disease of only upper socioeconomic class, but it is not so various survey studies have indicated towards the increased prevalence of obesity in middle and lower socioeconomic classes. Starchy foods providing bulk of the cheap meals, and fatty foods with alcohol causes obesity in lower and upper socioeconomic classes respectively.

It is clear that the prevalence of obesity is not related with quantum of money it depends upon made and code of life style and eating habits. Obesity may occur in any class of society which tends towards overeating and sedentary habits.

**Urbanization:**

The prototypic convenient foods are preparation of fat and sugar such as biscuits, Urban population constantly consumes these foods. In addition, urban civilization also reduces the need for strenuous physical exercise. These trends increase energy intake and decrease energy output leading to obesity.
Smoking:

It has been demonstrated that exposure to Nicotine increases metabolic rate. Cigarette smoking suppresses body weight, discouraging many smokers from trying to quit. The study of Williamson D.F. and others confirmed that discontinuing smoking causes weight gain in 80% at ex-smokers by about 2.3-4 Kg. With the gain slightly greater among females than males.

Environmental features:

Actually environmental factors are not cause of obesity but they are only subordinate factors heredity. A variety of environmental factors are needed to display this susceptibility.

There is also some evidence to indicate that obese persons are insensitive to internal stimuli regulating hunger and satiety and respond more to external stimuli such as the appearance, aroma, and taste of foods. As a result, the feeding behaviour of obese persons is dominated by non physiological external stimuli.

TREATMENT OF OBESITY AS DESCRIBED IN MODERN MEDICAL SCIENCE:

Obesity occurs when caloric intake exceeds the metabolic expenditure. The aim of therapy is to modify this disparity by decreasing intake and increasing expenditure. This involves changes in the individual’s lifestyle. Thus treatment is difficult and patient needs motivation.

The treatment in all includes six phases

1) Patient counselling 4) Exercise therapy
2) Behaviour therapy 5) Drug therapy
3) Dietary management 6) Surgical treatment

1) Patient counselling:

Patient counselling is of most importance for the treatment of an obese person. Under this heading detail knowledge of the disease needs to be given to the obese
person. An obese person should be explained about the cause and treatment of this disease. Emphasis must be put on to reduce the fat instead of weight loss. The primary goal of the treatment is to lose body fat while maintaining muscle or lean body mass. The problem is that calories are stored in fat and during the diet, the body protects itself by conserving calories and burning muscle instead. Lost muscle lowers metabolism, (the rate at which the body burns the calories) making it even more difficult to lose weight.

A kilo at muscle burns 70 times as many as a kilo of fat. So, to achieve weight loss, it is vital retain and build muscle. Increasing muscle mass nearby 1.5 kg., makes the body of person to burn 150 to 300 more calories a day, regardless of his activity level.

The real key is not what one weighs but where the weight reduction comes from Kilo for Kilo, muscle is \( \frac{1}{7} \) the dimension of fat. When one exercise and put on a few kilos of muscle mass, his weight mat not change much, but his clothes will fit better and those will be in smaller sizes. Patient must be made aware of the role of diet in accelerating weight. So emphasis must be put on dietary reduction than restriction, continuity of treatment, re-enforcement regular follow-up in the Clinic. At the same time, patient must be displayed confident opinion concerning the successful out come of the prescribed programs as well as importance of gradual weight loss therapy instead of drastic weight loss. The weight loss will be very rapid in early period of dieting and then the weight loss is gradual. This should be explained to patients to avoid disappointment.

2) **Behaviour Therapy:**

Behaviour Therapy is a team which covers wide variety of treatments at approached. This therapy is based on an attempt to produce permanent changes in behaviour by involving the patient in his own management. Obese persons are addicted by some behavioural pattern. This living pattern invites obesity, so that this therapy guides the patients to observe his current life style, eating habit, activities etc. and encourage to change it. The programme includes monitoring intake, modifying causes that signal "Inappropriate eating ", modifying the act of eating itself, increasing exercise and self reward for more appropriate behaviour. Self-monitoring means keeping a written record of every thing eaten the circumstances in
which it was eaten. A food diary or what we call it as "A Dear Diary" helps keep track and by deciding what to eat when a dieter can avoid the rushed decisions that can lead to over eating.

Elimination the cases that signal inappropriate eating include controlling the environment in which the person is e.g. changing habits such as snacking while watching TV or studying, eating at late nights. These are all common problems of an obese person. It is observed that obese individuals respond less well, than normal ones to internal cues that regulates eating behaviour such as gastric contractions, fear and previous food ingestion. Conversely, obese subjects over respond to external cues such as taste, small food attractiveness, food abundance, and the ease of obtaining food. The increase the prevalence of obesity is attributed to several including decrease in energy expenditure and increase in the availability and consumption of processed and refined foods containing a variety of nutritive and non-nutritive sweetness. An increasingly large proportion of refined sugar is consumed through hidden sources such as processed for and beverage. Dietary advice to stay away from "Fattening" carbohydrates and to eliminate snacking between the meals would appear reasonable.

