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SANGYAHARAN SHODH

(A Peer-Reviewed International Journal)

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EDITORIAL

My heartiest congratulation to all the A.A.I.M. members for celebrating Foundation Day of the Department of Sangyaharan on 17th January 2015. At this occasion an Oratory competition on "Medical Education in India - Pt. Madan Mohan Malviya's Vision" was held. Members and Faculties expressed their views with historical reference in favor of integration of Pathies. Sangyaharan Day was also celebrated at the Department of Sangyaharan with a C.M.E. on 6th February 2015. Sangyaharan Day was also celebrated at different institutions all over the country. Department of Sangyaharan observed this great event in the form of C.M.E. on "Medical Institution in India - Pt.Madan Mohan Malviya's Vision". Three best papers were selected and were awarded.

This programme encouraged the present generation to follow Malviya's vision in present context and convince the present day global leaders to incorporate integration world wide.

JAI HIND JAI SANGYAHARAN JAY AYURVED

Devendra Nath Pande

Chief Editor, Professor & Founder Head, Deptt. of Sangyaharan,

I.M.S., B.H.U., Varanasi.

Why Mercury is a Unique Metal in Ayurveda

CB Jha,* B Bhattacharya,** KK Narang***

यस्य रोगस्य. यो योगस्तेनैव सह योजयेत् ।
रसेन्द्रो हरति व्याधीन् नरकुञ्जरवाजिनाम् ॥ २१८ ॥
जिस रोग की जो औषध है उसी रोग में उस औषध का प्रयोग करे ।
रसेन्द्र, मनुष्य, हाथी तथा घोड़ों के रोग को दूर करता है ॥ २१८ ॥

yasya rogasya yo yogaha tenaiva saha yojitaha |
rasendro hantitan rogam naira kunjara vajenam ||
~ Rasārnava, patala 11/218

Introduction: In critical conditions, where pain and palliation of symptoms is dire, mercurial preparations have an established reputaion for efficacy, that has been acquired from clinical experiential evidence over thousands of years by competent physicians. Because pain is a hallmark of disease, and considered the fifth vital sign, 35-40% of all total medicaments of classical Ayurveda involve mercury. Good Ayurvedic physicians know that complex pain can be tackled by using quality mercurial preparations, especially cardiac and neurological complications, due to mercury's ability to penetrate tissues. Despite the controversy regarding Mercury, rasa-aushadhies are considered important and vital life-saving drugs.

While 50% are purely herbal preparations, near half of herbo-minerals outside of rasa-mercurials, such as abhraka bhasma, tamra bhasma, and lauha bhasma also require marana (incineration/calcination)which is facilitated by processes of mercury. Due to processing variations, many medicaments used for palliation or pain therefore may likely have some nano or micro presence of mercury.

Mercury has a wide range of therapeutic efficacy when properly made into medicine. For palliative purposes, compounds such as makhardhwaj, purna chandrodaya, malla chandrodaya, malla sindoor, hemagarbha pottali, swarna parpati, and various kajjalis may be used for different organ systems of the body. Each also has a role in acute manifestations, as well as chronic conditions of many complex diseases.

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Hydrargyrum (symbol Hg), popularly called Mercury in chemical language and Parada in Ayurveda, is unique as a liquid metal, capable of mixing with most materials in solid state. Many metals easily get dissolved in this liquid to form Amalgams. The entire discipline of Rasa Shastra, a branch of Ayurveda, has developed around preparative procedures and applications of Mercury-based formulations in treatment of diseases since olden times.

The development of Ayurvedic medicine started from the use of plant materials when therapeutic utility of minerals and metals were recognized, they were integrated. Among the metals, Mercury was identified as a material which was able to consume other materials on the earth. It was found to be so potent that it couldbe used in all types of diseases, giving longevity to create a senility-free, strong and healthy body. To make mercury safe and effective it was subjected to many processes and procedures. These procedures involved addition of different materials, cyclically heating, boiling, mixing, or triturating. Ultimately mercury could be converted into therapeutically suitable compounds.

As science progressed in all dimensions, the properties of elements available on Earth have been extensively studied. The elements have been classified and documented in the form of a Periodic Table based on atomic number and atomic size, as well as periodic properties such as ionic size, ionization potential, electron affinity and electronegativity. Through the patterns seen in the periodic table, the chemical behavior of elements can be predicted and easily understood.

Of late, heavy metal toxicity has been, and continues to be,the subject of considerable interest and concern for the health of humanity. With the publication of the article "Heavy Metal Content of Ayurvedic Herbal Medicine Products" in JAMA[1], the use of metals like Parada (Mercury) and Naga (Lead) in Ayurveda and Siddha medicine has been criticized, without identifying the specific species or reactivity in the system. The phenomena of drug preparation, delivery, assimilation and rejection are extremely complex. Ancient Acharyas claim to have treated patients for several types of diseases using these heavy metals in the form of Bhasmas along with herbal mixtures.

In the present communication, the authors try to educate people with knowledge condensed in texts, providing probable chemical reactions and explaining the behavior of materials involving Mercury.

To begin, let us refer to a stepwise reaction process from an ancient text of rasa-shastra. In the Rasa Ratna Samucchaya of the 14th century, written by Rasa Vagbhatacharya, there is a discussion on medicines prepared from plants and minerals.
The sloka[2] states that plant products can be assimilated into lead, lead into tin, tin into copper, copper into silver, silver into gold, and gold into mercury.

काष्ठौषध्यो नागे, नागो वंगे अथ वंगमपि शुल्वे।
शुल्वं तारे, तारं कनके, कनकंच लीयते सूते ॥

काष्ठ औषधियाँ नाग में, नाग वंग में, वंग ताम्र में ताम्र रजत में, रजत स्वर्ण में तथा स्वर्ण पारद में लीन हो जाता है।

Our ancestral rishis and sages experimented with a variety of materials. Lead, with atomic number 82 and an electron configuration of ($6s^26p^2$), is a large atom, among the heaviest of elements that exist, in material form, for a substantial length of time and occurs in nature in ores or in native form. Lead has been found to react easily with herbs and herbal extracts.

In modern chemistry, we attribute this reactivity tothe presence of –OH and –COOH groups in herbal chemicalsand due to inert pairing of electrons(6s2), the two 6p2 electrons are easily lost, to give Pb2+ ion or Pb2+ salts with herbal chemicals.
Pb and Sn easily form alloys with each other. Similarly, Sn and Cu form alloys to give bronze. Cu – Ag form alloy to give brass. Ag – Au also form alloys, and Au – Hg form an amalgam.

In the modern Periodic Table of elements, the placement of these metals is immediately adjacent to each other, so their physico-chemical properties are helpful to form alloy formation, which very much depends on the size of atoms for mutual accommodation in each other's lattice.

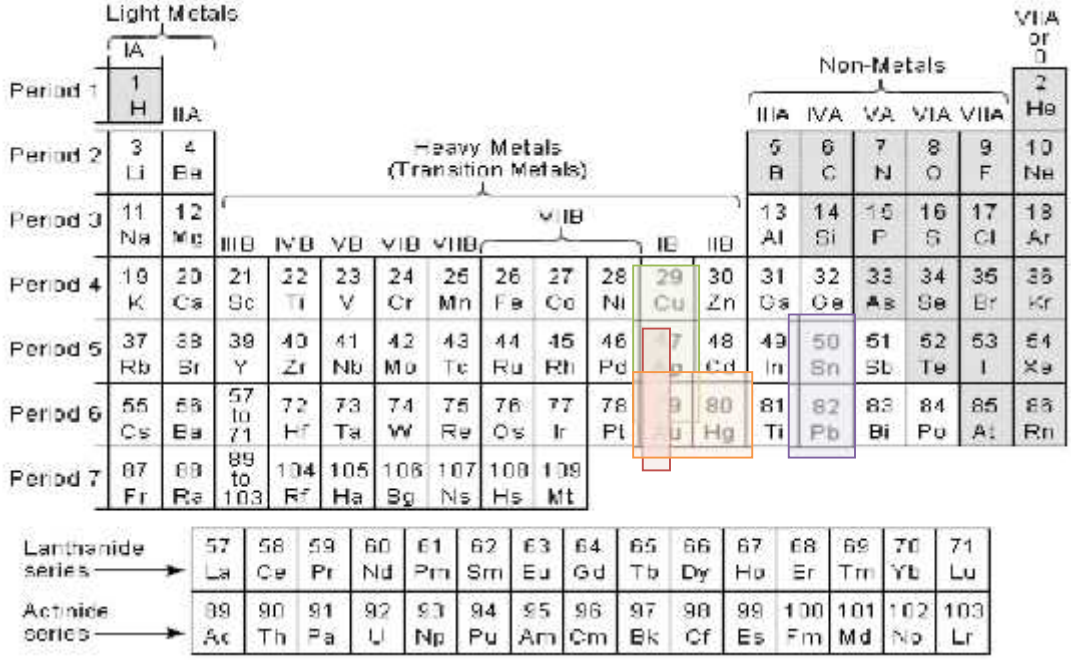
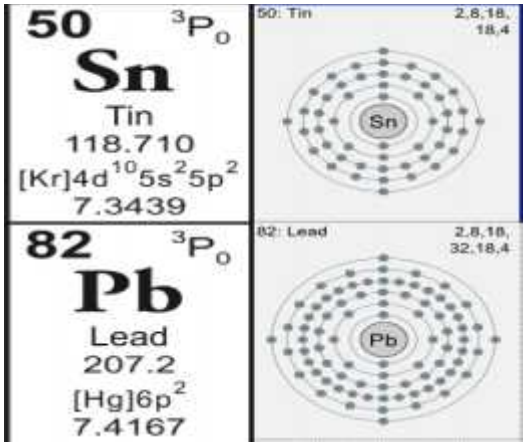
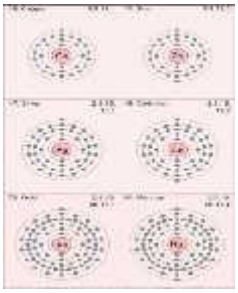


Figure 1. The Modern Periodic Table.
Source: http://cbsemocha.com/chemistry/ModernPeriodicTable.html



For example, tin (Sn), with an atomic radius of 1.45 Ångstroms (10-10 meters) is smaller than lead (Pb), with an atomic radius of 1.54 Ångstroms (10-10 meters). Sn atoms can easily fit into the structure of Pb.



In the same way, Ag is related to Au, and Cu is related to Ag, while Au and Hg are horizontally placed and are of similar atomic size. They just mix in each other by substitution as atom for atom.They maybe forming Hg - Au bond in amalgams. There are innumerable other alloys, alloy phases,and intermetallic compounds. Binary, ternary, quaternary and even multi-metal alloys also are now known.

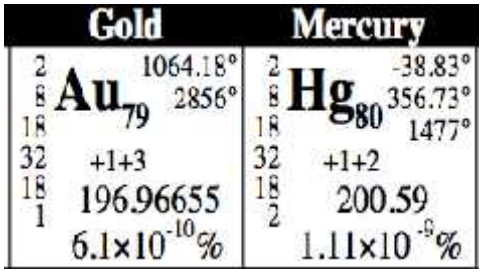
Another excerpt, given below and taken from the Rasarnava,[3] written by Bhairava Ananda Yogi in the 12th century CE, describes the relationship between mercury, gold and fire.

हेम पवाकयो: सख्यं सख्यः सूतक हेमयोह ।
सूत पावकयोर्वैर तयोर मित्रेन मित्रता ॥

अर्थात्- सोने और अग्नि की मित्रता है तथा सोने एवं पारे की भी आपस में मित्रता है । दूसरी ओर पारे एवं अग्नि में मित्रता नहीं है किन्तु सोने के माध्यम से पारे एवं अग्नि में मित्रता स्थापित हो सकती है ।

हेम पवाकयो: सख्यं सख्यः सूतक हेमयोह I
सूत पावकयोवर तयोरमित्रेन मित्रता II

अथात्- सोने और अग्नि का मित्रता है तथा सोने एवं पारे का भी आपस म मित्रता है I दूसरा ओर पारे एवं अग्नि म मित्रता नहा है किन्तु सोने के माध्यम ||



This observation actually relates to melting, mixing and vaporization of elements. Fire can melt gold but not vaporize it. Gold has a high melting point, 1064^{o}C, with an even higher boiling point, at 2856^{o}C. Gold and mercurymix readily to form amalgamswhile Mercury has a low boiling point of 356^{o}C and easily vaporizes with mild heating. Mercury is thermostabilized by amalgamating with gold.

Bhasma formations

Bhasmas are produced by incineration of metals, minerals and associated materials in air. This amounts to reaction with O_2 of air or decomposition of material in air.

$Fe + O \rightarrow Fe_2O_3$	Lauha Bhasma
$CuSO_4 \rightarrow CuO + SO_3$	Tamra Bhasma
$CaCO_3 \rightarrow CaO + CO_2$	Praval Bhasma

Precisely, metals and nonmetals in general, react to form compounds, known as *bhasma*.Mercury (Parada) reacts with Sulfur to give black Mercuric Sulfide Hg+S → HgS (Kajjali), similar to other compound formation. So Kajjali preparation resembles Bhasma Preparation, chemically.

Preparation of Kajjali

To prepare kajjali, Hg and S are taken in equal amounts by weight, in ratios of 1:1 or even up to ratios of 1:6. Even higher ratios can be used. The two are ground for several hours in a pestle-mortar system as the main instrument (yantra), to finally produce a rich black-coloured, slimy compound in the presence of a herbal extract.

Chemically 200 gm of Hg requires only 32 gm of S for HgS preparation but use of excess S in 1:1 (Hg : S) ratio, means use of sufficient excess of S to enable complete reaction of Hg with S. The ancient acharyas decidedly knew about the harmful effect of Hg, if it remained free or partly reacted in Kajjali.

Other metallic bhasmas could be produced by understanding different reactivities of the metal in relation to the nobility of Hg.

Cu+ Hg (Alloy) + S → CuS□ + Hg or Fe + Hg (Alloy) + S → FeS□ +Hg.

On heating Hg evaporates, leaving behind CuS or FeS.

Ag + Hg (Alloy) +S (on heating)→ Ag + Hg S ↑ (sublimes).

HgS sublimes, leaving behind Ag particles, called Rajat Bhasma.

Au + Hg (Alloy-Amalgam) + S (on heating) → Au + HgS ↑(sublimes).

Swarna Bhasma is the resultant residue.

The word Bhasma probably means any material produced after heating or incineration, which may be even in metallic state, as in case of Ag & Au Bhasma.
Black HgS sublimes to deposit as a red-coloured HgS species, called Rasa Sindura.
HgS (Black) → HgS (Red) is a sublimate.
Fe, Cu, Zn, Sn, Pb are baser metals and are more reactive than Hg towards Oxygen in air that even FeS, CuS, ZnS, SnS, PbS on heating in air give Fe_2O3, CuO, ZnO, SnO, PbO whileSO_2 evaporates.
Kajjaliis prepared by reacting Hg+S in different ratios by weight from 1:1 to 1:6, signifying sufficient excess of S than required for preparation of simple chemical HgS (200:32 in ratio of atomic weight). The slow grinding in Stone or Iron Pestle-Mortar system may contaminate Kajjali with small amount of impurities. Further use of vegetable juice to help smooth mixing and grinding adds organic chemical impurities to Kajjali.
Moreover, S element exists in a ring structure of $\mathbf{S_8}$ which partly breaks up or reacts with Hg to give several types of free radicals, generating heat during grinding, just as freshly ground wheat grains produce free radicals and the fresh powder (Atta) is hot. The probable species present in Kajjali may be HgS, Hg-SS , SS-Hg-SS, S-S-S-Hg-S-S-S along with free radicals, like $\overset{\bullet}{S}$-S-S$\overset{\bullet\bullet}{,}$S-S-S-$\overset{\bullet}{S}$ and many more,because S has tendency to form chains as in Poly-Sulfanes, H_2S_n and Poly Sulphides $(NH_4)_2S_n$.

In a recent study on Rasa Sindura,[4] the authors found it to contain high percentages of carbon and nitrogen, besides other elements. Whereas Rasa Sindura is believed to be only the sublimate of HgS, the analysis however clearly indicates the presence of organic matter, obtained from the decomposition of herbal extracts used for preparation of Kajjali. These organic molecules may contribute to the efficacy of Rasa Sindura, as medicinal content.
In the same manner yet another Mercury - Sulfur preparation, popularly called Parpati, needs close scrutiny of the chemical content and conditions under which it is prepared. Maybe the minor components contribute to the efficacy of the formulations. It has been observed that Kajjali is laxative while Parpati prepared from the same Kajjali by melting and pressing the melt between two banana or some other leaves to give flakes of Parpati, is not laxative. However, it is *grahi*,and reduces the frequency of defaecation (reduces the intestinal motility) and effective in Irritable Bowel Syndrome / Malabsorption Sprue Syndrome[5]. It appears that free radicals or easily decomposable Poly sulfides, as above, rejoin on heating to produce elemental Sulfur,$\mathbf{S_8}$which has different properties.

Mercury - toxicity and poisoning
Mercury in different oxidation states, Hg^0, Hg_2^{2+} , Hg^{2+}, yield inorganic or organo-metallic compounds that are considered toxic.The toxic effects include damage to the brain, kidneys and lungs. Several diseases have been associated with increasing level of mercury concentration in body fluids. The symptoms of mercury toxicity have been identified with effect on vision, hearing, speech, disturbed sensation or lack of coordination in activities. Visible symptoms of

mercury toxicity are associated with itching, burning, pain, skin discoloration, swelling and/or peeling of skin.

Hg^{2+} salts are usually more toxic than Hg_2^{2+} salts because of the different solubilities in water. Hg^{2+}salts are readily absorbed in gastrointestinal tract. $Hg(CN)_2$is highly poisonous because it releases HCN on reacting with acid in stomach.

Organo-mercury compounds like $(CH_3)_2Hg$ or $(C_2H_5)_2Hg$ are highly toxic because such compounds are lipo-soluble and are able to cross the blood-brain barrier, causing brain and liver damage.

Several mercury compounds have been investigated for lethal dose. The following data on LD_{50} has been reported for some of the mercury compounds.

The LD_{50} of different mercuric (Hg^{2+}) compounds, such as HgO, $HgCl_2$, $Hg(NO_3)_2$, $Hg(OCOCH_3)_2$ and mercurous (Hg_2^{2+}) compounds such as Hg_2Cl_2, $Hg_2(NO_3)_2$, $Hg_2(OCOCH_3)_2$ generally range between 10-100 mg/kg body weight for studies on rats. But at the same time LD_{50} of HgS is for more than 10,000 mg/kg; virtually it is indeterminate.

Mercury sulfide HgS is typically stable, insoluble in any other solvents and even in mineral acids. However, it dissolves only in *aqua regia* which is a mixture of three parts of concentrated HCl and one part of concentrated HNO_3, which gives highly reactive nascent chlorine in solution.

$$3HCl + HNO_3 \rightarrow \quad 2H_2O + NOCl + 2Cl^{\bullet}$$

The $Cl^{\bullet}$ reacts with HgS

$$HgS + 2Cl^{\bullet} \quad \rightarrow \quad HgCl_2 + S_8$$

The elemental sulfur, S_8 is separated from Hg.

Such a strong acid, as *aqua regia*, is not produced in the body system. Therefore, HgS remains undissociated or undegraded and unabsorbed in the system. That is why LD50 of Hg is indeterminate. Several studies have confirmed this. Recently HgS has been reported to be non-biodegradable in the larvaes of bees.[6] Kajjali, Rasa Sindura, Parpati mainly contain HgS. Therefore, they find extensive use in Ayurvedic formulations in significant amounts.

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Shigru As Antiinflammatory Drug In Ayurveda

***Dr. B.N. Maurya, **Dr. D.N. Pande**

Introduction: The pharmacological action and therapeutic actions of Shigru are well described in Ayurvedic literature. This is kapha-vata shamak.This paper is only focused on its Morphology, Properties, Chemical Composition, Action and Uses.

Key words: Therapeutic, pendulous, pterygospermin & Sunthi.

Botanical Name	-	*Moringa oleifera* Lam
Family	-	Moringaceae
Sanskrit	-	Sigruh, Sobhanjan, Aksheeb Teckshnagandha, Mochak
Hindi	-	Sahijan, Mungana
English	-	Horse-radish tree Drum stick tree
Malayalam	-	Murinna
Tamil	-	Murunkai
Telugu	-	Munaga, Mulaga
Bengali	-	Sajina

Habitat and Distribution: *Moringa oleifera* is found throughout India. It is also cultivated.

Morphology: Shigru is an unarmed middle sized graceful tree with corky gray bark and easily breakable branches, leaves usually tripinnate, rachis slender, thickened and articulated at the base, leaflets elliptic or obovate, rounded at the apex, nerves obscure, flowers white in large puberulous axillary panicles, fruits pods up to 45 cm long, pendulous, greenish, triangular, 9-ribbed, seeds trigonous, the angles winged.

Properties:

Rasa	-	Katu, Tikta
Guna	-	Laghu, Ruksha, Teekshna
Virya	-	Ushna
Vipak	-	Katu

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Chemical Composition : The root bark contains two alkaloids moringine and moringinic. In 1932 Chopra and De conducted a series of experiments with the two alkaloids. The root of Shigru also contains pterygospermin.

Action and Uses: The pharmacological action and therapeutic actions of Shigru is well described in Ayurvedic literature. This is kapha-vata shamak.
According to Charak, soup of Shigru fruits with marica, salt and yavaksara checks hiccough and asthma (Ch.Chi. 17/98).

According to Siddhabhesaj Manimala, pills made of rock salt, borax and sunthi with Shigru juice remove colic (Si. Bhe. 4/514).
On the other hand according to Vaidya Manorama decoction of sunthi and Shigru alleviates colic within three days. So does that of agastya bark added with rock salt and hingu (Vai. Ma. 8/21).

In Harit Samhita it is mentioned that decoction of Shigru root added with marica, yavaksara and honey removes colic caused by kapha (H. Sam. 3/7/48).

1. In Harit Samhita it is also mentioned that headache is removed by the snuff of Sobhanjana juice mixed with jaggery (H. Sam. 3/40/21).
2. Chakradatta has mentioned that juice of sobhanjana mixed with honey, oil and rocksalt removes earache (Ch. Dutt. 57/5).
3. Banga Sen has described that warm juice of Shigru root-bark eradicates ringworm (Ba. S. Krimi 22).

The action and uses of *Moringa oleifera* has been mentioned by modern researchers. In Ceylon the barks, leaves and roots are taken to promote digestion. The expressed juice of fresh roots, bark and leaves is poured into the nostrils in stupor and coma. A decoction of fresh roots and bark is given internally (Roberts). All parts of the plant are useless in the treatment snake-bite (Mhaskar and caius) or scorpion-sting (caius and Mhaskar) whether administered internally or applied externally.

The alkaloids from this plant closely resemble ephedrine in action. They are likely to be useful as a cardiac stimulant and in the treatment of such conditions as asthma (Chopra and De, 1930).

In 1932 Chopra and De conducted a series of experiments with the two alkaloids, moringine and moringinine, isolated from the root bark. Neither of these gives the chemical and physical tests of ephedrine. Moringine was found to be comparatively inert, moringinine showed physiological activity. The latter (1) acts on the sympathetic nerve endings as well as the cardiac and smooth muscles all over the body, it produces a rise of blood pressure, stimulation of heart and contraction of blood vessels, it also relaxes the bronchioles, inhibits the tone and movements of the intestines and contracts the uterus in guinea pigs and rabbits; (2) it produces a slight diuresis due to rise of blood pressure. (3) It is detoxicated by the liver; (4) it depresses the sympathetic motor fibers of the vessels in large doses only.

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Principles behind Success of Aganikarma Therapy in Different Joints Pain

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Abstract: Ayurveda deals with the changes of lifestyle .Ayurved- the science of life is time tested science which does not require experimental evidences. It's all principles are universally applicable to each individual to have a long healthy life. It is such a treaty which is enriched in medicaments and different management for number of diseases.Agni Karma is also a very good management for Pain.This paper includes the Principles of Agni Karma.

Key Word: environmental factors, low backache, Parasurgical procedures.

Introduction: At present the human society is leading with mechanical life, frequent changing of lifestyle, environmental factors, climate, etc. The critical busy schedule, restless, anxiety, stress & strain, running after comfortable life, comparing to higher group curses different psychosomatic disorders. The major somatic disorders involves, the constant work schedule in improper sitting posture, continuous & over exertion, prolonged traveling by different vehicles, less sports activities, exercises, etc. which in fact cause undue pressure on spinal cord, knee joints, shoulder joints, wrist joint, etc. and produce low backache, joint pain, while estimating the joint pain and low backache the incidence rate of this disease goes higher than 60%. If this joints pain sustain for a prolonged period with the affection of individual body then the disease tends to manifest its severity and chronicity. Such tedious, painful disease nowadays is enhanced its rate.

Ayurvedic literature states that every substance on this earth has medicinal properties, as the basic five elements are present in both living and non living things. Despite of emergence of newer medical facilities, till today no specific treatment is present for painful joints. "Agni Karma" therapy has been recommended in various musculoskeletal disorders by Aacharya Sushruta[1] as systemic medicine plays very less role in management of skeletal disorders and they are full of unwanted results.

Ayurvedic text book has mentioned various Para surgical procedures useful in the diseases of Vata and Kapha disorders where patients conditions are not life threatening. 'Agni karma' is one amongst these Parasurgical procedures- Anushastra Karma.

Acharya Sushruta in 2 A.D. before evolution of other medical aids indicated 'Agni karma' in various disorders of skin, muscles, vessels, ligaments joints and bones. He has also explained that the diseases treated with Agni karma modality don't reoccur[1]

Agnikarma* orTau-dam*** is a basically a traditional Himalayan therapy practiced by the rural Himalayan people for liver troubles, stomach troubles, backache, etc. According to Tewari (2002), this therapy was also practiced by the ancient people and is also mentioned in Ayurveda as *Agnikarma*. He mentions that in other parts of India this therapy is also used for stomach and liver troubles.

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Tau therapy is generally practiced by the older people of village and is compulsory for 6 month to 1-year old children. A 45-60 cm long iron rod is called the *tau*, which is sharply curved at one end and has one or two holes depending upon the diseases. In this therapy, *tau* device is placed on burning fire till it gets red-hot. The older people touch this red-hot *tau* on the affected skin for only a few seconds. And after branding it, they massage the affected area with the mustard or olive oil.

In the *dam* technique, fresh seeds of *Terminalia chebula* or *Anaphalis araneosa* are burned on fire and touched on the required part of the body for only few seconds. After this, like *tau*, the effective area is massaged with the mustard or olive oil.

PROBABLE MECHANISM WHICH PROVE THE ACTION OF AGNI KARMA THERAPY (Ayurvedic and Modern View): According the Sushruta Samhita, patients treated with Agni Karma procedure never suffers from the same disease again, i.e. it never recurs. Thus Agni Karma cures the disease completely. Hence Agni Karma is said to be superior to other therapeutic procedures like oral medicine, Kshar Karma or even surgery. In modern terminology, "Agni Karma" therapy can be termed as "therapeutic heat burns".

MECHANISM OF ACTION:

Theory 1

Agni Karma

⇩

Ushna guna(Hot properties)

⇩

Qualities against Vata & Kapha Dosha

⇩

Pacifies Vata & Reduces Kapha

⇩

Reduces Pain

- According to Ayurveda, Ushna treatment acts against the qualities of Vata and Kapha dosha and hence Agni Karma cures the Vataja and Kaphaja disorders.
- The cause of Pain anywhere in the body is Vata. Agni Karma being Ushna Chikitsa pacifies Vata. Thus the pain is relieved immediately after Agni Karma.
- Similarly Kaphaja disorders also get cured by the Ushna i.e. hot qualities of Agni Karma.

Theory 2

According to Ayurveda, every Dhatu (tissue) has its own Dhatvagni which is responsible for the nourishment, increase, and decrease of Dhatu. When this Dhatvagni becomes low (*manda*), diseases begin to manifest.
Thus in this condition, Agni Karma works by giving external heat, thereby increasing the Dhatvagni which helps to digest the aggravated dosha and hence cures the disease.

Agni Karma
⇩
Ushna guna(Hot properties)
⇩
Increase in Dhatvagni
⇩
Manda Dhatvagni gets rectified
⇩
Enhance nourishment & formation of newer tissues.

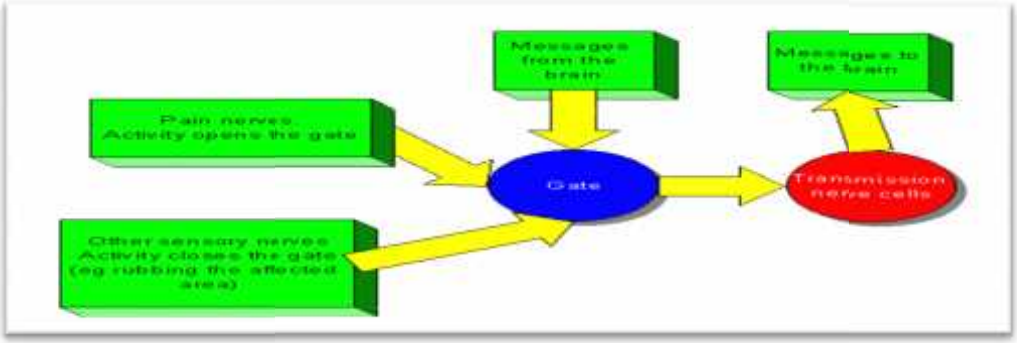
Also perform *Ama Pachana* & removal of accumulated toxins.

Theory 3: According to scientist Dr. VenHanff, at the place where heat burns, the local tissue metabolism is improved, thus various metabolic and rejuvenating changes take place at the site of heat burns, and thus it leads to increased demand of oxygen and nutrient of the tissues at the site of heat burn. It also excretes the unwanted metabolites and toxins.
After performing Agni Karma the superficial sensory nerves get stimulated which leads the dilatation of local blood vessels, resulting in increased blood circulation. Apart from this it also decreases the viscosity of blood and thus leads to better transport of metabolites.
Due to increased local metabolism, the waste products (metabolites) which are produced gets excreted, which normalizes the blood circulation thus resulting in reduction in intensity of pain.

Theory 4: (Gate Control Theory of Pain)
Melzack and Wall (1965) carefully discussed the shortcomings of the Specificity and Pattern Theories—the two dominant theories of the era—and attempted to bridge the gap between these theories with a framework based on the aspects of each theory that had been corroborated by physiological data. Specifically, Melzack and Wall accepted that there are nociceptors (pain fibers) and touch fibers and proposed that that these fibers synapse in two different regions within the dorsal horn of the spinal cord: cells in the substantia gelatinosa and the "transmission" cells. The model proposed that signals produced in primary afferents from stimulation of the skin were transmitted to three regions within the spinal cord: *1*) the substantia gelatinosa, *2*) the dorsal column, and *3*) a group of cells that they called transmission cells. They proposed that the

gate in the spinal cord is the substantia gelatinosa in the dorsal horn, which modulates the transmission of sensory information from the primary afferent neurons to transmission cells in the spinal cord. This gating mechanism is controlled by the activity in the large and small fibers. Large-fiber activity inhibits (or closes) the gate, whereas small-fiber activity facilitates (or opens) the gate. Activity from descending fibers that originate in supraspinal regions and project to the dorsal horn could also modulate this gate. When nociceptive information reaches a threshold that exceeds the inhibition elicited, it "opens the gate" and activates pathways that lead to the experience of pain and its related behaviors. Therefore, the Gate Control Theory of Pain provided a neural basis for the findings that supported and in fact helped to reconcile the apparent differences between the Pattern and Specificity Theories of Pain.

Heat | Stimulation of Lateral Spinothalamic Tract (SST) stimulation of descending pain inhibitory fibres (DPI) | Release of endogenous opioid peptide which bind with opioid receptors at substantia gelatinosa rolandi | Inhibition of release of P-substance (Pre-synaptic inhibition) | Blockade of transmission of pain sensation. Proposed by Melzack and Wall in the 1960's



Gate opened or closed by 3 factors

1. Activity in the pain fibres - opens the gate
2. Activity in other sensory nerves - closes the gate
3. Messages from the brain - concentrating on the pain or trying not to think about it

Theory 5: The muscle fibers contract & relax more quickly although the strength of the contraction is low. It does not affect relaxation of the antagonist muscles, but permits a free action of the prime movers. Provided that the heating is not excessive, it appears to reduce the excitability (quick response to stimuli) of nerves. Muscles relax most readily when tissues are warm, and the relief of pain also is facilitated. Heat helps muscles achieve relaxation & relief from muscle spasm. The relief of pain makes it possible to walk or actively mobilize extremities efficiently.

Theory 6: Pain receptors of skin and motor end plate get stimulated at 45 ⌈C. Pathway for pain and thermal signals run parallel and end up at same area, but only stronger one can be felt. Therefore complete exclusion of pain impulse by heat occurs.

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Mootrakrichchhra Chikitsa by Uttar Basti in Female patients w.s.r. to Ch.U.T.I.- A Review

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ABSTRACT: Mootrarogas are prevalent since Vedic period and our Ancient Acharyas had detail knowledge about their management and pathogenesis[12345678]. All Acharyas had already been discussed a lot about Mootrakrichchhra and its management. Acharya Sushruta have mentioned Uttarbasti as one of the important treatment for Moootrakrichchhra in Uttartantra 59th chapter. According to Ayurvedic texts physiological action of urination and defaecation is under the control of apan vayu, seat of which is pakwashaya .If this vayu gets vitiated it causes various genitourinary and anorectal diseases of which mootrakrichchhra is one of the important disease (S.Ni.1/19). Uttarbasti is one of the important treatment for moootrakrichchhra[6]. In Modern medicine we can correlate symptoms of UTI (Urinary Tract Infections)with Mootrakrichchhra which is of vatika origin.(C.Su.20/15, C.Si.1/32,33,34, A.H.Su.1/25). So on the basis of presenting symptomatology UTI have close resemblance with mootrakrichchhra. So, here my aim is to give substantial proof with several references for the study of Uttarbasti Chikitsa in Mootrakrichchhra.

Keywords: Uti (Urinary tract infection), Uttarbasti, Mootrakrichchhra

MOOTRAKRICHCHHRA:

ETIOLOGY *(NIDANA):*

व्यायामतीक्ष्णौषध रूक्षमद्यप्रस¯ नृत्युद्रुत पृष्टयानात्।
आनुप मत्स्यद्यशनादजीर्णात्स्यु मृत्रकृच्छ्राणि नृणामिहाष्टौ।। (च0सि0 26/27)

DIETARY FACTORS:

- *Tikshna Aushada Sevana* (Taking strong medicine)
- *Ruksha Anna* (Dry meal)
- *Atimadyapana* (Excessive intake of alcohol)
- *Anupa Mansa Sevana* (Meat of marshy place animals)
- *Atimastya Sevana* (Excess fish intake)
- *Adhyashana* (Over indulgence of food)

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PHYSICAL FACTOR:

- *Ativyayama* (Excess Exercise)
- *Ati Maithuna* (Over indulgence of coitus)
- *Ati Nritya Karma* (Over dancing)
- *Prishtayana* (Riding vehides, animals etc.)

;SLrSjsoizfo';sua nks"kka dqoZfUrA ¼v0la0fu0 9@3½

Vitiation of *Vata Dosha* due to *Nidana Sevana* leads to *Mutrakrrichchra.*

ETIO-PATHOGENESIS *(SAMPRAPTI):* Vitiation of *Mala (*Doshas) of the body occurs due to *Nidana Sevana*. This vitiated *Dosha* single or in combination vitiates *Basti*, there by functional derangement of *Mutramarga* occurs leading to difficulty in micturition.

izFk³eyk LoS dqfirk funkuS% loSRFkok dksieqisR;cLrkSA
ew=L;ekxZ ifjih;fUr ;nkUrnk ew=;rhg d`PNªkrAA
¼ek0fu0 30@1½

Due to *Nidana Sevana, Doshas* gets vitiated and situated in *Basti* and thereafter they produce diseases like *Mutraghata, Prameha* etc which are broadly termed as *Mutravikara*.

Table :Classification of Mootrakrichchhra:

सु०सं०	च०सं०	अ०सं०	अ०ह्द०	योर०	भा०प्र०	भै०र०	मा०नि
Vataja	*Vataja*	*Vataja*	*Vataja*	*Vataja*	*Vataja*	*Vataja*	*Vataja*
Pittaja	*Pittaja*	*Pittaja*	*Pittaja*	*Pittaja*	*Pittaja*	*Pittaja*	*Pittaja*
Kaphaja	*Kaphaja*	*Kaphaja*	*Kaphaja*	*Kaphaja*	*Kaphaja*	*Kaphaja*	*Kaphaja*
Sanipataja	*Sanipataja*	*Sanipataja*	*Sanipataja*	*Sanipataja*	*Sanipataja*	*Sanipataja*	*Sanipataja*
Ashmarija	*Ashmarija*	*Ashmarija*	*Ashmarija*	*Ashmarija*	*Ashmarija*	*Ashmarija*	*Ashmarija*
Shakrita	-	-	-	*Purishaja*	*Shakrita*	*Purishaja*	*Purishaja*
	Raktaja	-	-	-	-	*Shonitaja*	-
	Shukraja	*Shukraja*	*Shukraja*	*Shukraja*	*Shukraja*	*Shukraja*	*Shukraja*
Sharkara	*Sharkara*	*Sharkara*	*Sharkara*	-	*Sharkara*	-	*Sharkara*
Abhighataja	-	-	-	*Shalyaja*	*Shalyaja*	*Abhighata*	*Shalyaja*

CLINICAL FEATURES ACCORDING TO TYPES:

1. VATAJA MOOTRAKRICHCHHRA:

(i) अल्पमल्पं समुत्पीडय शुष्कमेहन बस्तिभिः।

फलाद्भिरिव कृच्छ्रेण वाताघातेन मेहति।। ¼lq0m0 & 59@4½

- Less quantity of urine
- Pain in scrotum, penis & supra pubic region
- Dysuria
- All type of *Vata Vedena*

(ii) rhoz:tks o³{k.kcfLres<ªs LoYia eqgqeq=k;rhg okrkr~A ¼p0fl0 26@29½

- Severe pain
- Less quantity of urine
- Frequency

All *Ayurvedic* scholars mentioned severe pain in suprapubic region as predominant symptom of *Vataja* Mootrakrichchhra and increased frequency of urine. As the severe pain is predominant symptom in *Vataj*a Mootrakrichchhra the *Vataja* Mootrakrichchhra may be corealated with acute condition of urinary tract like renal colic, prostatic abscess, acute urinary tract infections.

PITTAJA MOOTRAKRICHCHHRA:

(i) gfjnzkeq".ka jDra ok eq"desgu cfLrfHk%A vfXuuk náekukHkS% fiRrk?kkrsu esgfrAA ¼lq0m0 59@5½

- Yellowish urine
- Passage of hot urine
- Blood in urine (hematuria)
- Severe burning sensation in scrotum, penis and supra pubic region.

(ii) ihra ljDra lq:ta lnkga d`PNªk eqgwew=;rhg fiÙkkrA ¼p0fl0 26@29½

- Yellowish urine
- Blood in urine
- Burning micturition
- Frequency

Considering all above symptoms of *Pittaja* Mootrakrichchhra it can be said that burning micturition, haematuria are predominant symptoms. *Pittaja* Mootrakrichchhra may be considered as severe degree of cystitis, urethritis, acute prostatitis.