3) Dietary Management:

Diet plays an important role in obesity in the prevalence of obesity. Nutrition is (in addition to genetics), probably the single most important stimulus of the marked stimulatory effect of the high fat diet on lipid accumulation and enlargement of fat is not yet totally elucidated. It may reflect adaptive hormonal changes, adaptive enzymatic changes, or simply the greater efficiency of fuel storage in the form of lipid if the predominant dietary component is fat, as compared with carbohydrate or protein.

Even though it is believed that over nutrition at any stage of life increase the fat cell size, thus creating the conditions for new recruitment of fat cells ideal size reaches the critical level, it is not known whether an increase in cellularity can be produced throughout the life span of mammals.

The effect of dietary compositions have recently received attention. It has been shown that a high fat diet reduces fatty acid synthesis from glucose in fat cells. This is not surprising since one would expect that greater availability of free fatty acids
(FFA) from circulating lipids and lipoproteins may suppress. De novo fatty acid synthesis in adipocytes.

When planning diet for an obese person, a weekly weight loss at 0.5 to 1 kg should be aimed. Thus an obese middle age house wife lose weight satisfactorily an addict providing 800 to 1000 Kcal per day where as for an obese man engaged in active physical work, the diet containing about 1500 kcal per day is require. The diet should contains: Protein – 50gm, Carbohydrate – 100 gm, Fat 40gms., Supplements of Vitamin A & C, Minerals like Iron and calcium, Fluids, Salts.

The aim of designing a diet should be reduction of calories, to maintain health to establish a sense of well being and to minimum hunger. A simple way to calculate for an individual is to allow 15-20 calories per Kg of “Ideal weight”.

Omitting refined sugar brings about reduction of the energy value of the diet and by restricting the intake of fat and foods high is starch. Rapid weight loss should be discouraged, even though it is what every obese patient wants. If weight loss is rapid, not only the fat metabolism but also the muscles and liver are also depleted. Water and sodium diuresis accompanies it.

Diet plan must be prepared for the patients by matching calories more efficiently as he refuels early in the day with a low fat meal. Late night eating must be avoided. Metabolic needs are lowest at night, calories consumed are then tend to be stored as fat. Total cut of fats is not expected in the diet regiment. Even this dietary villain has a virtue. It gives the body, satiety. For obese person, the best diet is that which contains all the normal ingredients of food but cuts down only the total calories. It has been shown that weight loss more successful in the patient who take their diet as multiple meals than in those who take the same intake in one or two meals per day.

Diet containing liberal amounts of salads, fresh fruits and vegetables, dietary fibers such as whole grain, cereals is advisable. The bulk of vegetables and fruits containing few calories but high cellulose help to fill the stomach and relives hunger, minimizes the constipation and provides Vitamin A and Vitamin C content to meet the body’s needs.

A chart of dietary calorie value is given below, which will be very helpful in planning diet schedule for an obese person as given below.
### Table No.27: Diet & Calories*

<table>
<thead>
<tr>
<th>Foodstuffs</th>
<th>Calories</th>
<th>Protein (Thiamine)</th>
<th>Iron</th>
<th>Vitamin A (Thiamine)</th>
<th>Vitamin B1</th>
<th>Vitamin B1</th>
<th>Niacin</th>
<th>Vitamin C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maize</td>
<td>125</td>
<td>4.7</td>
<td>1.1</td>
<td>54</td>
<td>0.11</td>
<td>0.11</td>
<td>0.6</td>
<td>6</td>
</tr>
<tr>
<td>Ragi</td>
<td>328</td>
<td>7.3</td>
<td>17.4</td>
<td>70</td>
<td>0.42</td>
<td>0.19</td>
<td>1.1</td>
<td>0</td>
</tr>
<tr>
<td>Rice (milled)</td>
<td>345</td>
<td>6.8</td>
<td>3.1</td>
<td>0</td>
<td>0.06</td>
<td>0.04</td>
<td>1.9</td>
<td>0</td>
</tr>
</tbody>
</table>

#### 4) Exercise Therapy:

Research shows that exercise for 10 minutes at a stretch 4-5 times a day is as beneficial as exercise for 40-50 minutes a time. Exercise in the morning is suggested for keeping metabolism higher all the day, studies show that an exercise induced metabolic boost can last 24 hours or longer. Though exercise alone is not adequate to lose weight, however it is an adjunct to calorie reduction. It is very useful to expend calories reduction, It is very useful to expend calories and to increase lean body mass instead of Adipose tissue. The most common method for promoting caloric expenditure in obese is to practice regular exercise. Patient should be emphasized to start with light exercise, gradually increase it and then maintain it regularly. In nutshell, jogging, walking, swimming, aerobic exercise i.e. exercise in open air are considered better for obese persons. Unfortunately the dislike of physical activity or any type of exercise is the characteristic feature of an obese patient.