KAPHAJA MOOTRAKRICHCHHRA

(i) fLuX/k 'kqDyaeq".ka p eq"desgu cfLrfHk%A
lag`"Vjksek xq:fHk% 'ys"ek/kkrsu esgfrAA
¼lq0m0 39@6½

- Oil like urine
- Whitish urine
- Heaviness in scrotum, penis and suprapubic regions
- Horripilation

(ii) **cLrs lfy³xL; xq:Ro'ksQkS ew=a lfiPNa dQew=d`PNªsA**
¼p0fl0 26@30½

- Heaviness in bladder and penile region
- Swelling in bladder and penile region
- Unctuous micturition

In *Kaphaja* Mootrakrichchhra pain and burning micturition or haematuria are not predominant symptom but they are present in some terms. Main symptoms according to all the *Ayurvedic* scholars are sticky urine or sticky discharge and mild discomfort. In chronic prostatitis or chronic pelvic pain syndrome one of the diagnostic symptom is sticky discharge and mild discomfort. So *Kaphaja* Mootrakrichchhra may be correlated with chronic prostatitis.

TRIDOSHAJA MOOTRAKRICHCHHRA:

(i) nkg 'khrnzqtkfo"Vks ukuko.kZ eqgqeqgq%A
ukekuqLrq d`PNsª.k lfUuikrsu esgkfrA
¼lq0m0 59@7½

- Burning micturition
- Cold feeling
- Pain during micturition
- Mixed coloured urine
- Frequency
- Dificulty in micturition

(ii) lokZf.k:ikf.k rq lfUuikrkRHkofUr rr~ d`PNªrr fg d`PNªeA ¼p0fl0 26@30½

Mixed symptoms of all three *Doshas* with severe dysuria are present.
In *Tridoshaj* variety of Mootrakrichchhra all varieties of manifestations like various kind of pain, burning sensation, different coloured urine are present. This condition can be compared with urinary tract infection i.e. cystitis, urethritis, prostatitis.

SHALYAJA MOOTRAKRICHCHHRA:

(i) ew=okfg"kq 'kY;su {ksrs"ofHkgrs"kq pA
lzksr% lq ew=k?kkrLrq tk;rs Hk`'kosnu%A
okr cLrLrq rqY;kfu fyM+xkfu y{k;srA
¼lq0m0 59@7½

According to *Sushruta*, injury to *Mutravaha Srotasa* produces symptoms like *Mutraghata*. Where as *Madhava Nidana* and *Bhava Prakash* says that, injury to *Mutravaha Srotas* produces symptoms similar to *Vataja* Mootrakrichchhra .Genitourinary trauma can be correlated with *Shalyaja* Mootrakrichchhra**.**

PURISHAJA MOOTRAKRICHCHHRA:

(i) 'kd`rLrq izrh/kkr}k;qfoZxq.krka xr%A
vk/ekua okr'kwya p ew=la³x djksfrp A
¼lq0m0 59@9½

Due to *Purishaja Vega Dharana* the *Vayu* gets vitiated, leading to distension, pain, retention of urine and dysuria. This condition is called *Purishaja* Mootrakrichchhra
Same symptoms are also described by *Madhava Nidana* and *Bhava Prakash*. In *Purishaja* Mootrakrichchhra main feature is retention of urine and pain. This condition can be correlated with acute retention of urine due to various factors like prostatic abscess, vesicle calculi obstructing outflow etc.

ASHMARIJA MOOTRAKRICHCHHRA:

(i) v'ejh gsrq rRiwoZ eq=d`PNªeqnkgjsrA
¼lq0m0 59@10½

Difficulty in micturition due to *Ashmari* is called *Ashmarija* Mootrakrichchhra

(ii) ew=L; psUekxZ :)k ew=a rL; djksfr cLrkS%A
llsouh esgucLrh'kwya fo'kh.kZ/kkje~ djksfr ew=e~A
e`nukfr es<ª l rq osnukrksZ eqgq% 'kd`UeqP;fr esgus pA ¼p0fl0 26@32½

- Difficulty in voiding.
- Pain in suprapubic region
- Pain in raphe, penis, bladder region
- Passage of urine in multiple streams

- Presses penis during micturition
- Increased frequency of micturition
- Increased frequency of defecation

The classical symptoms of *Ashmarija* Mootrakrichchhra i.e. suprapubic pain, bifurcated stream, frequency closely resembles ureteric colic. All scholars described that pain is colicky in nature.

SHARKARAJA MOOTRAKRICHCHHRA:

(i) g`RihMk osiFkq% 'kwya dq{kkS ofUg lnqcZy%A
vkfHkHkZofr ewPNkZ p ew=k?kkrÜp nk:.kk%AA
ew=osxfujLrkHkqrkLÙkq 'kkE;fr osnukA
;konU;k] iquusZfr xqfMdka lzksr lkseq[ke~AA
'kdZjk lkHkCLlSzy Hkwek/kku y{k.keAA
¼lq0m0 59@12½

- Pain in cardiac region
- Tremors
- Pain in abdomen
- Reduced digestive power
- Fainting
- Anuria
- If *Sharkara* gets flushed out then subsidence of symptoms occurs.
- During passing of *Sharkara* severe pain is produced.

(ii) {kksHkkr~ {krs eq=;rhg lkl`d rL;k lq[ka egfr p O;ik;kr~A
,"kk·'ejh ek:rkfHkUuewfrZ% LoPNdZjkew=iFkkr {kjUrhAA ¼p0fl0 26@34½

- When *Ashmari* breaks and expels out through urethra, due to influence of *Vata*, it is called *Sharkara.*

Symptoms mentioned under *Sharakarajanya* Mootrakrichchhra are fainting, tremors, anuria, and severe pain in abdomen. This type of pain occurs when stone travels downwards in urinary tract.

SHUKRAJA MOOTRAKRICHCHHRA:

(i) 'kqØeyk'pSo i`Fkd i`FkXok ew=k'k;LFkk% izfrok¸ ;fUrA
rnO;kg~ra esgucfLr 'kwya ew=a l'kqØa dq:rs foc)eAA
LrC/kÜp 'kwuks Hk`'kosnuÜp rqn~;sr cfLro`Z"k.kkS p rL;AA ¼p0fp026@35&36½

Obstruction of *Shukra* due to *Doshas* produces following symptoms

- Pain in bladder and penile region
- Less quantity of urine
- Semen mixed urine
- Stiffness and swelling
- Severe pain

- Intense pain in bladder and scrotum

RAKTAJA MOOTRAKRICHCHHRA:

{krkfHk?kkrku~ {krta {k;kn ok izdksfira cfLrxra foc)e~A
rhozkfrZeq=s.k lgk'ejhRoek;fr rfLeUufrlafprs pAA
vk/ekruka fonfUr xkSjoa p cLrsya?kqRoa p fofuL`rs·fLerAA
¼p0fp0 26@36½

Injury to urinary system produces following symptoms.

- Severe pain
- Colicky pain
- Distension
- Heaviness in bladder regions
- Feels light if content of the bladder is evacuated.

Raktaja Mootrakrichchhra is described only by *Charaka*. The symptoms of Shalyaja Mootrakrichchhra and Raktaja Mootrakrichchhra are same. These symptoms are present in urinary system trauma.

In treatment of Mootrakrichchhra

Su.U.59/17-19 Acharya Sushruta has explained Anuvasana vasti, Uttarbasti as treatment of Mootrakrichchhra.

Su.U.59/21 Acharya Sushruta has explained Uttarbasti as treatment of Mootrakrichchhra.

Su.U.59/22 Acharya Sushruta has explained Anuvasana vasti, Niruha basti and Uttarbasti as treatment of Mootrakrichchhra.

C.Chi.26/45 Acharya Charaka has explained Uttar basti by sneha in treatment of mootrakrichchhra.

So from above references it is clear that Uttarbasti *karma* is effective and important treatment of *mootrakrichchhra.* It can be inferred from above description that *Basti*, Uttarbasti chikitsa proves to be effective and first line of treatment of *Mutrarogas*. In the view of above facts attempts are being made to discuss Uttar *bastikama*. Hereby *Basti* therapy proves its applicability and rationality in *Mootrakrichchhra.*

INTRODUCTION: Urinary tract is comprised of kidney, ureter, bladder, and urethra. Urinary tract infection is an infection caused by pathogenic organisms in any of the structures that comprise the urinary tract. This is the broad definition of urinary tract infection. Many authors prefer to use more specific term that localize the urinary tract infection to the major structural segment involved such as urethra (urethritis), urinary bladder (cystitis), ureter infection, kidneys (pyelonephritis).

UTI is common in women than men, leading to approximately 8.3 million doctor visits per year and necessitate or complicate over 1 million hospital admissions in the united states annually (Patton et al , 1991 ; Haley et al , 1985). Survey screening for bacteriuria have shown that about 1% of school girls aged 5 to 14 years (Kunin et al, 1962) have bacteriuria and that this figure increases to about 4 % by young adulthood and then by an additional 1% to 2% per decade of age. The prevalence in young women is 30 times more than in men. Women tend to get more often because of their shorter urethra and closer to the anus than in men, because of this,women are more likely to get an infection after sexual activity or when using a birth diaphragm for birth

control. Menopause also increases the risk of uti in females. Because of the absence of prostatic bactericidal secretions also it is common in females. The 1st stage in the development of UTI is colonization of the periurethral zone with pathogenic organisms. Urine is an excellent culture medium for bacteria; in addition the urothelium of susceptible persons may have more receptors to which virulent strains of E.coli become adherent. In women the ascent of organisms into the bladder is easier. Instrumentation of the bladder may also introduce organisms.

UTIs can cause problems that range from dysuria to organ damage and even death. It is a worldwide problem. Though with the introduction of antibiotics its management has greatly improved, however several problems still remain. The antibiotics used generally have limitations because of the fact that the infective organisms develop resistance to them and toxic side effects are also common.

Problems that arise with the use of antimicrobial agents are[10]:

1. Toxicity Local – eg gastric irritationSystemic- almost all antimicrobial agents produce organ toxicity. Eg aminoglycosides causes 8th cranial nerve and kidney toxicity.
2. Hypersensitivity reactions Eg. cephalosporin
3. Acquired Drug resistance It is the development of resistance by an organism (which was sensitive before) due to the use of an antimictobial agent over a period of time. This can happen with any microbe and is a major clinical problem.
4. Superinfection
5. Nutritional deficiencies
6. Masking of an infection etc.

Uttara basti: Uttara basti is a type of basti upakrama, a mode of administration of drug. Uttara basti has been well highlighted in the classics. Uttara basti is….

- The administration of drugs through uttara marga.
- The administration of drugs through the route above guda marga.
- Administration of drug to utkrishta avayava.
- The procedure which imparts superior qualities.
- The administration of drug which serves the purpose of both Anuvasana and Nirooha.

Thus uttara basti may be defined as a route of administration of drugs throughVesicular / urethral route in males and vesicular / urethral or genital route in females.

Nirukti:

उत्तरमार्गेण दियमान्त्या किवां श्रेष्ठगुणतया।।
सनिरूहादुत्तरेण वा मार्गेण दीयत इत्युत्तरवस्तिः।। (C. Si. 9/50 Chakrapani teeka)

i.e., that which is administered through the *uttaramarga* and has *shreshta guna* is known as *Uttarabasti*. *Uttaramarga* means the *mootra* and *shukramarga* i.e., penis in male and the *mootra marga* and *yoni marga* i.e., urethral meatus and the vaginal orifice in the female.

Charaka has given the definition of Uttarbasti as a means by which fluid or liquid dravya is made to pass through medhra (penis) or yoni (vagina) or through apathya path into the garbhashaya. As it is given through uttarmarga and it gives shreshtha guna (best effect) it is called Uttarbasti.

INDICATIONS OF UTTAR BASTI:

शुक्रं दुष्टं शोणितं चा ¯नानां पुष्पोद्रेकं तस्य नाषं च कष्टम्।।
मूत्राघातान्मूत्रदोषान् प्रवृद्धान् योनि व्याधिं संस्थितिं चापरायाः।।125।।
शुक्रोत्सेकं शर्करामष्मरीं च शूलं बस्तौ वक्षंणे मेहने च।।
घोरानन्यान् बस्तिजांÚाापि रोगान् हित्वा मेहानुत्तरो हन्ति बस्तिः।।126।। (Su.Chi.37/125-126)

Uttara basti is indicated in some of the following conditions.

1.	Shonita dusti	2.	Yoni Vyapath	3.	Pushpodreka
4.	Pushpanasha	5.	Mutravaha Sroto Vikara	6.	Apara dushti
7.	Yoni vibhramsha, Yoni Shula			8.	Asrigdara etc.
9.	Garbhashaya Vikaras.				

CLASSIFICATION OF UTTARA BASTI:

- On the basis of form of drug to be administered Uttara basti may be classified as snaihika Uttara basti, niruhika Uttara basti. (**A. Su. 27/6)**
  1. **Snaihika uttara basti** – The uttara basti procedure where only sneha dravyas are used is called as snaihika Uttara basti. There is no mentioning of addition of any avapa dravyas in it.
  2. **Niruhika uttara basti** – The uttara basti procedure where only kashaya dravyas are used. There is no mentioning of addition of madhu, sneha, kalka etc. which are being added commonly in Niruha basti.
- On the basis of route of administration it is classified as
  1. **Mutrashayagata uttara basti** – The administration of drugs through urethral route.
  2. **Yonigata uttara basti** - The administration of drugs through vaginal route.
  3. **Garbhashayagata uttara basti** - The administration of drugs through uterine route.

DRUGS: Kashaya or sneha is to be used according to the need**.** This should be prepared from drugs which are mild in action.

DOSE: Snaihika Uttara basti **matra:** Sneha in Uttara basti to be administered in the dose **of 1/2 pala, or 1 prasruta** (the quantity that can be held in the palm of the individual) or **1pala.** For shodhana purpose the dose is 2 prasruta. The dose may be decided according to the individual and condition. (S**. Chi. 37/116)**

Modern Aspect of Uttarbasti: Intra-vesical therapy is the direct reference of Uttarbasti in modern medical science. Instillation of medicines into the bladder by transurethral catheterization is known as Intravesical therapy.

Conclusion:

In treatment of Mootrakrichchhra-

S.U.59/17-19 Acharya Sushruta has explained Anuvasana vasti, Uttarbasti as treatment of Mootrakrichchhra

S.U.59/21 Acharya Sushruta has explained Uttarbasti as treatment of Mootrakrichchhra

S.U.59/22 Acharya Sushruta has explained Anuvasana vasti, Niruha basti and Uttarbasti as treatment of Mootrakrichchhra

C.Chi.26/45 Acharya Charaka has explained Uttar basti by sneha in treatment of mootrakrichchhra.

It can be inferred from above description that *Basti*, Uttarbasti chikitsa proves to be effective and first line of treatment of Mutrarogas. In the view of above facts attempts are being made to discuss Uttarbastikarma. Hereby Uttarbasti chikitsa proves its applicability and rationality in Mootrakrichchhra.

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Anaesthetic consideration in Preeclampsia

***Vimal Kumar **AK Srivastava *** DK Singh**

ABSTRACT: Hypertensive disorders of pregnancy are the most common medical disorders of pregnancy and are associated with increased maternal and perinatal risks. Preeclampsia can be a major contributor to maternal morbidity and mortality as an immediate consequence of the progression to eclampsia. The aim of this review is to summarise the key management issues for anaesthetists in the light of the current literature. Substandard care is often present and many deaths are preventable.

KEY WORDS: Preeclampsia (Pre-eclamptic toxaemia), Hypertension, Proteinuria, Odema, PIH, HELLP.

INTRODUCTION: Preeclampsia (Pre-eclamptic toxaemia) represents a multisystem disease with many other manifestations. Thus there has been a move in definition away from the classic triad of PET above, toward the definition of PIH with or without features. PIH occurs in 10-12% of pregnancies whilst PET itself has an incidence of 2-3%.Severe PIH causes or contributes to 20-40% of maternal deaths and 20% of perinatal deaths. Maternal deaths are usually due to stroke, pulmonary edema, and hepatic necrosis or rupture.

DEFINATION: Preeclampsia definend as the following occurring after the 20^{th} week of pregnancy:

- **Hypertension:** systolic, mean or diastolic BP >140, 105 or 90 mmHg respectively, or an increase in systolic or diastolic BP greater than 30 and 15 mmHg respectively.
- **Odema :**(Hand and face).
- **Proteinuria: More** than 0.3 gm/l.

RISK FACTORS –Known risk factors for preeclampsia include:

Nulliparity (never given birth).
Diabetes mellitus.
Kidney disease.
Chronic hypertension.
Prior history of preeclampsia.
Family history of preeclampsia.
Advanced maternal age (>35 years).
Obesity.

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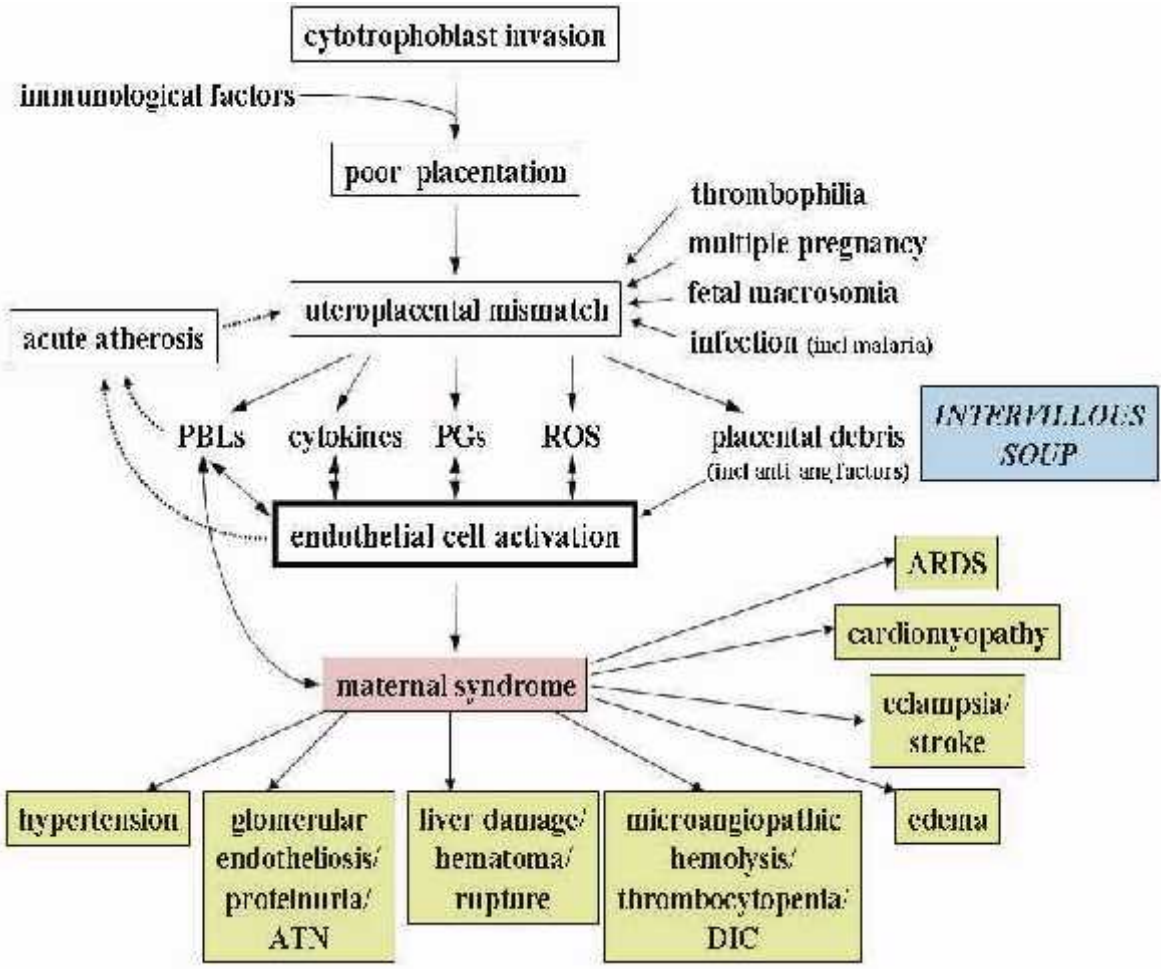
- Antiphospholipid antibody syndrome.
- Multiple gestation.
- Having donated a kidney.
- Having sub-clinical hypothyroidism or thyroid antibodies.

PATHOGENESIS :



MATERNAL FEATURES:

- **Cardiovascular:** Increased sensitivity to angiotensin II(sensitivity is normally decreased in pregnancy) and catecholamines with vasoconstriction, reduced plasma volume, odema and increased arterial BP (>160/110).
- **Renal:** Renal blood flow, GFR and urine output are decreased with proteinuria.
- **Haematological:** Fibrinogen,fibrin and platelets turnover is increased. HELLP syndrome (haemolysis, elevated liver enzymes, low platelets) may occur. Platelet function may be impaired.
- **Respiratory:** Pulmonary odema may occur.
- **Neurological:** Hyperexcitability and hyperreflexia; visual symptoms and headache, convulsion (eclampsia).

TREATMENT: Complete bed rest, control of hypertension, prevention of convulsions and delivery of the fetus if possible.

Antihypertensive drugs: alfa methyldopa, labetalol, hydralazine and calcium channel blocking drugs orally (nifedipine sublingually). Severe hypertension may requiresi.v.treatment with

- Labetolol 5-10 mg increments, or 10-200 mg/hrinfusion.
- Hydralazine 5-10 mg increments, or 5-50 mg/hr infusion.
- Sodium nitroprusideor NTG 0.1-5.0 microgram/kg/min.

Intravenous fluid: Because intravascular compartment generally depleted; central venous cannulation may be required. The choice of fluid is colloid should begin before iv vasodilators are given to avoid precipitous fall in BP and placental perfusion.

Diuretics – Dopamine (low dose) or furosemide along with fluid replacement in oliguria

Anticonvulsant drugs – Magnesium sulphate reduce the incidence of eclampsia by almost 60%.

ANAESTHETIC MANAGEMENT: Anaesthetic involvement may be required for analgesia during labor, Caesarean section or assistance with management of fluids, BP, etc.

Anaesthetic techniques:

- **Epidural anaesthesia:**

- Prevents the increase in catecholamines associated with pain, thus increasing placental blood flow.
- Avoids the risks of general anaesthesia.
- Contraindicated if there is a coagulopathy or low platelet count.
- Careful fluid management and local anaesthetic administration is required to avoid cardiovascular instability following blockade. Sensitivity to sympathomimetic drugs is increased

Use of relatively large doses of local anaesthetic agents has been questioned in patients with neurological symptoms.

Avoidance of adrenaline in local anaesthetic solution has been suggested but this is controversial.

If hypotension occurs, a crystalloid bolus(500 ml) may be administered, with the patient in lateral tilt and receiving O2. If this is not adequate, a bolus of ephedrine 3 mg or phenylephrine 50-100 microgram may be given.

- **Spinal anaesthesia:** Spinal anaesthesiahas traditionally been avoided because of the fear of sudden severe hypotension, but this may not necessarily occur if there is adequate volume expansion and pretreatment of the condition.
- **General anaesthesia:**

- Risks include difficult intubation (because of facial and laryngeal oedema), the hypertensive response to intubation and cardiovascular instability. Administration of antihypertensive drugs or opioid analgesic drugs (fentanyl 1-4 microgram/kg) before intubation has been used.
- Anticonvulsant effect of thiopental may be beneficial.
- Magnesium sulphate may result in increased sensitivity to neuromuscular blocking drugs.

Careful monitoring should continue after delivery.

DISCUSSION: Pre-eclampsia is a leading cause of maternal morbidity and mortality. Substandard care is often present and many deaths are preventable. The disease is defined within the context of hypertensive diseases, and early recognition of pre-eclampsia and its complications, as well as multidisciplinary expert team management is highlighted.

CONCLUSION: The ultimate goal of treatment of hypertension in pregnancy is delivery of a healthy newborn without compromising maternal health. Early diagnosis and subsequent close monitoring of both mother and fetus are crucial. Elevated blood pressure without proteinuria usually has a benign course and can be managed on an outpatient basis. Antihypertensive medications should be used judiciously, and fetal risks from intrauterine exposure must be carefully evaluated. Severe preeclampsia (both pure and superimposed) represents an obstetrical emergency, with potential fatal outcomes for both fetus and mother. Optimally, such women should be hospitalized and treated with bed rest, antihypertensive medications, and magnesium sulfate for seizure prophylaxis. The definitive treatment of preeclampsia is delivery. For mild forms remote from term, postponing delivery is desirable and, if possible can improve neonatal prognosis by decreasing prematurity.

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Etiologic Factors of Disease as per Ayurvedic Perspective

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ABSTRACT: Disease is as old as mankind itself. Human being has always tried to understand natural phenomena and attempted to give his own explanation to it. The philosophy of *Ayurveda* the Indian system of medicine has its origin in the Vedas dated about 5000 years ago. Later *Charaka Samhita* (400 B.C.), *Sushruta samhita* (1000 B.C.) and *Vagabhatta* (*Ashtanga sangraha*) (600B.C.) had scientifically documented the medical concepts. Disease or *byadhi* was described as an imbalance of the three physiological humours of the body viz *vata*, *pitta*, and *kapha.* The western perspective on disease is recorded around 400 BC. Hippocrates, the father of modern medicine, had proposed the four humeral theories in which the Black bile, Yellow bile, Phlegm and blood are the governing factors of health or disease. Prior to Hippocrates, disease was thought of as the handiwork of witches and sorcerers over which humans had no control. During Renaissance in Europe, scientific thought grew by leaps and bounds and rational explanations were sought for every phenomenon. This situation gave rise to many discoveries in medicine too. Unlike *Ayurveda*, whose theories are founded on universal and holistic principles, and remained the same for over 5000 years, the western theories of philosophy of health and disease aetiology have a relatively short history. Modern theories on disease aetiology like the germ theory and the genetic theory were described in *Ayurvedic* scriptures in Sanskrit language. The notion that these theories were already proposed in *Ayurveda* is increasingly being recognized after the translation of *Ayurvedic* books into English and other languages. Lifestyle disorders are gained utmost attention in recent times. In this regard *Ayurveda* attributes three prominent factors as the basic disease aetiology. They are *asatmendriyarthasamyoga, prajnaparadha* and *parinama*. In this paper, we tried to trace the ancient Indian concepts of disease aetiology.

Keywords: Aetiology, Disease, Prajnaparadha, Ayurveda

Introduction: The harmony in *Doshas*, *Dhatus*, *Malas*, *Agni* and *Mana* in their amount and function in the body is the primary condition for health. This equilibrium is maintained by following to the laws of nature. When it is disturbed by wrong food habits, lifestyle changes etc. diseases set in. The primary cause of a disease is vitiated *Doshas*. Both under pathological and healthy conditions, the *Doshas* behave in different patterns and elicit various signs and symptoms as per their vitiation, the spreading area and manifestation. *Dosha* vitiation can occur either in a normal manner or abnormally. The normal vitiation is produced by inevitable and natural factors such as seasonal variations, diurnal variations and various stages of digestion. This type of vitiation is easy to treat and does not require any specific treatment. Abnormal vitiation of *Doshas* is caused by deliberate exposure to specific etiological factors, both external and internal such as abuse of functions of sense organs, wrong bodily habits, suppression of body urges, bad food habits, evasion of seasonal and daily regimes etc. By studying the signs and symptoms triggered by the vitiated *Doshas* and *Dhatus*, one can find the cause of a disease. The basic principles of treatment in *Ayurveda* are to eliminate the root cause of a disease and to bring back the vitiated *Doshas*, *Dhatus*, *Malas* and *Agni* to the normal functioning state.

TRIDOSH THEORY: *Vata*, *Pitta*, and *Kapha* are called *tridoshas* in *Ayurveda*. *Tridosha* theory is the foundation of *Ayurvedic* concepts of health and disease. Further, there is an inherent link between *Tridosha* and *Panchamahabhuta* (five gross elements) theories. All material substances in the universe animate or inanimate, are the manifested form of the *Panchamahabhutas*, viz. *Akasha* (Sky), *Vayu* (Air), *Agni* (Fire), *Ap* (Water) and *Pruthvi* (Earth). Human is no different from the universe where he inhabits, and actually is a microcosm of the macrocosm called universe. Human body is the manifestation of the said gross elements wherein the *tridoshas* can be said as the bio-energies. Amongst the *tridoshas*, *Vata* is the outcome of the wind and earth elements, *Pitta* is the manifestation of fire element and *Kapha* is the result of the water and earth elements in the human body.

Importance of Aetiology: Aetiology of disease is a crucial concept that facilitates proper treatment. Diagnosing the root (actual) cause of disease removes from the mental or physical pain. After all, removing the root cause of disease is the first step towards proper treatment, and sometimes, is the only step that is essential. Medical tradition in the west has paid considerable attention to aetiology due to its valuable stake in the treatment process. *Sushruta* stated that ' *duhkha'* (grief) is the root cause of diseases-"***taddhukhasamyoga vyadhaya ucyante***" (association of miseries is called '*duhkha'*). That means, if one avoids *duhkha*, there would be no disease. In order to avoid, the removal of causes that are attributed with *duhkha*, needs attention. *Charaka* has listed the causes for *duhkha*. "Derangement of intellect, restraint and memory, advent of time and action and contact with unsuitable sense objects should know as the cause of misery".

Charaka Samhita has discussed three main reasons for disease:

"...asatmyendriyarthasamyoga, prajnaparadha, parinamashcetitrayastrividhakalpa hetavo vikaranamsamayogayuktastu prakrtihetavobhavanti "

1. *Asatmyndriyartha samyoga-* Is extreme use, under use and abuse of sense organs while aligning with their objects. That means indulging cognitive organs, viz., eyes, nose, ear, tongue and skin, in contrary methods cause disease. For instance, listening music in high decibel triggers ear related diseases, which is called ' *aindriyaka'*.

2. Prajnaparadha- Is intellectual blasphemy. Improper understanding of objects by intellect will result in adverse actions such as, negative thinking, misbehaviour with noble people, lack of knowledge controlling mind, lack of good conduct are some of the reasons for intellectual errors.

3. Kala- Is seasonal variation. Improper intake of food such as untimely consumption of eateries while ignoring seasonal changes etc., are due to *kalaviparinama*. Improper intake of food also causes lot of life style disorders such as stress etc. As an effect, people also suffer with stress/anxiety disorders, work tensions and so on and so forth.

DISCUSSION: More or less, the five elemental theory (*Panchmahbhuta siddhanta*) is the basic fundamental principal of *Ayurveda* systems of medicine. Humours make up one's constitution in both systems though respective constitutive members of humours vary in character, except the phlegm. In fact, the concept of 'phlegm' has been intended differently by *Charaka* as its scope is much wider than in modern system of medicine. Even though *tridosh* as per vade the entire body, specific regions are attributed as their seats. Phlegm is said to occupy the upper part of the body consisting head, neck, thorax, chest, upper stomach, fat tissues, lymph glands and joints. Further, it can be postulated that the *tridosh* as are not measurable using scientific equipment. Factors listed in *Charaka* were said to produce disease due to the in-equilibrium of *doshas*. Both systems agreed upon the root cause of hereditary diseases. Also, both believed that poor diet and seasonal variation affects the equilibrium of humours. But the three fold aetiology of *Ayurveda* is unique in many ways compared to modern medical system. *Astamyendriyarthasamyoga* is related to incompatibility senses with their objects. Most professional hazards like continuous peering into computer monitors, listening high decibel sounds through various means, looking at high beam lights, exposure to extreme heat or cold temperatures, smelling of chemical pollutants are the *'asatmya'* (incompatibility) of objects to corresponding sense organs. Abusing the sense of taste or its excessive usage could be termed as overeating which is the instigator for lifestyle disorders like Diabetes Mellitus. *Prajyaparadha* carries a greater relevance in contemporary times. Committing errors will fully is nothing but an intellectual blasphemy. Errors like smoking, alcoholism, abusing elders and noble people, drug abuse and sedentary life style with faulty food habits results in *Prajyapradha*. Humans have no control over seasonal variations. Hence, *Ayurveda* advocates *Ritucarya* and *shodhan* therapy for prevention and treatment purposes. Sushruta did consider divine/supernatural origin of disease amongst the other types. But in '*aadibalapravrutta'* type of diseases ' genetic disorders' were discussed for the first time in indigenous medicine. This concept speaks of the disorders that can take place in the seed of the male and the female that leads to genetic disorders in the progeny.

The concept on *Janmabalapravrutta'* byadhies states about disease occurring due to problems during pregnancy and child birth. For instance, prolong labour and birth asphyxia during child birth can lead to mental retardation of child. Looking again at the modern theories of disease it is apparent that Ayurveda addressed the issue of the genetic theory and lifestyle disorders or metabolic disorders like gout, obesity etc. in the texts. Neoplasia (karkatarbuda) was explained as an abnormal and unexplained growth of cells and tissues. Nutritional deficiencies too were considered a reason for disease. The best example for that being *Pandu* or Anaemia. Lohatatwa or iron was advised for this right from the *Samhita* times. Although vitamins and essential minerals were not explicitly named in those times, certain foods were considered healthy and certain to be unhealthy. Diet was a major factor behind health or sickness. Immune disorders are described in *Charaka*. Immunity is known as '*ojas'* and *'balam'*. The impression that the body's immune system kills its own cells and tissues is an invention of the twentieth century which was not known earlier. It is a matter of issue that auto immune disorders respond positively to *Ayurvedic* immune modulators like *Guduchi* (*Tinospora cordifolia*) etc. Amongst the study of most important aspect of all these theories the germ theory leads to interesting findings.

Although *Ayurveda*, the Indian system of medicine, lays emphasis on the *tridosha* disturbance being the cause of disease in as much they cause the in-equilibrium or the *tridoshas*. Germs were recognized and called with different names such as, *Bhuta, Krimi* etc. *Charaka* has enumerated about twenty such germs/microbes. *Graha rogas* (epidemical) mention isolating patients to avoid the spread of disease from person to person. In *Charaka Samhita*, a chapter called *janapadodhwamsavimanadhyaya* discusses epidemic breakouts and lists out polluted air, water and earth as its reasons. So, germ theory is entirely not unknown yet not overtly emphasized in the Indian system of medical thought. All factors of aetiology were examined for the disturbance they could cause to *doshas*. In addition to these *Ayurveda* went a head of its times to describe the psychological component of disease aetiology. Phobias and grief could cause disease. When Charaka spoke about the strong body-mind link and physical diseases causing psychological disturbances and psychological diseases causing physical disturbance, he was in fact speaking about psycho-somatic and somato-psychic diseases. This demands preventive or healing measures to mind and body.

CONCLUSION: The origin of the modern concepts of aetiology was already laid in Indian system of thought (*Ayurveda*) in context of disease. The four humeral theories from the time of Hippocrates paved the way for other concepts of aetiology like the germ theory and the genetic theory in the modern times. The *tridosha* theory is as relevant to *Ayurveda* today as it was 5000years ago. The significant concepts of aetiology in *Ayurveda* need a through revisit from the modern perspective.

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Critical Incident Reporting and Learning

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Abstract: The success of incident reporting in improving safety, although obvious in aviation and other high-risk industries, is yet to be seen in health-care systems. An incident reporting system which would improve patient safety would allow front-end clinicians to have easy access for reporting an incident with an understanding that their report will be handled in a non-punitive manner, and that it will lead to enhanced learning regarding the causation of the incident and systemic changes which will prevent it from recurring. At present, significant problems remain with local and national incident reporting systems. These include fear of punitive action, poor safety culture in an organization, lack of understanding among clinicians about what should be reported, lack of awareness of how the reported incidents will be analyzed, and how will the reports ultimately lead to changes which will improve patient safety. In particular, lack of systematic analysis of the reports and feedback directly to the clinicians are seen as major barriers to clinical engagement. In this review, robust systematic methodology of analyzing incidents is discussed. This methodology is based on human factors model, and the learning paradigm which emphasizes significant shift from traditional judicial approach to understanding how 'latent errors' may play a role in a chain of events which can set up an 'active error' to occur. Feedback directly to the clinicians is extremely important for keeping them 'in the loop' for their continued engagement, and it should target different levels of analyses. In addition to high-level information on the types of incidents, the feedback should incorporate results of the analyses of active and latent factors. Finally, it should inform what actions, and at what level/stage, have been taken in response to the reported incidents. For this, local and national systems will be required to work in close cooperation, so that the lessons can be learnt and actions taken within an organization, and across organizations. In the UK, a recently introduced specialty- specific incident reporting system for anesthesia aims to incorporate the elements of successful reporting system, as presented in this review, to achieve enhanced clinical engagement and improved patient safety.

Keywords: Good quality; procedure error, care of healthy being; safe and management of risk

Introduction: Patient safety has been, and still is, a cause for concern in health-care systems all over the world, including the NHS.Every year, _900 000 incidents and near misses are reported around NHS care, _2000 of which result in death. Additional hospital stay costs are approximately £2 billion a year, and the negligence claims amount to an extra £400 million a year.Incident reporting systems have been a key tool to improve safety and enhance organizational learning from incidents in a range of high-risk organizations (commercial aviation, rail industry, and others). Although incident reporting has been instituted in health-care systems in many countries for sometime now, similar positive experience is yet to be fully realized.

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In this article, I aim to review the essential components of a successful incident reporting system, framework for analyzing the reported incidents, and current understanding of barriers and enablers to successful incident reporting.

Incident reporting Systems: Investigation of critical incidents was first used in the 1940s by Flanaganas a technique to improve safety and performance among military pilots. Cooper and colleagues,in 1978, used a 'modified critical incident technique' in which they interviewed anesthetists and obtained descriptions of preventable incidents. Individual departments of anesthesia now have systems in place to record and discuss adverse incidents and near misses with a view to improve patient safety by learning from these incidents.

The main reason for reporting incidents to improve patient safety is the belief that safety can be improved by learning from incidents and near misses, rather than pretending that they have not happened.In the last two decades, authors have highlighted the need to gather information which can be used to improve hospital systems to minimize errors in healthcare,and many strategies and tools have also been developed to reduce errors.The calls have been made by quality and safety organizations, and the consumers of health-care systems,for incident reporting to better understand errors and their contributing factors. Internationally, WHO has work in progress to develop.

Analysis of the reported incidents: A good quality report should lend itself for detailed analysis of the chain of events that lead to the incident. This knowledge can then be used to consider what interventions, and at what level in the chain, can prevent the incident from occurring again. The concept and proposed framework of investigating and analyzing clinical incidents, as reviewed in the following, highlight the areas of information which a good quality report should be able to capture.

In the context of individual and organizational factors, often a complex chain of events can be seen that lead toan adverse outcome.It has been argued that the comprehensive analysis of incidents must pay attention topsychological and human factors in the nature, mechanisms, and causes of the error.In this regard, the national reporting systems have to work alongside the local risk management structures for comprehensive analyses andcross-learning from the incidents. Therefore, it becomes logical that a standardized framework is used at all levelsfor analysis of the incidents.

A high-level analysis of the number and kinds of incidents can be performed at the national level, and disseminated widely. This has the advantage of highlighting the areas for improvement (e.g. medication errors, retained throat packs), and for further focus by national organizations to trigger further actions such as raising awareness, research, audits, training initiatives, curriculum, and specific guidelines. However, such high-level analyses are not sufficient, on their own, to improve safety at the local level. Local safety initiatives of investigating and analyzing incidents are extremely important to get into the root cause of the incidents and how these can be prevented in the future.

The paradigm for analysis and learning: The traditional approach of quick judgments and routine assignment of blame often obscure a more complex truth. Also, the usual practice of analyzing only those incidents which lead to actual patient harm, in fact, misses big opportunities to learn from near misses, or where an incident was effectively managed without actual harm. Hence, the learning paradigm for incident reporting has to be shifted from the traditional 'judicial' approach towards a mutual search for opportunities for improvement.

Human factors approach: The causation of a safety incident which, at first, is identified by an obvious departure from good practice, or active failures, often has a number of factors related to the workingenvironment and wider organizational context working in the background and influencing the outcome. The 'human factors' approach focuses on the humancomponent within complex organizational (socio-technical) systems. Thus, it has less focus on the individual who makes an error and more on the pre-existing organizational factors that set up the conditions for an error to occur.The approach, based on Reason's model of organizational accidents and adapted for medical settings,allows examination of the chain of events that lead to an accident.