#### The Role of Exercise:

The weight reduction by itself can improve the physical work capacity and metabolic complication associated with obesity, it is not clear to what, physical training contributes to the benefit effects of weight reduction.
Some studies has shown that physical exercise:

- Accelerates the rate of weight loss.
- An effect on body composition by increasing the loss of adipose tissue and minimizing the amount of body cell mass.

Metabolic effects manifested by a greater –

a) decrease in insulin levels
b) increase in insulin sensitivity
c) decrease in serum triglyceride level.
d) Increase in HDL cholesterol
e) Improved physical work capacity.

A practical consideration for any one contemplating exercise as a means of weight reduction is, what form of exercise is to be selected and how to implement it. A person intent on faster losing as weight as possible, may make the reasonable assumption that increasing the speed of running will increase the level of energy expenditure.

This assumption is wrong for two reasons:

i) Setting a running pace at an exercise speed will call for oxygen supply that the ventilatory and respiratory mechanism cannot supply. Such an individual will therefore rely on limited aerobic sources of energy and prevent prolonged utilization of fat fuel, which required an abundant supply of oxygen.

ii) While in walking, magnitude of energy expenditure is proportional to the speed. This is not the case of running. Energy cost of running one mile (1.6 km) is 0.9 – 1.5 Kcal/kg body weight and its independence of the speed of running.

Weight loss regardless of speed will be proportional to the distance run. Running shorter distance at high speed will not result in equivalent loss of calories as running longer distances at lower speeds despite different levels of fatigue and subjective perception of effort in the two situations.
The most important fuel for exercise is carbohydrate, because it can be utilized rapidly and regardless of the adequacy of oxygen supply to the working muscle. Carbohydrates are utilized through anaerobic glycolysis, when supply of oxygen to the muscle falls short of the energy requirements of exercise. Fat cannot be oxidized when the oxygen supply to muscle is inadequate and thus anaerobic carbohydrate utilization is obligatory at the start of the exercise before the sympathetic nervous system, fully activates, ventilatory and circulatory adjustments that ensure the shunting of oxygen enrich blood to the working muscle.

The most abundant fuel for exercise is body fat. Whenever oxygen delivery is sufficient for energy needs of exercise, body fat will be preferentially used as energy for physical activity. At the cellular level, metabolic substrates and energy rich metabolites that are generated during aerobic oxidation of lipid, suppress utilization of carbohydrate by reducing the activities of glycolytic enzymes—golphfructokinase, pyruvate dehydrogenase. Utilization of body lipids will proceed at a high rate, when we set a pace of exercise at a level at which our ability to supply oxygen to the working muscles is not a limiting factor.

A brief comparison of the related amounts of carbohydrates and lipid fuels should bring home the point that endurance, physical activities are the only form of exercise that can tap large supply of energy stored in the fat depot. Moderate pace of endurance physical activities, ensures that oxygen delivery meets the oxygen demand of exercise and that body fat will be oxidized aerobically. Whenever the physical activity is set too high of an individual's capacity to supply working muscles with oxygen metabolism with shift to utilization of carbohydrates and upon their early depletion, the fatigue will set in and curtail exercise and lipid utilization.

The success of using endurance physical activity as a means of weight control, will be further enhanced by emphasizing that in running energy loss is a function of distance run and not at speed of activity and that utilization of body fat is possible only at low to moderate intensities of exercise at which adequate supplies of oxygen are supplied to working muscles.
### Table no.28: Energy need for every day activities