The contribution of human decisions, actions, or both to an accident can be due to active failures, latent failures, or both.Active failures are unsafe acts or omissions performed by the front-end workers (anesthetists, surgeons,nurses), and these include slips (wrong label, wrong syringe), cognitive failure (memory lapses, ignorance, misreading a situation), or violations (deviations from safe practices, procedures, or standards). Latent failures,in the context of health-care systems, refer to decisions taken by senior management or clinicians, which create the conditions in an organization for unsafe acts to occur; these conditions include inadequate or inappropriate staffing, heavy workload, poor supervision, stressful environment, poor communication, poor maintenance of equipment, and conflict of priorities (finance vs clinical need). Hence, in the analyses of adverse events, a systematic approach of understanding the anatomy of evolution or generation of incidents, and a hierarchy of the factors which are involved, should be undertaken.

For healthcare, Vincent and colleagueshave described a framework for analyzing critical incidents. This framework includes factors of relevance to medicine by combining the strengths of Reason's model of organizational accidents with socio-technical pyramid of Hurst and Ratcliffe. The framework has been summarized in Table 1. In this framework, the hierarchy of factors has been derived from previous publications,and includes the factors which are known to influence clinical practice and outcome. In this hierarchy of factors, patients and staff as individuals are at the front-end (bottom) of the factors, team factors and working conditions in the middle, andorganizational/institutional factors at the top. The condition of the patient, clearly, is an important direct predictor ofoutcome. Also, the adverse events are more likely to occur when the patient is already seriously ill.The experience, training, and familiarity with the working environment of the staff may also be influential. Each member of the staff is part of a team, and his/her performance may be influenced by other members of the team, and how teams are organized, and how they support, supervise, monitor, and communicate with each other. The team performance, in turn, is influenced by management decisions made at a higher level in the

Table 1 Framework as proposed by Vincent and colleaguesforanalyzing critical incidents

Main factorsContributory factors: Institutional Economic pressures, regulations, NHSexecutive, clinical negligence schemes,Organizational Financial priorities, structure, local policies,standards, safety culture,WorkStaffing, skill mix, workload, shift patterns,EnvironmentDesign, equipment availability and maintenance, support,Team factorsCommunication, supervision, team culture
IndividualKnowledge, skills, competence, health,Task factorsTask design, availability and use of protocols,test results, patient notes—Accuracy and availability,Patient factorsComplexity and seriousness, language, communication, personality,social factors Organization. Hence, the senior clinicians and managers may influence a team's performance by influencing the'work environment', which includes factors such as staffing level, working hours, equipment availability and maintenance, guidelines and protocols, and education and training. Finally, external factors such as political climate and priorities, financial constraints, regulatory bodies, and public expectation may have a powerful effect on the working of an organization.
The framework for analysis can also be taken to understand what components of information are required in agood quality report to allow a detailed, systematic, and meaningful analysis. Crucially, the framework provides theresearchers and the risk managers a formal structure for collection of information and analysis of critical incidents, where rather than focusing mainly on the actions of the front-line staff, the emphasis is on examining the whole gamut of possible influences. The safer practice can only come from acknowledging all the possible factors in the potential for error, and building in multi-level error reduction strategies at every stage of the chain that leads to generation of anerror.This comprehensive approach of multiple levels of intervention requires the clinicians and the managers to significantly shift away from the often practiced, and rarely effective, approach of one-level of intervention (e.g. stafftraining or tightening protocols).
Components of incident analysis: A clear definition is required of which incidents should be reported and investigated. An incident that leads to patient harm always gets investigated according to its seriousness as per local governance policies. In this regard, some investigations are started almost immediately. However, this process should not underestimate the potential of analyzing incidents that are near misses, or which have not led to patient harm.
The elements of an investigative or analytic process, as in practice, are summarized in the following.
Identifying the most obvious active failure (s): The active failures are also known as care management problems (CMP). These include delayed diagnosis, inadequate handover, failure to monitor, lack of preoperative check, protocol violation, incorrect treatment, not seeking help, inadequate supervision, etc.
Framing the problem: This is not straightforward. Often the problem originates at a time point which is earlier than the time point at which the problem occurs. Therefore, accurate assessment of chronology and the details of the events leading on to the incident is important in framing the problem.

Defining the problem (what, how, and why): In addition to the reported incident, case notes studies and interviews of the key staff members may be undertaken. The line of enquiry should first determine exactly what happened in terms of CMPs and chronology of the events. In the next stage, it should establish, without being punitive, how it happened. All important acts or omissions made by staff, and with hindsight the important chain of events which set up the conditions for the incident to occur. Subsequent line of enquiry should elicit the reasons behind certain acts or omissions. The next step is to define why. For each CMP, contributory factors, as outlined in the framework, should be explored. These could be specific contributory factors at different levels (e.g. lack of knowledge or training at individual level, unavailability of protocols at task level, poor communication at team level, or inadequate staffing at organizational level). The specific factors will need to be distinguished from, or studied in context with, general contributory factors such as poor safety culture within an organization, overall poor communication, poor training, overstretched staff rotes, or faulty/incomprehensibleguidelines.
A separate analysis should be carried out for each CMP using a standardized framework. The final analysis willreport summary of chronology, CMPs, and their contributory causes, and give recommendations for further actions for each contributory factor (in particular, the general contributory factors).

Strengths, limitations, barriers, and enablers: Among different strategies to gather information and reduce errors, review of a randomly selected, or targeted, sample of medical records has also been used to identify problem areas. However, because of the limitations in the exiting classification system, infrequently occurring errors may not be picked up using this method. One of the strengths of incident reporting is that it tends to capture more contextual information about the incidents.Also, successfullyimplemented incident reporting can detect more preventable adverse events than medical record review, and it is more cost-effective.
The medical records, although reasonably good at describing adverse events, rarely document near misses. Inpractice, near misses occur more frequently than the adverse events37 and provide equally valuable informationfor drawing up of important clinical lessons without the detrimental consequences of an adverse event.Hence,reporting of near misses provides valuable information for systems improvement without patients or staff having suffered the consequences of adverse events. Despite the known and well-advertised strengths of the incident reporting systems, under-reporting, in particular by doctors, remains a significant problem. It is possible that the incidents are just not recognized, or are not simply documented properly.However, there may be deeper cultural issues acting as barriers to incident reporting. The rates of adverse events are estimated to range between 2.9% and 16.6% in acute care hospitals.It is therefore only logical to assume that the doctors and the nurses working in hospitals will be familiar with these events, and would have come across and reported them. In a recent study,despite most staff being aware of the existence of an incident reporting system, 25% did not know how to access an incident form, and more than 40% of consultants and registrars hadnever completed a report.
The research has shown that, in general, only a small percentage of doctors report incidents formally.One of the reasons could be unfamiliarity with the process.Other factors which have

been identified are cultural issues such as fear of punitive action,legal ramifications, and discrimination at the workplace.Poor reporting practices by doctors may also reflect prevailing deeply entrenched belief in medicine that only bad doctors make mistakes.

Other factors responsible for poor reporting are related to lack of clarity regarding what should be reported, and how the reports might lead to improvement in the existing systems.A recent study has confirmed the commonlyobserved phenomenon that the incidents which were immediate, and often witnessed (e.g. falls, equipment problems,drug errors) are better reported than the incidents which had gradual development, and could not be assigned
to a single causative factor, or were considered to be known complications of hospitalization (hospital acquired infections, deep vein thrombosis).Many staff do not consider near misses to be reportable incidents, which are a rich source for learning.Also many doctors do not consider omission of medication to be reportable, which again indicates lack of essential knowledge about what should be reported, given that acts of omission have been implicated in twice as many adverse events as acts of commission.Organizational factors which make reporting difficult (long forms, insufficient time, and no feedback) have also been identified as major barriers to reporting.

Studies that have shown to improve incident reporting have used strategies of intense facilitation, either throughward rounds or staff reminders.The level of reporting in an organization has also been correlated with the existing safety culture.Therefore, at organizational level, any effort to improve incident reporting and learning should begin with assessment of prevailing safety culture within an organization, and long-term, sustained programmed of improving it. The key to the success of incident reporting systems in improving patient safety lies in the fact that the front-line clinician must know and believe that the reported incidents will not end up in a ‘dark hole’, but will be analyzed in a systematic non-punitive

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Clinical Effects of Tarpana Therapy on Refractive Errors

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ABSTRACT: Eye is an important sense organ. It has been enjoying privilege as the main sense organ hence it is very important to protect vision from various diseases. Inspite of rapid development of modern ophthalmology still there are many diseases where modern techniques do not prove much effective .Refractive errors, Errors of Accommodation, Progressive cataracts, different condition of retinopathy, severe allergic problems of the eyes etc are conditions where Ayurvedic treatment can be appreciated .*Tarpana* therapy with ocular exercises and Ayurvedic medicaments prove to be effective in improving vision. Refractive errors like myopia,hypermetropia,astigmatism are conditions where *Tarpana* therapy is effective .Thus a randomized study was carried out on the patients who have presented with complain of diminished vision and asthenopic symptoms in Shalakya OPD of IMS BHU and have been prescribed with *Tarpana* therapy .The cases were studied with an objective to find out the effect of *Tarpana* therapy on refractive errors.*Tarpana* therapy was found to be effective in improving vision and asthenopic symptoms especially in children .

Keywords*:* *Kriyakalpa, Tarpana karma, Snehana, Nadi swedana, Nasya, Urdhawang pradesh.*

INTRODUCTION: Emmetropia (optically normal eye) a state of refraction where in parallel rays of light coming from infinity are focused at the sensitive layer of retina(innermost layer of eyeball) with the accommodation being at rest. Ametropia (a condition of refractive error) is defined as a state of refraction where parallel rays of light coming from infinity ,with accommodation at rest ,are focused either in front or behind the sensitive layer of retina . Ametropia includes myopia, hypermetropia and astigmatism. [1]

Inspite of the development of modern ophthalmology still there are many problems where modern techniques has its limitation and is not as effective. Refractive errors (specially in children) are such conditions where ayurvedic treatment is found to be effective. In Kayachikitsha *Panchkarma* forms an important part of treatment as such in Netrachikitsha *Kriyakalpa* plays an important role in treatment. [2] *Kriyakalpa* includes *Tarpana* , *Putpaka, Seka/Pariseka, Aschyotana, Anjana*. Patients in which eyes have become very inactive,dry,rough,hard ,eyelashes fallen off,dirty,irregular and afflicted greatly by diseases derive strength without doubt from *Tarpana* therapy. [3] Also in the patients of krchronmilan (difficulty in opening eyes),arjuna(subconjunctival haemorrhage),after relief from redness,lacrimation,pain ,swelling and excretions *Tarpana* therapy can be given. During temperate seasons both in morning and evening when patient is lying with his face upwards in a room devoid of breeze ,*Tarpana* therapy can be given. [4] Also when one eight part of day has elapsed (after lapse of three hours from sunrise) it can be given. [5] Considering its indications and contraindications when given with appropriate methods *Tarpana* therapy with ocular exercise and ayurvedic medicines has been found to be effective in improving vision.

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AIMS AND OBJECTIVE: To evaluate the efficacy of Tarpana therapy on patient with refractive errors and asthenopic symptoms.

MATERIAL AND METHOD: It was an open randomized study on refractive error patients of Shalakya OPD of Sir Sunderlal Hospital, IMS, BHU .Previous and fresh cases were taken for the study.

Three sittings of *Tarpana* was given to each patient with fifteen days gap in between each sitting, one sitting of *Tarpana karma* was completed in ten days .

It included: First three days: *Snehana* of urdhwang pradesh (above clavicle region) with Ksheera bala taila, followed by Swedana with Dasmool kwatha and Nasya Karma with Anu taila.

Next seven days: *Tarpana karma* with *Mahatriphaladya Ghrita.*

Masha pali was prepared (a chamber formed around the eyes with black gram paste) and stick around the eyes to a height of 2 angula(finger's breadth).Keeping the eyelids closed the medicated ghrita liquified by hot water was poured into the cavity upto the level of tip of the eyelashes. [6] With continuous blinking of the eyes the ghrita was retained for 15 minutes followed by removing the ghrita by doing perforation in *masapali* at temporal side. [7] The *masapali* was then removed followed by cleaning the region with cotton soaked with lukewarm water.

OBSERVATION AND RESULTS:

Pt.OPDNo.	Age	Chief Complaint	Before *Tarpana* therapy		After *Tarpana* therapy	
SK7528/13	10 yrs	Pain and Heaviness in both eyes	VA BE With glass	6/36 6/9	1st sitting BE with glass 2nd sitting BE with glass 3rd sitting BE with glass	6/24 6/9 6/18 6/6 6/9 6/6
SK7529/13	6yrs	Diminished vision BE	VA RE LE with glass RE LE	4/60 6/12 6/36 6/9	RE 1st sitting 6/36 With glass 6/18 2nd sitting 6/18 with glass 6/9 3rd sitting 6/9 With glass 6/6	LE 6/12 6/9 6/9 6/6 6/9 6/6

Pt.OPDNo.	Age	Chief Complaint	Before Tarpana therapy		After Tarpana therapy	
SK6243/13	10yrs	Difficulty in studies	VA BE with glass	6/36 6/12	1st sitting BE with glass 2nd sitting BE with glass 3rd sitting BE With glass	6/24 6/12 6/18 6/9 6/9 6/6
SK3087/13	6yrs	Heaviness in both eyes Difficulty in studies	VA BE with glass	6/18 6/9	RE 1st sitting 6/12p with glass 6/9 2nd sitting 6/9p with glass 6/6p 3rd sitting 6/9 with glass 6/6p	LE 6/18 6/9 6/12 6/9 6/9 6/6p
SK2287/13	13yrs	Diminished vision	VA BE with glass	6/24 6/6	RE 1st sitting 6/18 with glass 6/6 2nd sitting6/12p with glass 6/6 3rd sitting 6/12 With glass 6/6	LE 6/24 6/6 6/18 6/6 6/12p 6/6

VA (Visual Acuity with Snellen's chart), BE(both eyes), RE(right eye), LE(left eye)

DISCUSSION: Akshi tarpana is a unique procedure where medicated ghee is retained over the eyes for a specific period of time with continuous blinking of the eyes .The medicated ghrita nourishes and strengthens the eye structure .[7] Before Tarpana therapy *sodhana karma* (purification) is to be done for which *Nasya* therapy was given.The process is to be done during morning or during evening thus morning hours was selected .The season was such that it was neither too hot nor too cold nor cloudy atmosphere.The *pali*(the chamber formed around the eyes with urad paste)size is said to be *2 angula pramana* in height. therefore it was kept approximately 4cm in height with appropriate thickness.For healthy individuals the ghrita is to be retained for 500 *vakmatra* and in diseased according to *vatadidosha.* [8] Here the ghrita was retained for 15 minutes which approximated to 450 to 500 blinks. With the appropriate *Tarpana* therapy patient gets ability to withstand bright light, normalcy(in size, shape, colour etc)

,clearness of vision, feeling of lightness. [6] It was found that *Tarpana* therapy is effective in relieving asthenopic symptoms which occur due to fatigue of the ciliary muscle. Improvement in the diopteric power of spherical , cylindrical lens was seen.The lipophilic nature of ghrita facilitates the entry of drug into the eyeball through corneal surface since corneal epithelium is permeable to lipid soluble substances thus there may occur change in the refractive index of cornea also there may be change in axial length of the eyeball.

CONCLUSION: *Tarpana* therapy with ocular exercises (like palming, rolling,squeezing etc) and ayurvedic medicaments are effective in improving vision (specially in children).Along with improvement in vision and improvement in asthenopic symptoms was also seen. . Specially in school going children,children with hypermetropia resuls were good. Thus we see that *Tarpana* therapy is effective in treating refractive errors(specially in children).In order to see how actually *Tarpana* therapy works researches are being carried on this direction.

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Concept of Anupana in Ayurveda

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Abstract: Ayurveda is the nature based system of medicine known for its holistic approach. Its main aim is to provide healthy long life. It is achieved by the proper use of diet, bodily activities and medicines. Medicines are considered as the *karan* (means) to establish the *Dhatusamya*. It is possible when these are used considering the formulation, processing, dose, duration, Anupana (after drinks) and many other significant factors. The concept of *Anupana* is of greater importance and has been given weight age in the Ayurvedic medicine. *Anupana* means a liquid material which is taken along with or after the food and drug. *Anupana* word has been used in Ayurveda mainly in two sense i.e. *Anupana* (after drinks) and *Sahapana* (drinks taken along with diet or medicine). The common *Anupana* used are cold and warm water, honey, milk, ghee, buttermilk, juices and decoctions of different medicines etc. It is advised considering the *dosha*, *dushya*, *roga*, *rogi*, *desha* (habitat), *satmya* (acclimatization), properties of substance being used and purpose. It is equally important for the efficacy of diet and medicines. It causes the proper digestion and assimilation of dietary preparations. *Anupana* is necessary because if *Anupana* is not taken in advised form, then the solid food which is taken will not mix with liquid portion, remains without any movement and causes disorders. It increases the potency of medicines by enhancing the bioavailability. It is useful in *Shaman* as well as *Shodhan-chikitsa*. One drug may be administered in various diseases with different *Anupana*. It is a substance used to increase the action of principal ingredient or to modify it.

Keywords: *Anupana*, diet, medicine, Ayurveda.

Introduction: *Bheshaja-avacharana vidhi* (Drug administration) in Ayurveda is based on the specific scientific fundamentals. It includes the selection of drug, its dose and duration, its form being used, significantly *Anupana* and above all examination of the patient. The concept of *Anupana* (after drink) is equally important for the diet and the medicine which forms an integral part of *chikitsa* (treatment) and has been discussed in Ayurvedic literature since long back. It brings certain changes in the efficacy of a substance along with which it is used. *Anupana* has multidimensional effects as it acts as a vehicle, carrier, nutritive, stimulant, neutraliser, as an adjuvant in preventive and curative medicine. According to Monier Williams, *Anupana* is a fluid vehicle in medicine; drink taken with or after medicine; drink after eating; drink to be had near at hand[1]. Ayurveda is the unique system of medicine known for its holistic approach. Its main aim is to provide healthy long life. It is achieved by the proper use of diet, bodily activities and medicines. Medicines are considered as the *karan*[2] (means) to establish the *Dhatusamya*. It is possible when it is used considering the formulation, processing, dose, duration, Anupana (after drinks) and many other factors. The concept of *Anupana* is of greater significance and has been given weight age in the Ayurvedic medicine. *Anupana* means a liquid material which is taken along with or after the food and drug. The common *Anupana* used are cold and warm water,

honey, milk, ghee, buttermilk, juices, decoctions of different medicines and many others. It is advised considering the *dosha*, *dushya*, *roga*, *rogi*, *desha* (habitat), *satmya* (acclimatization), properties of substance being used and purpose. It is equally important for diet and medicine. It causes the proper digestion and assimilation of dietary preparations. *Anupana* is necessary because if *Anupana* is not taken in advised form then the solid food which is taken will not mix with liquid portion, remains without any movement and causes disorders[3]. It increases the potency of medicines by enhancing the bioavailability. It is useful in *Shaman* as well as *Shodhan-chikitsa*. One drug may be used in various diseases with different *Anupanas*. It is a vehicle or carrier used to increase the action of principal ingredient or to modify it.

Etymology and definition of *Anupana:* *The* word '*Anupana'* is derived out of two words: *Anu+Pana.* The word *'Anu' is* prefix which means after or along with and the word *'Pana'* is derived out of *"Paa" dhatu* (root) and *'Lyut'* suffix which means drink or drinking. Hence the term '*Anupana'* means the potable taken along with or after the diet and medicine[4].

As per *Acharya Charak*, *Anupana* should possess the properties opposite to the food but at the same time should not contradict with the qualities of *Dhatus*[5]. According to *Dalhan*, the liquid substance which is taken after the meal is called *Anupana*[6]. *Acharya Vagbhat* states that "*Anupana* is advised to be taken daily and it should have qualities which are exactly opposite to the food consumed prior to it. At the same time,*Anupana* should not adversely react with the food"[7]. *Adhamalla*, the commentator of *Sharngadhar- Samhita*, says that any liquid medium which is used after administration of the drug is known as *Anupana*[8]. The *Anupana* is claimed to distribute the drug throughout the body within no time. It spreads like oil drops on water means spread in all the direction fast. In modern system of medicine, the term *Anupana* may be taken as vehicle, adjuvant or carrier through which the actions like digestion and absorption of the drug are facilitated.

Significance of *Anupana:* As per *Acharya Charak and Sushrut Anupana* is refreshing, pleasing, energy providing, nourishing, brings satisfaction and steadiness in food consumed, help in breakdown of food particles, soften the food, brings the unctuousness of food, helps in proper digestion, helps in proper assimilation, helps in instant diffusion of food, improves the taste, causes stoutness, virility, relieves fatigue, acts as an appetizer, alleviates *doshas*, quenches thirst, improves strength and complexion [9]. Looking towards these properties of *Anupana*, it seems that either it is the digestion, assimilation and healthy effects of diet or alleviation of *dosha* and diseases, *Anupana* is to be considered sincerely. It is quiet difficult to have the desired effects of diet and medicine without proper *Anupana*.

Glimpse of *Anupanas:* All the scholars of Ayurveda have discussed about the *Anupana* considering *desha* (habitat), *kala* (season), *dosha*, disease, nature of diet and medicine. As per *Acharya Charak*, *snigdha* and *ushna Anupana* in *Vatik* disorders, *madhura-shita* in *Paittik* and *Ruksha-ushna* in *Shlaishmik* disorders should be advised. He considers milk as nectar for those who are exhausted due to fasting, way faring, excessive talk, sexual intercourse, exposure to wind and sun, excessive physical activities, *sura* (a type of wine) for emaciated persons, *madhudak* (honey mixed with water) for obese, *madya* (wine) for the persons who are suffering from weak digestive fire, insomnia, drowsiness, grief, fear and for those who are acclimatized to

madya and meat[10]. *Acharya Sushruta* has discussed a separate *Anupana-varga* in his text. He states that *madhura- rasa Anupana* should be advised to those who have aversion due to excessive use of *amla-rasa* and vice versa. Further he says that luke warm water should be advised as *Anupana* after ghee, oil etc. *snehas* except *Bhallatak* and *Tuvarak* oil, *yusha* and *amla* congee for oils, cold water for honey and rice flour products, milk and meat-juice for those who take *Shali* variety of rice, green gram etc. as diet, and who are indulged in battle, way faring, exposed to sun, poison, wine and having pain in the body, *madya* for those who are acclimatized to *madya* and all variety of meat, milk and sugarcane juice for the patients suffering from *Raktapitta* and *Asavas* prepared with *Arka*, *Shelu* and *Shirish* in poisonous conditions. Apart from all these he has indicated *Anupana* for *Vatadi-doshas* and even for different classes of dietary substances. He has stated that the *Mahendra-toya* (directly collected rain water) is considered the best among all the *Anupanas*[11]. *Acharya Vagbhat* has suggested some specific *Anupana* such as cold water after taking *Yava* (barley), *Godhuma* (wheat) preparations, curd, alcohol, poison, and honey, luke warm water in the preparations of *Pishti* (rice flour), curd water, Takra (buttermilk), Amlakanjika (fermented gruel) after vegetables, green gram etc., meat juice or soup in shosha (consumption) and many others[12]. Yogaratnakar has vividly discussed the Anupana of different diseases and designed a separate chapter on Anupana in his text. He has enumerated the Anupana of numerous diseases such as Chirayata, Nagarmotha, Pittapapada in Jwara roga, Takra in Grahani roga, Kutaja-twak kwath in Atisara, Vayavidang in Krimiroga, Bhallatak in Arsha-roga, Mandur in Pandu roga, Shilajit in Kshaya-roga, Brahmadandi and Shunthi in Shwas-roga, Amalaki and Haridra in Prameha-roga, Swarna nirvapit Jala in Trishna-roga, Hingu and Karanja in Shula-roga, castor oil with Go-mutra in Amavat-roga, Pippali in Pliha-roga, Shirish in Vish-roga, Suvarna bhasma in Gara-vish, Laghu-Kantakari in Kasa-roga, Shuddha Guggulu and Rason in Vatavyadhi, Vasa-kwath in Raktapitta,Vacha and Brahmi in Apasmar-roga, rechan (purgative) in Udara-roga, Guduchi in Vatarakta-roga,Vatak prepared with black gram in Ardit-roga, Madhu and Jala in Medo-roga, Lodhra in Pradar-roga, Bijora Nimbu in Aruchi, newly harvested Guggulu in Vrana, madya in Shok (grief), Draksha in Amlapitta, Shatavari and Shwet-Kushmanda in Mutrakrichchhra, Triphala in Netra-roga, Puran-Ghee in Unmada, Khadir-sarodak in Kushtha-roga, Buffalo milk in Nidra-kshaya, Bakuchi-phala in Shwet-Kushtha, Nidra in Ajirna, Santosh (consolation) in Bhaya (fear), Laja and Madhu in Chhardi-roga, Tikshna-Nasya in Urdhvajatrugata-vyadhis, Pushkarmul in Parshvashul, cold measures in Murchchha, Mamsa-rasa in Krishata, Pashanbheda in Ashmari-roga, Shigru-twak in Gulma–roga, Raktamokshana in Vidradhi, Laksha-nasya in Hikka roga, Shital kriya (cold measures) in Daha (burning sensation), paste of the bones of earth worm and dog prepared with the blood of donkey in Bhagandar-roga and Pushkarmul with Madhu in Swarbheda roga should be given[13]. In Sharngadhar Samhita, the quantity of Anupana for churna (powders), Avaleha (linctus), gutika (tablets) etc. is mentioned according to the doshaja vikara. e.g. in Vata Roga -3 pala (120 ml), Pitta roga -2 pala (80ml) and in Kapha Roga -1pala (40 ml)[14].It is an average quantity indicated and physician can decide more or less depending upon the need.

Contraindications of *Anupana:* In the context of *Aharopayogi-Anupana*, various contraindicated conditions have been enumerated in *Sushrut-Samhita* such as difficulty in breathing, cough, diseases above the clavicle, injury of chest and hoarseness of voice[15]. As per *Vagbhat*, it is not good in diseases of the organs above the clavicle, dyspnoea, cough, injury to chest (lungs), rhinitis, for those engaged in singing and speaking, in hoarseness of voice, those who are over hydrated, who are suffering from *Meha* (diabetes), diseases of the eye, throat and wound[16]. If the *Anupana* is given in above mentioned conditions, it causes the *Amashaya Dusti*. It diminishes the *Agni* by the *Abhisyandi* properties of *sneha* which is located in *urah* and *kanth* region and causes vomiting etc. disorders[17]. In this regard, *Anupana* of plain water is contraindicated. Other *Anupanas* suitable for particular disease are not contraindicated. As per *Acharya Sushrut*, after the use of *Anupana*, the person should not indulge in the activities such as journey, exercise, too much of study, too much of talk, too much of sleep and too much of singing. It may hamper the normal functioning and benefits of *Anupana*.

Discussion: The ancient *Acharyas* like *Charak*, *Sushrut* and *Vagbhat* have given the definition of *Anupana* in relation to food only such as; *Anupana* should be opposite to the qualities of food, but it should not be opposite to the qualities of *dhatus* (body tissues). Such *Anupana* is always considered as an appropriate *Anupana*, because it causes all the benefits of the diet. The later *Acharyas* like Sharngadhar Mishra, author of *Yogaratnakar* have mentioned the *Anupana* in the context of administration of *Aushadha* (medicine) also. The *Aushadha* becomes more potent when given with suitable *Anupana* by considering the *avastha* (stage) and *bala* (strength) of the *rogi* (patient) and *roga* (disease). *Acharya Sharangdhara* has stated that, the *bheshaj* followed by *Anupana* spreads quickly in the body as the oil drop added to the water spreads in fraction of time[18]. It indicates that when we administer the medicines with the *Anupana*, it spreads quickly, due to the *yogavahi*, *sara* etc. properties of *Anupana* which act like catalyst. When two substances are administered simultaneously, one may alter the response of the other which may be a beneficial or harmful effect. The basic aim to use *Anupana* is to get desired effects completely such as *Sitopaladi-churna* with *Anupana* of ghee and *madhu*, *Yasthimadhu* (Liquorice) with *dugda* (milk) etc. In some cases *Anupana* is given to check undesired effects such as milk and cow ghee as an *Anupana* in *Bhallataka-rasayana. Yogaratnakar* contains a long list of *Anupanas*. It should be taken as the gradual development of the concept. *Nidra* in *Ajirna*, *Santosh* (consolation) in *Bhaya* (fear), *Raktamokshana* in *Vidradhi*, *Laksha-Nasya* in *Hikka roga*, *Shital-kriya* (cold measures) in *Daha* (burning sensation) advised as *Anupana* in *Yogaratnakar* are debatable as they are not having the meaning of after drinks. It seems that the author of *Yagaratnakar* considers *Anupana* in the form of therapeutic procedures, regimens also. Most of the scholars are of the opinion that the *Anupana* means any liquid substance used with or after diet and medicines and it seems appropriate if we consider the etymological meaning of the term. Effect of *Anupana* depends on the selection of *Anupana*, time of administration, habitat, season, nature of medicine and diet, disease and diseased person such as buttermilk in *Grahani*, *Arsha* and *Udara-roga*, milk in *Pitta-vikara*, *Rasayana* and *Vajikaran*, honey and *yusha* in *Kaphaja-vikaras*, *Tandulodak* in *Pradar-roga* are considered as good *Anupana*. Effect of *Anupana* depends upon *desha* (habitat) and *Kal* (time) such as *snigdha Anupana* may be

appropriate in certain cases in arid land where as *ruksha Anupana* in marshy land will be appropriate *Anupana* in most of the cases. *Anupana* taken before,with or after food causes emaciation, normalcy of the body tissues and obesity respectively as stated by *Sushrut* in his text. *Anupana* should be advised according to the acclimatization also as generally those substance causes benefits in the body which are suitable. *Anupana* depends upon the *doshas* also because the nature of *Vata, Pitta* and *Kapha* dosha is not same. Disease also creates the difference in *Anupana* such as bleeding disorders needs cold *Anupana* where as dyspnoea, common cold, cough require hot one, constipation needs *sara-guna Anupana* where as in *Atisar, sthir* and *grahi-guna Anupana* is needed. Some medicines are fat soluble while other are water soluble, so considering this fact, *Anupana* should be decided otherwise proper effect cannot be achieved. *Anupana* is being applied in many forms such as plain water, water processed with medicines, decoction, juice, hot and cold infusion, milk, ghee, oil, wine, meat-juice, *Yusha*, *Asava-Arishta* and many more. Pure rain water is considered as the best *Anupana*, because it does not have any taste and contamination. Due to absence of taste, it does not counteract with the diet or medicine with which it is used. It provides a good media to the diet and medicine to be diffused in the body. Quantity of *Anupana* cannot be fixed because it varies from person to person on the base of *dosha*, age, season and nature of substance.

While we see the contra-indications of *Anupana, Urdhvajatrugata vyadhi, Kasa, Shwasa, Hikka* and who indulges in excess talking and singing etc. are not advised to take Anupana and it is for *jala* (water) as an *Anupana*. Because the *jala* (water) *Anupana* removes the *snehamsha* (unctuousness or moisture) of these parts leading to *Vata prakopa* (aggravation of vata) hence the *Anupana* act as *rogakara* (causes diseases) instead of *rogaghnata* (mitigating). In such a condition, one can advise the *Anupana* which is beneficial in that particular disease. Generally it is observed that *Anupana* of the juice or decoction of the medicine which is useful in particular disease, is more beneficial.

Conclusion: The concept of *Anupana* plays significant role in the prevention as well as cure of the diseases if properly used. *Anupana* means liquid substance taken with or after the diet and medicine. It has been used as both the *Sahapana* and *Anupana* in Ayurvedic treatment. *Acharya Charak, Sushrut* and *Vagbhat* have discussed it as *Annanupana* but advised different *Anupanas* in the context of diseases and their treatment also. *Acharya Sushrut* has discussed a separate *Anupana-varga* and *Yogaratnakar* contains a separate chapter on *Anupana* which shows the gravity of the concept. *Anupana* increases the properties of the substances but on the other hand, it checks the untoward effects of the medicine. It is like a carrier, vehicle, adjuvant, stimulant, neutraliser and even a medicine.

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Efficacy of Swarna and Lauh Shalaka in Comparison to Panchdhatu Shalaka for Agnikarm Therapy

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Abstract: Agnikarm is a well established ayurvedic therapy for the management of different diseases eg Skin diseases. Sandhivata, Gridhrasi, Kativata and Vrana. These all diseases which are mentioned in ayurvedic text are painful in nature. It is observed that this therapy was useful for management of pain too. Till date there is no safe drug or therapy available for the management of pain. Every drug possesses side effects eg. Gastritis, Constipation or Addiction. Acharya Sushruta has explained that the diseases treated with Agni karma modality don't reoccur.

Key Words: visual analogue scale, Karnofsky performance Scale, Pricking Sensation, Radiation of pain, Vedanahara, Sandhivata, Aloevera, Yashtimadhu, Triphala ,Panchdhatu ,Shalaka

INTRODUCTION: Pain is subjective feeling. There are many words which have been used for pain e.g. Vedana, Shoola, Dukha, Ruja, Pida. Pain is pathological symptom predominantly caused by vata. Hence while screening the drug for this problem we shall have to keep these in mind and select some outstanding drugs which may prove useful in this regard. A clinical study was planned to evaluate the efficacy of different types of shalaka used for Agnikarma therapy . Agnikarma is mentioned in ayurvedic texts to cure several disease one of these are vata vyadhi. Although the pain in vata vyadhi is not fatal one but its duration and attack at night makes the sufferer life miserable and crippled. On the basis of clinical parameters, it has been kept under Vataj lakshana. It is an established fact that pain inducing disorders in which the predominance, vata doshas are relieved by Agnikarma.

Materials & Methods:

Technique to be employed –

(i) Sterilization of the area with triphala kwath

(ii) Agnikarm with Panchdhatu shalaka/ Swarna shalaka/Lauh shalaka

(iii) After agnikarm, application of Aloe Vera & Yashtimabhu (*Glycerrhiza glabra*) churn.

(iv) The site of Agnikarma will be covered by bandaging. The observation will be noted on a standard Performa and finally on the basis of observation a result will be concluded

Selection of patients

Exclusion criteria: The patient of Hypertension, Heart diseases, diabetes Renal disorder, Bleeding disorder, Hyperpyrexia etc. will be excluded for research work.

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Inclusion criteria: All patient of suffering from Sandhivata- arthritis (Joint pain) of either sex age between 15 to 70 years.

Criteria for Assessment: Improvement in the patient has been assessed mainly on the basis of relief in the cardinal signs and symptoms. To assess the effect of therapy objectively, all the signs and symptoms were given scoring depending on their severity as below:

1. Pain (Ruja)

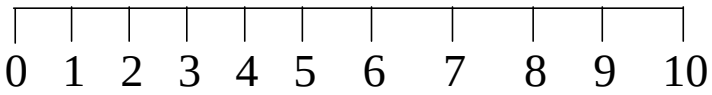
A) Visual Analogue scale – 0 to 10

0 = no pain

1 - 3 = mild pain

4 - 7 = moderate pain

8 - 10 = severe pain



2. Pricking sensation (Toda)

a)	No pricking sensation	0
b)	Occasional pricking sensation	1
c)	Constant mild pricking sensation	2
d)	Constant moderate pricking sensation	3
e)	Constant severe pricking sensation	4

3. Karnofsky performance scale–

a)	Normal activity with no special care	1
b)	Unable to work but able to live at home	2
c)	Needs hospital care	3

4. Radiation of pain

a)	No radiation of pain	0
b)	Pain radiates up to thigh	1
c)	Pain radiates up to knee joint	2
d)	Pain radiates up to leg	3
e)	Pain radiates up to ankle	4
f)	Pain radiates up to foot	5

5. Tenderness

a)	No pain on palpation	0
b)	Pain occurs on deep palpation	1
c)	Pain occurs on light palpation	2
d)	Patient does not allow to touch the Affected part	3

Observation & Results:

CLINICAL STUDY:

Table No. 1. The number of patients and treatment in the selected groups.

Group	No. of Patients	Treatment	Observation
Group I (Panchadhatu)	20	Agni Karma on the most painful part of the body with Panchadhatu Shalaka	Before treatment After treatment 1st sitting After treatment 2nd sitting After treatment 3rd sitting
Group II (Swarna)	20	Agni Karma on the most painful part of the body with Swarna Shalaka	Same as above
Group III (Lauh)	20	Agni Karma on the most painful part of the body with Lauh Shalaka	Same as above

Table No.2: The mean, standard deviation and statistical comparison of age, weight and height between the groups

Age, Weight and Height(Mean ± SD)			
Group	**Mean age**	**Mean weight (in Kg)**	**Mean height (in cm)**
I	51.50±8.94	56.30±4.26	157.80±4.27
II	50.55±13.42	63.25±5.58	162.70±5.08
III	48.40±9.09	61.85±5.19	160.85±5.08
Between the group comparison one way ANOVA	F=0.699	F=9.09	F=2.071
	p = 0.511 (NS)	p = 0.281 (NS)	p =.410 (NS)

Table No. 3A: The mean, standard deviation and statistical comparison of Visual Analogue Scale scores between the groups at successive visits

Visual Analogue Scale (Mean ï SD)				
Group	**B.T.**	**A.T. (1st sitting)**	**A.T. (2n sitting)**	**A.T. (3rd sitting)**
I	5.95±0.82	2.75±0.79	2.25±0.70	1.20±0.61
II	5.85±0.87	4.65±0.98	4.05±0.88	3.20±0.83
III	6.30±0.65	4.60±1.14	3.40±0.88	1.70±0.47
Between the group comparison one way ANOVA	F=1.40	F=10.75	F=14.62	F=24.62
	p =.226	p =.000	p =.000	p =.000
Remarks	(NS)	(HS.)	(HS.)	(HS.)
Post-Hoc test Significant Pairs p<0.05		(I,II)(I,III)	(I,II)(I,III)	(I,II)(I,III)

Table No. 3B: The statistical comparison of difference in mean value of Visual Analogue Scale score before and after treatment within the groups:

Visual Analogue Scale within the group comparison paired ' t ' test			
Group	**B.T. vs A.T. (1st sitting)**	**B.T. vs A.T. (2nd sitting)**	**B.T. vs A.T. (3rd sitting)**
I	3.20±1.05 t= 11.54 p=.000(HS.)	3.70±1.03 t= 12.83 p=.000(HS.)	4.75±1.02 t= 21.83 p=.000(HS.)
II	1.20±0.52 t= 16.25 p=.000(HS.)	1.80±0.83 t= 9.65 p=.000(HS.)	2.65±0.67 t= 11.66 p=.000(HS.)
III	1.70±1.17 t= 16.47 p=.000(HS.)	2.90±0.96 t= 21.39 p=.000(HS.)	4.60±0.75 t= 21.28 p=.000(HS.)

Table No. 4A: The mean, standard deviation and statistical comparison of Karnofsky's scale scores between the groups at successive visits:

Karnofsky's Performance Scale (Mean ï SD)				
Group	**B.T.**	**A.T (1st sitting)**	**A.T (2n sitting)**	**A.T (3rd sitting)**
I	0.95±0.39	0.40±0.50	0.20±0.41	0.00±0.00
II	0.65±0.48	0.25±0.44	0.00±0.00	0.00±0.00
III	0.70±0.57	0.30±0.47	0.05±0.22	0.00±0.00
Between the group comparison One way ANOVA	F=9.69	F=4.35	F=7.77	F=.000
	p=.026	p=.375	p =.017	p =1.00
Significant pairs (p<0.05)	(I,II)			
Remarks	(S.)	(NS)	(S.)	(NS)

Table No. 4B: The statistical comparison of difference in mean value of Karnofsky's scale before and after therapy within the groups:

Karnofsky's Performance Scale within the group comparison Wilcoxon Signed Rank Z test			
Group	**B.T. vs A.T. (1st sitting)**	**B.T. vs A.T. (2nd sitting)**	**B.T. vs A.T. (3rd sitting)**
I	0.55±0.55 Z= 1.317 p=.001(S)	0.75±0.44 Z= 2.114 p=.007(S)	0.95±0.39 Z= 4.24 p=.000(HS)
II	0.40±0.503 Z= 1.82 p=.005(S)	0.65±0.48 Z= 3.106 p=.000(HS.)	0.65±0.48 Z= 3.106 p=.000(HS.)
III	0.40±0.40 Z= 1.82 p=.005(S)	0.65±0.48 Z= 3.616 p=.000(HS.)	0.70±0.57 Z= 3.100 p=.000(HS.)