<table>
<thead>
<tr>
<th>Activities</th>
<th>Calory Consumption per minute</th>
<th>Activities</th>
<th>Calory consumption per minute</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Self-Care Activities</strong></td>
<td></td>
<td><strong>Recreational Activities</strong></td>
<td></td>
</tr>
<tr>
<td>Rest, lying face upwards</td>
<td>1.0</td>
<td>Painting sitting</td>
<td>2.0</td>
</tr>
<tr>
<td>Sitting</td>
<td>1.2</td>
<td>Car driving</td>
<td>2.8</td>
</tr>
<tr>
<td>Standing (Easy posture)</td>
<td>1.4</td>
<td>Slow horse riding</td>
<td>3.0</td>
</tr>
<tr>
<td>Eating</td>
<td>1.4</td>
<td>Volley ball</td>
<td>3.5</td>
</tr>
<tr>
<td>Talking</td>
<td>1.4</td>
<td>Bowling</td>
<td>4.4</td>
</tr>
<tr>
<td>Dressing, undressing</td>
<td>2.3</td>
<td>Cycling to verify</td>
<td>4.5</td>
</tr>
<tr>
<td>Washing hands face</td>
<td>2.5</td>
<td>Golfing</td>
<td>5.0</td>
</tr>
<tr>
<td>Walking to verify</td>
<td>3.6</td>
<td>Swimming 18 mtrs/min</td>
<td>5.0</td>
</tr>
<tr>
<td>Shower bath</td>
<td>4.2</td>
<td>Dancing</td>
<td>5.5</td>
</tr>
<tr>
<td>Walking downstairs</td>
<td>5.2</td>
<td>Gardening</td>
<td>5.6</td>
</tr>
<tr>
<td>Horse Hold Work</td>
<td></td>
<td>Tennis Playing</td>
<td>7.1</td>
</tr>
<tr>
<td>Sewing by hand</td>
<td>1.4</td>
<td>Horse Trotting</td>
<td>8.0</td>
</tr>
<tr>
<td>Floor Sweeping</td>
<td>1.7</td>
<td>Skiing</td>
<td>9.9</td>
</tr>
<tr>
<td>Furniture Polishing</td>
<td>2.4</td>
<td>Squash</td>
<td>10.2</td>
</tr>
<tr>
<td>Small cloth washing</td>
<td>3.0</td>
<td>Professional Activities</td>
<td></td>
</tr>
<tr>
<td>Scrubbing Floors</td>
<td>3.6</td>
<td>Sewing by machine</td>
<td>2.9</td>
</tr>
<tr>
<td>Making Beds</td>
<td>3.9</td>
<td>Brick placing</td>
<td>4.0</td>
</tr>
<tr>
<td>Ironing</td>
<td>4.2</td>
<td>Plastering</td>
<td>4.1</td>
</tr>
<tr>
<td>Hanging washed clothes</td>
<td>4.5</td>
<td>Tractor Ploughing</td>
<td>4.2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Carpententry</td>
<td>6.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hand mowing of lawn</td>
<td>7.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Shovelling</td>
<td>8.0</td>
</tr>
</tbody>
</table>
5) **Drug Therapy:**

Recently an alteration medically based outcome measure for obesity treatment has been advocated by scientists and physicians. Rather than focusing primarily on body weight, body fat or BMI (weight / height\(^2\)), this measure called metabolic fitness, tracks the metabolic health of obese individuals.

Metabolic fitness is defined as the absence of biochemical risk factors associated with obesity, such as elevated fasting concentration of cholesterol, triglycerides, glucose or insulin, impaired glucose tolerance or elevated blood pressure.

Interestingly, reduction in the biochemical risk factors may not always be dependent on weight loss, e.g. Insulin, sensitivity and cholesterol levels can be improved by physical activity in the absence of weight loss.

The hope is that by using metabolic fitness as a measure of success, health professionals can shift the patient's focus from unrealistic, culturally imposed goals, (e.g. Dress size or Belt size) to the more appropriate and achievable goal of better health. Anti obesity drugs can be classified according to their primary mechanism of action on energy balance. When daily energy intake, matched daily energy expenditure, body weight remains constant. If intake exceeds expenditure, then a state of positive energy balance is achieved; body weight will increase. Conversely if, energy expenditure exceeds intake, then a state of -ve energy balance is achieved and the body weight will decrease. The goal of anti obesity drugs is to induce and maintain the state of -ve energy balance until the desired weight loss is achieved.

There are four general classes of anti obesity drugs.

(a) Inhibitors of energy (food) intake, or (appetite suppressants) reduce hunger perception increase the feeling of fullness and reduce the food intake by acting on brain mechanism. As a result these drugs facilitate compliance with calories restrict.

(b) Inhabitants of fat absorption reduce energy intake though a peripheral Gastrointestinal mechanism of action and do not alter Brain Chemistry.

(c) Enhances of energy expenditure act through peripheral mechanisms to increase thermo genesis without requiring planned increases in physical activity.
(d) Stimulators of fat mobilization act peripherally to reduce fat mass or decrease triglyceride synthesis or both without requiring planned increases in physical activity or decreases in food intake.

Importantly, the beneficial actions of all four drug classes can be easily overcome by increase intake of food (especially calorically dense food items) or decreased voluntary physical activity.

The major drugs to treat obesity are shown in the following table. Currently the only drugs approved for use are a small set of centrally acting appetite suppressant that reduce food intake by modulating the concentrations of monoamine neurotransmitters (serotonin and norepinephrine or norpinephrine alone) in the brain. This modulation can occur at the level of neurotransmitter release or reuptake or both. The identification of the specific subtypes of serotonin receptors involved in the regulation of food intake is a major focus of research. Appetite suppressants generally produce an average weight loss of about 10% initial body weight.