Table No. 5A: The mean, standard deviation and statistical comparison of Pricking Sensation between the groups at successive visits:

Pricking Sensation (Mean ï SD)				
Group	**B.T**	**A.T (1st sitting)**	**A.T (2nsitting)**	**A.T (3rdsitting)**
I	0.75±0.12	0.15±0.36	0.00±0.00	0.00±0.00
II	0.25±0.44	0.10±0.30	0.00±0.00	0.00±0.00
III	0.30±0.47	0.15±0.36	0.00±0.00	0.00±0.00
Between the group comparison One way ANOVA	F=12.115 p=.037	F=1.242 p=.986	F=1.234 p =.545	F=.00 p =.00
Remarks	(S)	(NS)	(NS)	(HS.)

Table No. 5B: The statistical comparison of difference in mean values of Pricking Sensation before and after treatment within the groups:

Pricking Sensation within the group comparison Wilcoxon Signed Rank Z test			
Group	**B.T. vs A.T. (1st sitting)**	**B.T. vs A.T. (2nd sitting)**	**B.T. vs A.T. (3rd sitting)**
I	0.60±0.50 Z= 4.46 p=.001 (S)	0.75±0.35 Z= 3.63 p=.000 (HS.)	0.75±0.55 Z= 1.63 p=.000 (HS.)
II	0.15±0.36 Z= 2.73 p=.083 (NS)	0.25±0.44 Z= 3.236 p=.025 (S)	0.25±0.44 Z= 3.236 p=.025 (S)
III	0.15±0.36 Z= 2.732 p=.083 (NS)	0.30±0.47 Z= 3.44 p=.014(S)	0.30±0.470 Z= 3.44 p=.014(S)

Table No. 6A: The mean, standard deviation and statistical comparison of Radiating Pain between the groups:

Radiation of Pain (Mean ï SD)				
Group	**B.T**	**A.T (1st sitting)**	**A.T (2nsitting)**	**A.T (3rdsitting)**
I	2.55±1.35	1.20±1.19	0.45±0.68	0.10±0.44
II	1.30±1.08	0.90±0.78	0.40±0.59	0.05±0.22
III	1.35±1.63	0.90±1.11	0.60±0.94	0.10±0.30
Between the group comparison One way ANOVA	F=11.85 p=.054	F=2.224 p=.943	F=3.219 p =.818	F=4.971 p =.024
Remarks	(NS)	(NS)	(NS)	(S)

Table No. 6B: The statistical comparison of difference in mean value of Radiating Pain before and after treatment within the groups:

Radiation of Pain within the group comparison Wilcoxon Signed Rank Ztest			
Group	B.T. vs A.T. (1st sitting)	B.T. vs A.T. (2nd sitting)	B.T. vs A.T.(3rd sitting)
I	1.35±1.13 Z= 2.48 p=.000 (HS.)	2.10±1.33 Z= 2.866 p=.000 (HS.)	2.45±1.39 Z= 2.84 p=.000(HS.)
II	0.40±0.75 Z= 1.33 p=.020 (S)	0.90±0.91 Z= 2.140 p=.002 (S)	1.25±1.07 Z= 2.345 p=.001 (S)
III	0.45±0.75 Z= 1.310 p=.021 (S)	0.75±0.91 Z= 1.877 p=.004 (S)	1.25±1.51 Z= 1.956 p=.003 (S)

Table No. 7A: The mean, standard deviation and statistical comparison of Tenderness between the groups at successive visits:

Tenderness (Mean ï SD)				
Group	**B.T**	**A.T (1st sitting)**	**A.T (2nsitting)**	**A.T (3rdsitting)**
I	0.35±0.48	0.00±0.00	0.005±0.22	0.00±0.00
II	0.20±0.41	0.15±0.36	0.00±0.00	0.00±0.00
III	0.15±0.36	0.15±0.36	0.05±0.22	0.00±0.00
Between the group comparison One way ANOVA	F=3.064 p=.408	F=4.35 p=.195	F=5.16 p =.290	F=8.08 p =.073
Remarks	(NS)	(NS)	(NS)	(NS)

Table No. 7B: The statistical comparison of difference in mean value of Tenderness before and after treatment within the groups:

Tenderness within the group comparison Wilcoxon Signed Rank Z test			
Group	B.T. vs A.T. (1st sitting)	B.T. vs A.T. (2nd sitting)	B.T. vs A.T. (3rd sitting)
I	0.35±0.48 Z= 2.646 p=.008 (S)	0.30±0.47 Z= 2.44 p=.014 (S)	0.35±0.48 Z= 2.646 p=.008 (S)
II	0.05±0.39 Z= .577 p=.564(NS)	0.20±0.41 Z= 2.00 p=.004(S)	0.20±0.41 Z= 2.00 p=.046 (S)
III	0.00±0.32 Z= .000 p=1.00(NS)	0.10±0.31 Z= 1.414 p=.157(NS)	0.15±0.36 Z= 1.732 p=.083(NS)

Conclusion: On the basis of the above observations made on patients treated by Agnikarma chikitsa with Panchdhatu, Swarna and Lauh shalaka this can be concluded-

- The trial procedure Agnikarma with Panchdhatu, Swarna and Lauh shalaka has Vedanahar (analgesic) and Shothahar (anti-inflammatory) properties.
- Agnikarma with Panchdhatu, Swarna and Lauh shalaka is a simple modality of treatment with minimum complication, which can be easily taken care of.
- Agnikarma Chikitsa with Panchdhatu, Swarna and Lauh shalaka does not produce any significant side effects.
- Agnikarma Chikitsa with Panchdhatu, Swarna and Lauh shalaka does not alter normal physiology. No significant changes were observed in mean blood pressure, pulse rate, respiratory rate and oxygen saturation during the whole course of the clinical study.
- The Agnikarma Chikitsa with Panchdhatu shalaka is slightly more effective than Swarna and Lauh shalaka as Vedanahar analgesic.
- Number of sittings of Agnikarma depends upon the chronicity and severity of disease..
- Further, a more detailed study on a large number of samples is required to evaluate biochemical and neurological changes during and after procedure to unfold other properties of Agnikarma.

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Mechanisms of Pain and It's Pathway

***Dr.AK Mishra**Dr.PS.Shindhe**

Abstract: Pain can develop after mechanical, thermal and chemical injury, when deleterious changes occur in injured neurons and along Nociceptive and descending modulatory pathways in the central nervous system. The myriad, substans P, glutamate neurotransmitters and other substances involved in the development and maintenance of pain also play a part in other neurobiological disorders. This might partly explain the high comorbidity rates for chronic pain, sleep disorders, and psychological conditions such as depression, and why drugs that are effective for one condition may benefit others. Neuropathic pain can be distinguished from non-neuropathic pain by two factors. Firstly, in neuropathic pain there is no transduction (conversion of a nociceptive stimulus into an electrical impulse). Secondly, the prognosis is worse: injury to major nerves is more likely than injury to non-nervous tissue to result in chronic pain. In addition, neuropathic pain tends to be more refractory than non-neuropathic pain to conventional analgesics, such as nonsteroidal anti-inflammatory drugs and opioids. However, because of the considerable overlap between neuropathic and nociceptive pain in terms of mechanisms and treatment modalities, it might be more constructive to view these entities as different points on the same continuum. This review focuses on the mechanisms of pain and pathway, with special emphasis on clinical implications.

Keywords: Nociceptor, Neuropathic pain, Pain, spinothalamic tract .

Introduction: Perception of Pain is a vital function of the nervous system.Providing the body with a warning of potential or actual injury.It is both a sensory and emotional experience. Affected by psychological factors such as past experiences , beliefs about pain, fear or anxiety. Define as

"An unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage"

IASP

Described in many ways like pricking, shooting, burning , Throbbing , dull ,cutting etc.Pain is a vital function of the nervous system in providing the body with aWarning of potential or actual injury. It is both a sensory and emotional

Experience, affected by psychological factors such as past experiences,beliefs about pain, fear or anxiety.

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This article provides an overview of the physiological mechanisms of pain and the important pain pathways. We will discuss pain receptors, transmission of pain signals to the spinal cord and pain pathways within the spinal cord. Wewill also look at how pain can be modulated at different levels along thepathway. Finally we discuss different types of pain including visceral and neuropathic pain.

Aims and Objectives

- To review mechanism of pain and its pathway in modern view.

Nociceptors : Nociceptors are the specialised sensory receptors responsible for thedetection of noxious (unpleasant) stimuli, transforming the stimuli intoelectrical signals, which are then conducted to the central nervous system. They are the free nerve endings of primary afferent A delta and C fibres. Distributed throughout the body (skin, viscera, muscles, joints, meninges) they can be stimulated by mechanical, thermal or chemical stimuli. Inflammatory mediators (egbradykinin, serotonin, prostaglandins, cytokines, and H+) are released from damaged tissue and can stimulate nociceptors directly. They can also act to reduce the activation threshold of nociceptors so that the stimulation required to cause activation is less. This process is called primary sensitization

Primary afferent fibers: In addition to the A delta and C fibers that carry noxious sensory information, there are primary afferent A delta fibers that carry non-noxious stimuli. Each of these fiber types possesses different characteristics that allow the transmission of particular types of sensory information.

• *A* beta *fibers* are highly myelinated and of large diameter, thereforeallowing rapid signal conduction. They have a low activationthreshold and usually respond to light touch and transmit nonnoxiousstimuli.

• *A* delta *fibres*are lightly myelinated and smaller diameter, and henceconduct more slowly than Aβ fibres. They respond to mechanicaland thermal stimuli. They carry rapid, sharp pain and are responsible for the initial reflex response to acute pain.

• *C fibres*are unmyelinated and are also the smallest type of primaryafferent fibre. Hence they demonstrate the slowest conduction. Cfibres are polymodal, responding to chemical, mechanical andthermal stimuli. C fibre activation leads to slow, burning pain.

	Aβ fibres	Aδ fibres	C fibres
Diameter	Large	Small 2-5μm	Smallest <2μm
Myelination	Highly	Thinly	Unmyelinated
Conduction velocity	> 40 ms-1	5-15ms-1	< 2ms-1
Receptor activation thresholds	Low	High and low	High
Sensation on stimulation	Light touch, non-noxious	Rapid, sharp, localised pain	Slow, diffuse, dull pain

Table 1 Characteristics of primary afferent fibres

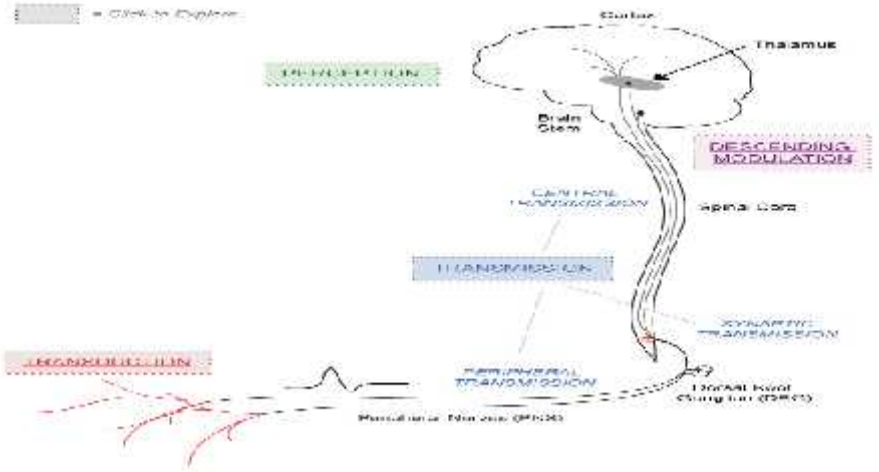
CLASSIFICATION OF PAIN

1. Nociceptive Pain
2. Neuropathic Pain
3. Psychological (Phantom Pain)

NOCICEPTIVE PAIN-Nociceptive stimulus - an actually or potentially tissue damaging event transduced and encoded by nociceptors. Nociceptor is a sensory receptor that responds to potentially damaging stimuli by sending nerve signals to the spinal cord and brain.

PHASE-

- Transduction
- Transmission
- Perception
- Modulation



Dorsal horn of the spinal cord

Adelta and C fibres synapse with secondary afferent neurones in the dorsal hornof the spinal cord. The dorsal horn can be divided histologically into ten layerscalled Rexedlaminae. Adelta and C fibres transmit information to nociceptivespecificneurones in Rexed lamina I and II, in addition to projections to otherlaminae. Primary afferent terminals release a number of excitatoryneurotransmitters including glutamate and substance P.Complex interactions occur in the dorsal horn between afferent neurones,interneurones and descending modulatory pathways (see below). Theseinteractions determine activity of the secondary afferent neurones. Glycineand gamma-aminobutyric acid (GABA) are important neurotransmitters actingat inhibitory interneurones.

PATHWAY-

Ascending tracts in the spinal cord

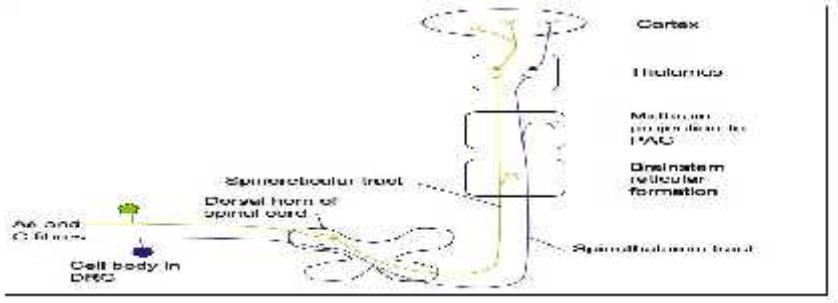
There are two main pathways that carry nociceptive signals to higher centersin the brain.

• **The *spinothalamic tract*:**

Secondary afferent neurones decussate within a few segments of the level of entry into the spinal cord and ascend in the contralateral spinothalamic tract to nuclei within the thalamus. Third order neurones then ascend to terminate in the somatosensory cortex. There are also projections to the periaqueductal grey matter (PAG).The spinothalamic tract transmits signals that are important for painlocalization.

• **The *spinoreticular tract*:**

Fibers also decussate and ascend the contralateral cord to reach the brainstem reticular formation, before projecting to the thalamus and hypothalamus. There are many further projections to the cortex. This pathway is involved in the emotional aspects of pain.



Neuropathic pain

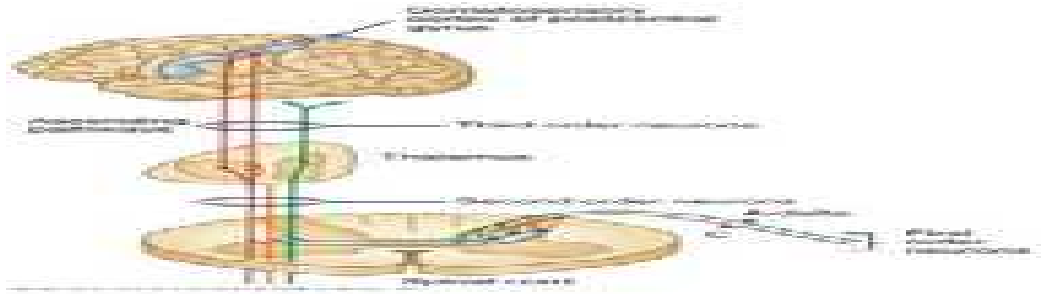
Neuropathic pain is caused by damage to nerves in the central or peripheralnervous system. Damage can be due a number of mechanisms includingtrauma or surgery, diabetes mellitus, chemotherapy, radiotherapy, ischaemia,infection or malignancy.Neuropathic pain has some different characteristics to nociceptive pain. Painis more likely to be spontaneous and is described as burning or 'like anelectric shock'. Pain may be experienced in response to a stimulus that doesnot usually cause pain (allodynia), or there may be a heightened response to a stimulus that is usually painful (hyperalgesia)

Visceral pain

Visceral pain is pain arising from the internal organs. The viscera are largelyinnervated by C fibers. Visceral pain is typically diffuse and poorly localized,often described as deep, dull or dragging. It can be associated with autonomicchanges such as nausea, vomiting, and changes in heart rate or blood pressure. It can also evoke strong emotional responses. In contrast to somatic pain, which is felt due to stimuli such as burning or crushing, visceral pain is triggered by smooth muscle distension or contraction, stretching of the capsule surrounding an organ, ischemia and necrosis, or irritation by chemicals produced during inflammatory processes.

Referred pain is pain experienced at a site distant from source of the pain. It is due to the convergence of different afferents on to the same dorsal horn neurons in the spinal cord. For example shoulder pain can be felt due to diaphragmatic irritation that occurs following laparoscopic surgery that can stretch the diaphragm.

ORDERS OF NEURONS:



Conclusion: Pain is both a sensory and emotional experience, and patients past experiences, fears and anxieties can play an important role. Pain transmission is a result of complex peripheral and central processes. These processes can be modulated at different levels and pain perception is a result of the balance between facilitatory and inhibitory interactions.Current areas of interest in pain research include investigating the effect of mood on pain processing in the brain and looking for novel drugs to block channels involved in pain transmission.

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Role of Agni and Ama in the Development of Pratishyay

***Dr. RK Singh **Agrawal Monika ***Dr.PS Byadgi**

ABSTRACT: Ama refers to raw, unripe, unprocessed or improperly digested condition of food substances develop due to mandagni (hypo functioning of Agni). Agni is responsible for longevity, strength and complexion. Acharya Vagbhatta described Ama concept in detail. It is very much necessary to understand Ama for the purpose of diagnosis of disease and treatment of disease. Ama is an antigen and induces immunological reaction inside the body in a susceptible individual. Excessively vitiated doshas (autoimmune) favors the causation of Ama to a greater extent. Abnormal agni is responsible for genesis of almost all diseases. Especially Mandagni is a causative factor for the production of Ama, which in turn favors development of Pratishyay.
Key words: Agni, ama, pratishyay

INTRODUCTION: Pratishyay manifests due to irritation and inflammation of the mucous membrane inside the nose by kapha and vata. Continuous discharge from nose is known as pratishyaya. Pratishyaya is challenging disease among all respiratory tract diseases because of its recurrence and chronic nature. It attacks the individual recurrently and can precipitate instantaneously in susceptible individuals due to exposure to etiological and risk factors. Recurrence of the disease occurs when the vitiated doshas have not been removed completely as a result doshas resides in their latent stage and gives rise to disease when they come in contact with triggering factors. In certain individuals it can remain as a chronic disease. It is characterized by nasal blockage, nasal discharge, headache, continuous sneezing, heaviness in head etc. If it is not treated in time it leads to severe disorders like dushta pratishyaya, asthama, and other complications of respiratory tract. Nose is the gateway for head and pratishyay manifest due amalgamation of dosha and dushya in nose. It can be correlated to rhinitis, sinusitis and common cold mentioned in modern medicine. *Dushta Pratishyaya* is the chronic stage/advanced condition of *Pratishyaya*, which manifest due to neglect or improper management of *Pratishyaya*. Chronic sinusitis can be correlated with *Dushta Pratishyaya* on the basis of the signs, symptoms, complications, and prognosis[5]. Pratishyay manifests due to involvement of kapha predominant doshas associated with abnormal agni causing development of ama. The entire range of digestive and metabolic activity of the body takes place with the help of biological fire of the body called **Agni**; Ayurveda conceives the following components of **Agni**, which functions at different levels of digestion, metabolism and absorption activity of

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body. **Jatharagni / Pachakagni (one) -** located in G.I.T., performs digestion of food. **Pachakamsa (Seven)** - generated in GIT as part of pachakagni but has function in dhatus. **Dhatwagni (Seven)** –located in respective dhatus, responsible for tissue Metabolism. **Bhutagni (Five)** – located in oral cavity and tissues responsible for finer molecular metabolism and assimilation. The Jatharagni is considered as the master agni and is claimed to govern the function of all other agnis besides its own function. The above 13 types of **agnis** in the body are responsible for digestion of food and metabolism at different levels when agni becomes weak a number of unwanted unripe byproducts of digestions and metabolism start forming and accumulating in the body at different levels. from gross to molecular level, from local GIT level to systemic levels over called ama and acts as a toxin and antigenic material. **Ama** is the classical term used in Ayurveda to designate a material which is essentially unripe, undigested or unmetabolized formed as a consequence sequels of malfunctioning of agni if allowed to prevail block the micro channels and precipitate antigenic reaction in the body. Ayurveda conceives the idea of allergy and intolerance caused by a variety of unwanted endogenous or exogenous material (Amavisa and dusivisa)[1&4].