**Classes of Anti – Obesity drugs:**

**Table No.29: Inhibitors of Fat Absorption**

<table>
<thead>
<tr>
<th>No.</th>
<th>Drug</th>
<th>Target</th>
<th>Mechanism</th>
<th>Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Orlistat</td>
<td>Pancreatic Lipase</td>
<td>Inhibits fat absorption</td>
<td>Under F.D.A review</td>
</tr>
</tbody>
</table>
### Table No.30: Inhibitors of energy intake (appetite Suppressants)

<table>
<thead>
<tr>
<th>No.</th>
<th>Drug</th>
<th>Target</th>
<th>Mechanism</th>
<th>Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Fenfluramine</td>
<td>Serotonergic Neurons</td>
<td>Inhibits serotonin re-uptake and serotonin release</td>
<td>Withdrawn</td>
</tr>
<tr>
<td>2</td>
<td>Phentermine</td>
<td>Noradrenergic Neurons</td>
<td>Inhibits norepinephrine re-uptake</td>
<td>FDA – approval (food &amp; drug Admin)</td>
</tr>
<tr>
<td>3</td>
<td>Fenfluramine &amp; Phentermine (Fen/Phen)</td>
<td>Serotonergic Neurons and Noradrenergic Neurons</td>
<td>Inhibits serotonin re-uptake &amp; stimulates serotonin release. Inhibs norepinephrine re-uptake</td>
<td>Combination of Individually approved drugs, (Fenfluramine now withdrawn)</td>
</tr>
<tr>
<td>4</td>
<td>Dexfenfluramine (Redux)</td>
<td>Serotonergic neurons</td>
<td>Inhibits Serotonin re-uptake and stimulates Serotonin release</td>
<td>FDA Approval</td>
</tr>
<tr>
<td>5</td>
<td>Sibutramine (Meridia)</td>
<td>Serotonergic neurons and Noradrenergic Neurons</td>
<td>Inhabit Serotonin &amp; non-epinephrine re-uptake</td>
<td>FDA approval</td>
</tr>
<tr>
<td>6</td>
<td>OB (Leptin)</td>
<td>OB receptor</td>
<td>Activates OB receptor in brain &amp; reduces food intake</td>
<td>In phase II clinical trials</td>
</tr>
</tbody>
</table>

There are no current drugs that enhance energy expenditure.

**Potential target for new anti-obesity Medicine:**

Perhaps the most significant new target is recently isolated hormone, OB (the product of the obesity gene OB, also known as Leptin) which has rapidly become appreciated as the critical signal in the regulation of the body fat and body weight.
Leptin is produced by fat cells, circulates in the blood and enters the brain where it functions to reduce food intake, reduce serum glucose and insulin levels, increase metabolic rate, ultimately leading to a reduction of fat mass and body weight.

Leptin mediates its effect through specific receptor, OB-R, which has been cloned and characterized.

Obesity is chronic disease and the possibility of long term treatment either continues or intermittent treatment throughout adult life, is a concept that is receiving more attention. In this context, the risk benefit and quality of life analysis of pharmacological treatment become increasing important.

6) Surgical treatment:

Because of the malignant nature of harbid obesity and the inability to achieve and maintain sufficient weight reduction, reduction by non-surgical means, surgery is justifies in this population.

Indications and Eligibility Criteria for Obesity surgery

- Refractoriness of the obesity
- Well informed patient
- Co-operative patient without disqualifying physicho-pathology
- Optional pre-operative Cardiff-pulmonary status.
- Minimum weight for height for persons aged 20 to 50 years (unless in the presence of a serious complication) weights given are midpoints for persons of large frame in the metropolitan life insurable tables.

- For Women – 150 cm – 103 Kg, 161 cm – 109 kg, 171 cm – 115 kg
- For men – 163 cm- 13 kg, 174 cm- 119 kg, 186 cm – 127 kg
- Presence at serious complications of obesity
- Commitment by surgeon, hospital and patient to life long follow up.

Twenty one different surgical procedure have been developed for the treatment of obesity (Keralm1983). Most surgeons have agreed that a criterion for considering
a patient for surgical treatment is 100% overweight or that patient has at least 45.5 kg to lose. The chief surgical procedure are as follows:

- Jejunoileal Bypass
- Gastric Bypass
- Jaw Wiring
- Truncal Vagotomy
- Gastric Plication
- Plastic Surgery
ESTIMATION OF LIPIDS

ESTIMATION OF CHOLESTEROL

Infinite cholesterol liquid of Accurex diagnostic Kit (Accurex Bio medical Pvt. Ltd.) was used for the estimation of cholesterol, which followed the enzymatic procedure described by Roescheau in 1974.

Principal:

Cholesterol esterase (CHE) hydrolysis cholesterol esters into free cholesterol and fatty acids. The free cholesterol is then oxidized by cholesterol oxidase (CO) to cholesterol -4 en-3 one with the simultaneous production of hydrogen peroxide. In presence of peroxide. In presence of peroxidase (POD), hydrogen peroxide oxidatively couples with 4-amino antipyrine and phenol to produce red quinoneimine dye which has absorbance maximum at 510nm (500-530). The intensity of the red colour is proportional to the amount cholesterol in the specimen.

\[
\text{Cholesterol esters} \xrightarrow{\text{CHE}} \text{Cholesterol + Fatty acids}
\]

\[
\text{Cholesterol + O}_2 \xrightarrow{} \text{H}_2\text{O}_2 + \text{Cholest} -4\text{en-3 one}
\]

\[
2\text{H}_2\text{O}_2 + 4 - \text{Aminoantipyrine + Phenol} \xrightarrow{\text{POD}} \text{Red quinoneimine dye + H}_2\text{O}
\]

Table No. 31: Reagents Components

<table>
<thead>
<tr>
<th>Components</th>
<th>Concentration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Buffer, Ph 6.8</td>
<td>50 m Mol/L</td>
</tr>
<tr>
<td>Cholesterol Oxidase</td>
<td>≥ 100 IU/L</td>
</tr>
<tr>
<td>Peroxide</td>
<td>≥ 150 IU/L</td>
</tr>
<tr>
<td>4-amino antipyrine</td>
<td>≥ 500 IU/L</td>
</tr>
<tr>
<td>Phenol</td>
<td>0.5 m Mol/L</td>
</tr>
<tr>
<td>Stabiliser/surfactants</td>
<td>10 m Mol/L</td>
</tr>
</tbody>
</table>
Specimen Collection

Blood should be collected in a clean dry container. Fasting blood is preferred for cholesterol assay.

Procedure:

<table>
<thead>
<tr>
<th>Reaction type</th>
<th>End point.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reaction time</td>
<td>10 min at 37°C / 20 min at R - 7</td>
</tr>
<tr>
<td>Wavelength</td>
<td>510 nm (500-530)</td>
</tr>
<tr>
<td>Zero setting</td>
<td>Reagent blank</td>
</tr>
<tr>
<td>Blank absorbance with limit</td>
<td>0.01 ml (10 ul)</td>
</tr>
<tr>
<td>Reagent volume</td>
<td>1.0 ml</td>
</tr>
<tr>
<td>Standard concentration</td>
<td>200 mg%</td>
</tr>
<tr>
<td>Linearity</td>
<td>1000 mg/dl</td>
</tr>
</tbody>
</table>

Pre-warm at room temperature, the required amount of reagent before use.

Table No. 32: Assay Procedure

<table>
<thead>
<tr>
<th>Reagent</th>
<th>Serum</th>
<th>Standard</th>
<th>Blank</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.01 ml</td>
<td>0.01 ml</td>
<td>--</td>
</tr>
<tr>
<td></td>
<td>1.0 ml</td>
<td>1 ml</td>
<td>1.0 ml</td>
</tr>
</tbody>
</table>

Incubation

Incubate the assay mixture for 10 minutes at 37°C or 20 minutes at room temperature (25°C–37°C). After incubation, the absorbance of assay mixture against blank at 510 was measured in RF 50 semi auto analyzer.

1. With Standard

\[
\text{Conc. (mg %)} = \frac{\text{Absorbance of sample}}{\text{Absorbance of standard}} \times 200
\]
2. With factor for wavelength range: 500 – 510 nm

Conc. (mg %): 543 X Absorbance of sample

ESTIMATION OF TRIGLYCERIDES:

GPO-PAP METHOD

ENZYMATIC COLORIMETRIC TEST WITH LIPID CLEARING FACTOR (LCF)

Method: The triglycerides are determined after enzymatic hydrolysis with lipase. Indicator is quinonemine formed from hydrogen peroxide, 4- aminoantipyrine and 4- chlorophenol under the catalytic influence of peroxidase.

REACTION PRINCIPLE

\[
\text{Triglycerides} \rightarrow \text{Glycerol + Fatty acids}
\]

\[
\text{Glycerol + ATP} \xrightarrow{\text{GK}} \text{Glycerol 3 – phosphate + ADP}
\]

\[
\text{Glycerol –3 – Phosphate + O}_2 \xrightarrow{\text{GPO}} \text{dihydroxyacetone phosphate + H}_2\text{O}_2
\]

\[
\text{H}_2\text{O}_2 + 4\text{- aminoantipyrine} \xrightarrow{\text{POD}} \text{quinoneimine + HCl + H}_2\text{O}
\]

+ 4- chlorophenol
Table No. 33: Contents, reagent composition in the test:

1. 10 X 15, 3 x 100 or 3 x 250 ml buffer solution

<table>
<thead>
<tr>
<th>Component</th>
<th>Concentration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pipes buffer pH 7.5</td>
<td>40 mmol/l</td>
</tr>
<tr>
<td>4-Cholerophenol</td>
<td>5 mmol/l</td>
</tr>
<tr>
<td>Magnesium ions</td>
<td>5 mmol/l</td>
</tr>
<tr>
<td>ATP</td>
<td>1 mmol/l</td>
</tr>
<tr>
<td>Lipases</td>
<td>≥ 150 mmol/l</td>
</tr>
<tr>
<td>Peroxidase</td>
<td>≥ 0.5 u/ml</td>
</tr>
<tr>
<td>Glycerol kinase</td>
<td>≥ 0.4 u/ml</td>
</tr>
<tr>
<td>Sodium azide</td>
<td>0.05 u/ml</td>
</tr>
</tbody>
</table>

2. 1 x 3, 3 x 1.7 or 3 x 4.1 ml Enzyme reagent

<table>
<thead>
<tr>
<th>Component</th>
<th>Concentration</th>
</tr>
</thead>
<tbody>
<tr>
<td>4-aminoantipyrine</td>
<td>0.4 mmol/l</td>
</tr>
<tr>
<td>Glycerol–3 phosphate oxidase</td>
<td>≥ 1.5 u/ml</td>
</tr>
<tr>
<td>Sodium azide</td>
<td>0.05 %</td>
</tr>
</tbody>
</table>

3. 3 ml Triglycerides standard → 200 mg/dl or 2.28 mmol/l

Reagent preparation:

Cat. No: 10 720

Pipette 250 ul of enzymes reagent from bottle 2 into one bottle 1 (buffer solution) and mix carefully.

Cat No: 10 721 and 10 722

Pour the contents of one bottle 2 (enzyme reagent) into one bottle 1 (buffer solution) and mix carefully.

Note: Prior to use, allow the working reagent to stand for atleast 15 min. at room temperature.

Cat. No. 10 163

The triglycerides standard is ready for use.
Reagent stability

The reagents are stable up to the date when stored at 2.8°C. Contamination must be avoided.

The working reagent is stable for 6 weeks at 2.8°C or for 3 days at 15-25°C protected from light.

Specimen:

Serum, heparinized plasma or EDTA plasma

Note: Lipemic specimens usually generate turbidity of the sample reagent mixture which leads to falsely elevated results. The Human Triglycerides GPO Liquicolor test avoids these falsely elevated results through its built-in Lipid-Clearing-Factor (LCF). The LCF clears up totally the turbidity caused by lipemic specimens.

Assay:

<table>
<thead>
<tr>
<th>Wavelength</th>
<th>500 nm, Hg 546 nm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Optical path</td>
<td>1 cm</td>
</tr>
<tr>
<td>Temperature</td>
<td>20-25°C or 37°C</td>
</tr>
<tr>
<td>Measurement</td>
<td>Against reagent blank (Rb).</td>
</tr>
</tbody>
</table>

Pipetting scheme

Only the HUMAN Triglycerides standard provided with the kits or separately available are used. Cat.No. 10 163

<table>
<thead>
<tr>
<th>Pipette into curvettes</th>
<th>Reagent blank</th>
<th>Calibrator/Sample</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water</td>
<td>10 ul</td>
<td>--</td>
</tr>
<tr>
<td>Calibrator/Sample</td>
<td>--</td>
<td>10 ul</td>
</tr>
<tr>
<td>R1</td>
<td>750 ul</td>
<td>750 ul</td>
</tr>
</tbody>
</table>

Mix gently and incubate exactly for 5 min at 37°C

| R2                     | 250 ul        | 250 ul            |

Mix gently, incubate at 37°C and read the absorbance A of calibrator and samples against RB after 5 min.
Table No. 34.

<table>
<thead>
<tr>
<th>Pipette into cuvettes</th>
<th>Rb</th>
<th>Sample or Standard</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample / Standard</td>
<td>----</td>
<td>10 ul</td>
</tr>
<tr>
<td>Working reagent</td>
<td>1000 ul</td>
<td>1000 ul</td>
</tr>
</tbody>
</table>

Mix and incubate for 10 min. at 20-25°C or for 5 min. at 37°C. Measure the absorbance of the sample (ΔA_sample) and the standard (ΔA_std) against the reagent blank within 60 min.

Calculation of the triglycerides concentration:

\[
C = 200 \times \frac{ΔA_{sample}}{ΔA_{std}} \text{ (mg/dl)} \quad \text{or} \quad C = 2.28 \times \frac{ΔA_{Sample}}{ΔA_{std}} \text{ (mmol%)}
\]

Estimation of HDL cholesterol:

Direct Homogenous Test for the Determination of HDL Cholesterol Enzymatic Colorimetric Test.

Method

The assay combines two specific steps: in the 1st step, chylomicrons, VLDL, and LDL cholesterol are specifically eliminated and destroyed by enzymatic reactions. In the 2nd step, remaining cholesterol from the HDL fraction is determined by well-established specific enzymatic reactions in the presence of specific surfactants for HDL. This combination makes the assay more specific for HDL than other methods.
Reaction Principle:

1st Step:

LDL, VLDL CHE + CHO $\rightarrow$ Cholestenon $+$ $\mathrm{H}_2\mathrm{O}_2$
And chylomicrons

Specific conditions

$2 \mathrm{H}_2\mathrm{O}_2$ $\xrightarrow{\text{catalase}}$ $2 \mathrm{H}_2\mathrm{O}$ $+$ $\mathrm{O}_2$

2nd Step:

HDL CHE + CHO $\rightarrow$ Cholestenon $+$ $\mathrm{H}_2\mathrm{O}_2$

Specific conditions

$2 \mathrm{H}_2\mathrm{O}_2$ + Chromogen $\xrightarrow{\text{Peroxidase}}$ quinone pigment

Contents, Reagent composition in the Test:

R1 1 x 60 ml Enzymes

Good's buffer pH 7.0 (200 C) 100 mmol/l
Cholesterol esterase 600 U/l
Cholesterol Oxidase 380 U/l
Catalase (in R1) 600 U/ml
N-(2 hydroxy -3 sulfopropyl)3,5
Dimethoxyaniline (HDAOS) 0.42 U/ml
R2  1 x 20 ml Substrate

Peroxidase

4-Aminoantipyrin (4-AA)

Good's buffer, pH 7.0 (200 C)  1000 U/l

Sodium azide  1.00 mmol/l

Detergents  100 mmol/l

0.05%

R3  1 x 4 ml Calibrator  > 1 %

Cholesterol

Reagent preparation and Stability:

The R1 and R2 are ready for use.

Stability: After opening, the reagents are stable upto 1 month when stored at 2...80 C. Avoid contamination. Do not freeze.

Calibrator:

Reconstitute the content of the vial with exactly 4 ml of distilled germ free water, close to vial and swirl carefully to dissolve all lyophilisate. Avoid foaming. Let stand for 30 minutes before use.

Stability

10 days at 2...80 C. If required, freshly prepared calibrator can be divided into portions and kept frozen at -200 C for maximum 30 days. Freeze and thaw only once, mix carefully after thawing.
Specimen:

Serum Plasma

: It is tested directly after sampling, otherwise store the wa at -200 C (up to several weeks, avoid repeated freezing and thawing.

Assay:

Wavelength: Hg 578 nm, 593 nm, (570 to 610 nm)

Optical path: 1 cm

Temperature: 370 C

Measurement: Against reagent blank, one blank per series is sufficient.

Procedure (manual procedure):

Warm the reagents and cuvette to 370 C. Temperature must be kept constant (+0.50 C) for the duration of the test.

Table No. 35

<table>
<thead>
<tr>
<th>Pipette into cuvettes</th>
<th>Reagent blank</th>
<th>Calibrator/Sample</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water</td>
<td>10 ul</td>
<td>--</td>
</tr>
<tr>
<td>Calibrator/Sample</td>
<td>--</td>
<td>10 ul</td>
</tr>
<tr>
<td>R1</td>
<td>750 ul</td>
<td>750 ul</td>
</tr>
</tbody>
</table>

Mix gently and incubate exactly for 5 min at 37\(^{0}\) C

| R2                   | 250 ul        | 250 ul            |

Mix gently, incubate at 37\(^{0}\) C and read the absorbance A of calibrator and samples against RB after 5 min.

The test can be run in a fixed time kinetic mode on analyzers.
Calculation

\[ C_{\text{sample}} = C_{\text{calibrator}} \times \frac{\Delta A_{\text{sample}}}{\Delta A_{\text{calibrator}}} \text{ mg/dl} \]

Conversion factor : \( C \) (mg/dl) x 0.02586 = \( C \) (mmol/l)

**IV Estimation of LDL Cholesterol**

LDL Cholesterol was calculated by using the formula

\[ \text{LDL Cholesterol} = \text{Total cholesterol} - \text{HDL Cholesterol} + \frac{\text{Triglyceride}}{5} \]

**V Estimation of VLDL Cholesterol**

VLDL Cholesterol was calculated by the formula

\[ \text{VLDL Cholesterol} = \frac{\text{Triglycerides}}{5} \]

**VI Calculation of HDL Ratio**

HDL ratio was calculated by the formula

\[ \text{HDL Ratio} = \frac{\text{HDL} \times 100}{\text{Total Cholesterol} - \text{HDL}} \]

**VII Calculation of Atherogenic Index**

Atherogenic Index was calculated by the formula

\[ \text{Atherogenic Index} = \frac{\text{LDL} + \text{VLDL}}{\text{HDL}} \]