Objectives- to collect the material from the texts and article related to ama and agni and its relation in the development in the pratishyay

Materials and Methods- an effort has been made to understand the concept of agni and ama in the development of pratishyay.

Schematic illustration of pathogenesis of Pratishyay indicating role of agni and ama[5]

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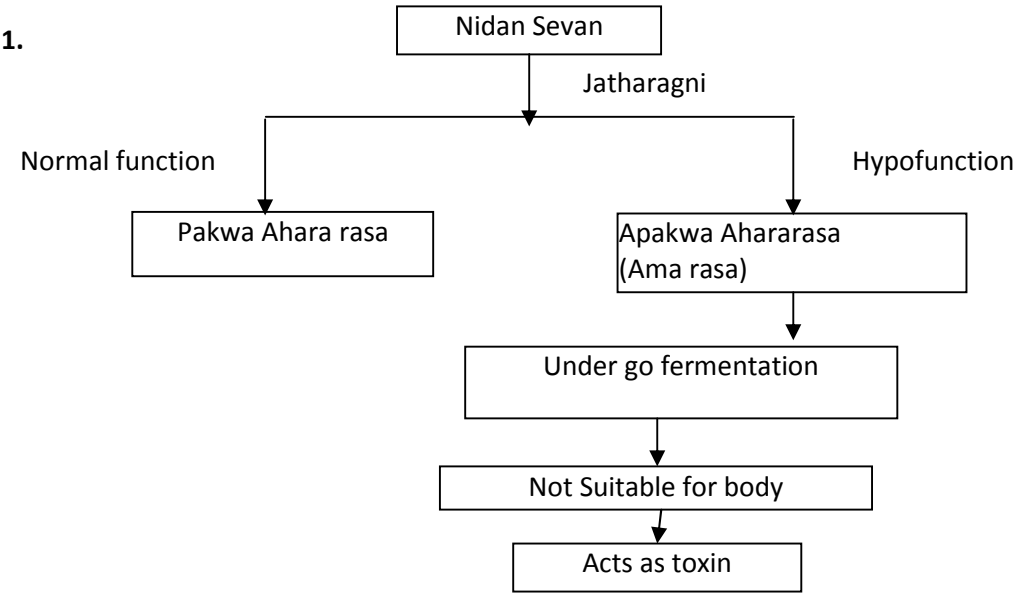

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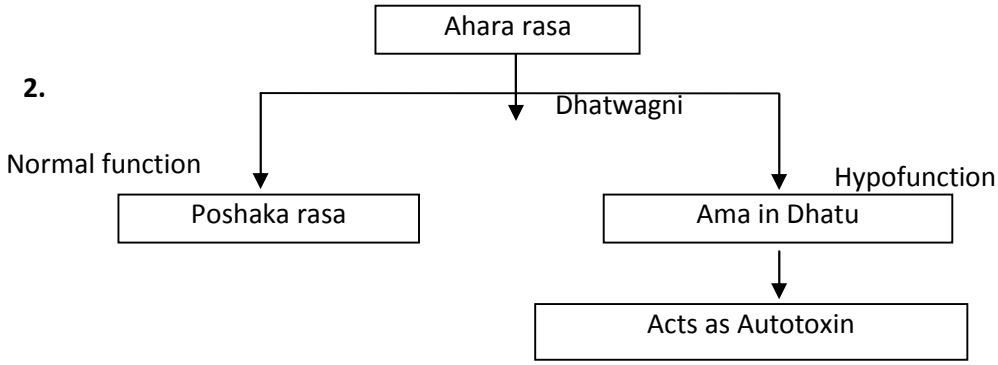

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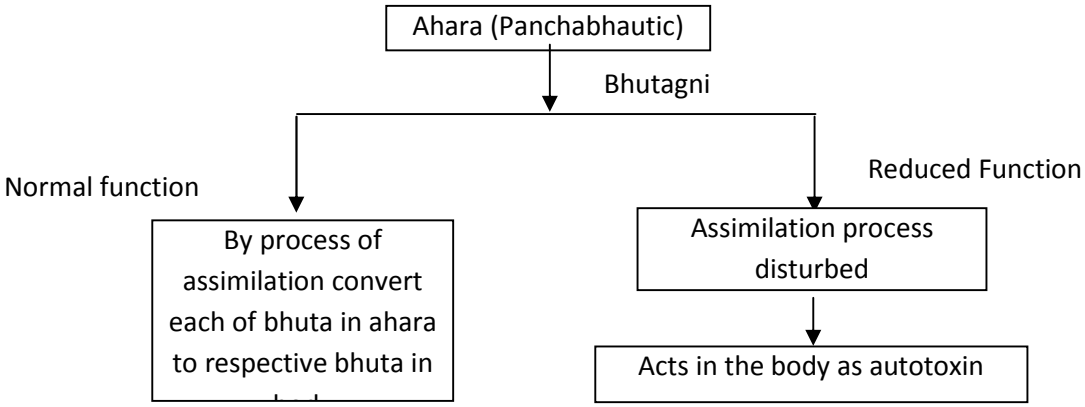

AGNI: Jatharagni is the principal among all types of agnis because function of bhutagni and dhatvagni are controlled by this. Provocation or decrease of jatharagni results in aggravation or diminution of bhutagni and dhatvagni. One should protect jatharagni by suitable healthy dietetics and activities since longevity and strength depends on regular state of agni. One, who consumes unwholesome diet due to greed, succumbs to disease caused by the vitiation of grahani. Every *Dhatvagni*(bio-energy fire) present in each *Dhatu* synthesize and transform the essential *Rasa Dhatu* requisite for that particular *Dhatu* or cell from the basic nutrients present in the *Anna Rasa* or essence of the diet that we eat. Each *Dhatvagni* has got a specialty to synthesize and transform the constituents suitable to its particular *Dhatu* and it is a sort of selective action. It has been widely accepted that the seven *dhatus* that are a support of the body contain their own *Agni*, and by their own *Agni* they digest and transform the materials supplied to them to make the substances identical to them for absorption and nourishment. Body is composed of the five *mahabhutas*(five basic elements). Obviously, each cell (*dhatu paramanu*) and tissues consists of five *Bhutagni* also. Food we eat also consist of the five basic elements with their respective *Agni*

or bio-energies. As a consequence, they are completely similar with respect to the five basic elements with their *Bhutagni* in our body cells and tissues. It is mentioned that the five *Bhutagni* digest their own part of the constituent present in the food resources. After the digestion of food by the *Bhutagni*, digested materials containing the elements and qualities similar to each *bhutas* nourish their own particular bhautika elements of the body. These *Bhutagnis* act after getting stimulation from the the *Jatharagni* in the oral cavity causes disintegration of food substances. In the modern physiological perspective, the action of *Jatharagni* can be equated with the digestion in the stomach and duodenum, and the action of the *Bhutagni* can be equated with the functions of salivary juices and other substances which assist digestion mechanism in oral cavity along with conversion of digested materials in the liver as well it works for the formation each dhatu[1, 3 &4].In case of Pratishyay agni becomes weak due to deranged kapha.

AMA: Ama means improper or partially digested food substances inside the body. Due to hypofunctioning of Kayagni, the improper adhya ahara dhatu (Rasa dhatu) formed in amashaya is known as ama. The substances, which remain undigested, disintegrated, foul smelling, excessive in quantity, slimy in nature, and causes stiffness of the whole body is the characteristic properties of ama. Ama in the form of sukshma Amarasa comes in systemic circulation which is not suitable for either dhatwagni paka or bhutagni paka. Because of its apakwa nature it becomes vijatiya dravya that works as antigen due to which antibodies are produced. Further it vitiates the doshas present in the body to cause diffirent kind of disorders depending on severity and site of its location. Ama is very hazardous substance being macromolecular in size causes obstruction to the micro channels initially ama manifests at gastrointestinal level and later in advanced condition it reaches the systemic circulation by the help of vata. In case of Pratishyay ama reaches the pranavaha srotas and manifest certain reactions in that place leading to development of inflammatory process in srotas causing discharge from the nose of different colours and smell. Acharya Chakrapani while commenting on Grahani Chikitsa has mentioned the existence of ama at different levels. Due to the consumption of nidan, which are capable of exacerbating dosha and brining mildness in agni's, after this whatever is again eaten or drunk by ignoramus person, the same becomes improperly digested, this transformed into sourness in amasaya is called amavisa[1&2].

CONCLUSION: Pratishyay manifests due to involvement of kapha predominant doshas associated with abnormal agni causing development of ama. Thus the differences between the presences of *ama* characters more dominantly or less dominantly is due to variation of various proportions of *doshic* influence in the disease manifestation along with active role of *ama in different varieties of pratishyay*. More dominancy of *ama* characters causes more severely appearances of *pratishyay* symptoms. *Dushta Pratishyaya* is the chronic stage/advanced condition of *Pratishyaya*, which manifest due to neglect or improper management of *Pratishyaya*. Chronic sinusitis can be correlated with *Dushta Pratishyaya* on the basis of the signs, symptoms, complications, and prognosis. In this condition jatharagni,dhatwagni and bhutagni involves simultaneously as a result disease becomes incurable due to development of ama in all cells and tissues.

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Comorbidites with Psoriasis – A Review

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ABSTRACT: The main objectives of this review article is to discuss the most common comorbid conditions associated with psoriasis,to discuss criteria for the appropriate referral of patients with suspected comorbidity, to provide information on how therapies for psoriasis may modify the course of associated diseases and to provide information concerning treatments prescribed for associated diseases that may have an impact on the course of psoriasis.The authentic subject material has been reviewed fromdifferent modern medical literature. Selected articles from dermatologic and psychiatric literature were reviewed and used as the basis for the discussion on associated comorbid conditions.The subject material has also been searched on internet.Severalepidemiological and clinical studies have confirmed that psoriasis is associated with many comorbid conditions which include, arthritis, cardio-metabolic disorders including myocardial infarction, stroke, diabetes, obesity, dyslipidemia and non-alcoholic fatty liver disease. These comorbidities confer a higher mortality rate.The presence of any co-morbid diseases worsen the psoriasis and also associated with an increase in concomitant medication.Systemic treatment of psoriasis with certain drugs may impact the co-morbid conditions. Adequate knowledge on comorbidities helps in choosing the appropriate therapy as well as timely intervention.

KEYWORDS: Psoriasis, Comorbidities, Comorbid conditions, Metabolicsyndrome, Psoriatic arthritis

INTRODUCTION: Psoriasis is a systemic chronic, relapsing inflammatory skin disorder with worldwide distribution, affecting 1–3% of the world population.Prevalence varies according to race, geographic location and environmental factors[1-3].Although traditionally psoriasis has been considered a dermatologic disease, contemporary medical literature is accumulating to support the assertion that psoriasis is actually amultisystem disorder[4].The disease has wide clinical spectra that range from epidermal (scaly) and vascular (thickened, erythematous) involvements of the skin to the malignant form known as generalized erythrodermia. Its most common form named psoriasis vulgaris or plaque-type psoriasis. Psoriasis recently received a lot of scientific and medical attention in great part, because of major advances in our understanding of the disease and the consequent development of new treatments that are dramatically effective in many patients.Key areas of science in which advances have been made are genetics and immunology. Psoriasis is one of the most common dermatologic disorders, clinically characterized by erythematous sharply demarcated papules and rounded plaques covered by silvery micaceous scales.Psoriatic plaques induce pain and pruritus, generating discomfort and persistent insomnia.

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Many studies suggest that patients of psoriasis tend to have concurrent illnesses (behavioral and systemic), termed as comorbidities,which include psoriatic arthritis, cardiovascular disease, nonalcoholic fatty liver disease, inflammatory bowel disease, lymphoma, skin cancer, anxiety and depression. Cardiovascular disease is approached through the study of its major risk factors which include obesity, diabetes mellitus, hypertension and dyslipidemia. Associations between psoriasis and other risk factors for heart disease, such as smoking and alcohol consumption are also included in this category. The best-known noncutaneous condition associated with psoriasis is joint disease, mostly expressed as Psoriatic arthritis (PsA).The clinical practice guideline on the management of comorbidities with psoriasisfocuses primarily on the diseases most often found in patients with psoriasis [5-8]. Quality of life decreases considerably because it is a physically disfiguring illness that disrupts social life, induces constant psychological stress, lowered self-esteem and feelings of being socially ostracized. Common among many patients are the use of tranquilizers, sleeping pills, antidepressants, consumption of alcohol and cigarette smoking. Pruritus is an important symptom in psoriasis vulgaris which may be severe and seriously affect the quality of life[9-12]. The coexistence of any disease with psoriasis influence the choice of therapy for psoriasis and the systemic treatment of psoriasis with certain drugs may impact them negatively.The presence of these co-morbidities haveimportant clinical implications for the therapy of patients with psoriasis and an effective prevention of cardiovascular events. Therefore, dermatologists should be aware of these associations as they may be in a position to detect them earlier, thus, allowing early intervention that may improve the overall quality of life.These comorbidities may share a common genetic background with psoriasis, but it is also possible that psoriatic cytokines can mediate some metabolic effects in particular insulin resistance [13].

COMORBIDITES WITH PSORIASIS: Recently, comorbidities have been re-discovered in dermatology. The basis for the observations of comorbid conditions began from the hypothesis that psoriasis is characterized by increased T-cell activation leading to production of various cytokines causing inflammation in the skin as well as other organ targets.The association of comorbidities and dermatosesis complex and multifactorialmaking it difficult to demonstrate direct relationships. Although numerous associations between skin diseases and other conditions have been reported, only a few are well documented. Lifestyle factors, impaired health-related quality of life and depression, therapeutic interventions and several biases may confound the relationship between skin diseases and comorbidities. Also, several biases, such as detection bias (ie, patients with a skin disease are more likely to be diagnosed with another disease while visiting their physician for their dermatosis) may affect observational study results.

Pathogenesis of psoriasis and comorbidities: Common inflammatory pathways and genetic predispositions for specific patterns in the immune response may play a vital role in the evolution of associated conditions, e.g. human leucocyte antigen HLA Cw6 in psoriasis and PsA, tumor necrosis factor alpha (TNF-α) in psoriasis and PsA, and interleukin (IL)-12/23 in psoriasis and Crohn's disease (CD).TNF-α plays a central role in the pathogenesis of psoriasis. It plays a critical role in activation of innate and acquired immune responses leading to chronic

inflammation, tissue damage and keratinocyte proliferation. Several regions on chromosomes 16, 6, 4, and 3 have been identified where genetic markers are linked to both psoriasis andCD [14]. Newer models on pathogenesis of psoriasis explainthe role of skin barrier function, T-helper 17 (Th17) pathway, innate immunity, signaling pathways, Th2 pathway and adaptive immunity involving CD8 T cells. These studies illustrate the importance of both the keratinocytes and the immune system for the pathophysiology of psoriasis.

Psoriatic arthritis (PsA): Psoriatic arthritis is a well-known comorbidity associated with psoriasis.Relationship between psoriasis and arthritis first noticed by Louis Aliberti in 1818 [15].Psoriatic systemic disease encompassing skin, joint and nail involvement is an autoimmune process as evidenced from animal models, the HLA-Cw6 association in human, T cells infiltration in lesional skin and the response to T cell targeted therapies. Psoriatic arthritis is inflammatory in nature with the presentation of pain, stiffness and swelling of affected joints.Prolonged morning stiffness, lasting more than 60 minutes is a common complaint and results from inflammatory involvement of entheses, the point at which tendons or ligaments insert to bone. It tends to improve throughout the day.Psoriatic arthritis usually develops between 30-50 years of age. The most commonly affected joints are wrist, knees, ankles, lower back, and neck joints.Though patterns of presentation are not helpful to identify PsA, involvement of distal inter-phalangeal joint (DIP) in asymmetric fashion may be the most readily recognizable because it is unique to PsA[16]. When compared with RA, in PsA all joints of one digit tend to be involved while sparing other digits, whereas in RA the same joint is involved in all the digits. Thesynovitis is the primary lesion in rheumatoid arthritis (RA), whereas synovitis along with enthesitisis characterstic to PsA[17]. Since the nails and joints are associated with inflammation at points of ligament or tendon insertion (i.e., enthesitis), it has been postulated that response to tissue stressing of the integrated nail-joint apparatus, rather than autoimmunity, driving the inflammatory process with a relative differential involvement of adaptive and innate immunity in the psoriatic disease [18].It affects both sexes equally and can develop at any age, even in childhood, but in most cases onset occurs between the ages of 30 and 50 years [19]. PsA is characterized by synovitis, enthesitis, dactylitis and spondylitis, usually manifesting in a person with skin and nail psoriasis. Our understanding about the PsA disease state, its genetics, pathophysiology and comorbidities as well as our ability to assess and treat the disease has advanced as a result of significant collaborative efforts by rheumatologists and dermatologists [20]. A number of different forms of PsA have been described, the most common being asymmetrical oligoarticulararthritis affecting up to 3 joints in the limbs. Other forms of presentation are as follows:

- Symmetricpolyarticular arthritis with a course similar to that of rheumatoid arthritis.
- Distal interphalangeal arthritis primarily affecting the hands.
- Arthritismutilans, a less common form that is extremely destructive and deforming and
- Arthritisaffecting the spinal bones and the pelvic or sacroiliac joints, with a course similar to that of ankylosing spondylitis.

Since patients present with an overlapping combination of the symptoms of these different clinical forms, the clinical spectrum is very wide and presentation differs in each case. Oligoarticular forms of the disease can progress to polyarticular disease in later stages.

Modified from: The number of actively inflamed joints as measure of disease activity and the number of clinically deformed joints as measure of damage were significantly related to the "Health Assessment Questionnaire" (HAQ) score,also useful for defining PsA types. Furthermore, interaction terms for illness duration with the number of actively inflamed joints were statistically significant with or without inclusion of the erythrocyte sedimentation rate and morning stiffness in the model. The influence of disease activity on HAQ scores declines with increased disease duration [22-23].

Cardiovascular diseases: Psoriasis may be a risk factor for development of coronary artery calcification (CAC).Cardiovascular disease is more prevalent in those with moderate to severe psoriasis than in the general population. A large retrospective cohort study of the UK General Practice Research Database, including more than 130,000 psoriasispatients ages 20 to 90 revealed a greater thannormal incidence of myocardial infarction in patients with psoriasis, reinforcing a connection between psoriasis and heart disease[24].Psoriasis appears as anindependent risk factor for heart attack. Even as severe psoriasis was shown to be an independent risk factor for death due to cardiovascular disease.After adjusting for traditional cardiovascular risk factors (age, sex, hyperlipidemia, hypertension, smoking, diabetes etc.), the relative risk (RR) of cardiovascular death associated with severe psoriasis was highest in younger individuals with a RR of cardiovascular death of 2.69 for a 40-year-old, as opposed to 1.92 for a 60-year old[25]. Other cardiovascular risk factors also share common pathogenic mechanisms with psoriasis. In metabolic syndrome, for example, obesity gives rise to a cytokine imbalance when adipocytes trigger excessive secretion of the most deleterious cytokines from the vascular point of view (IL-6, IL-18, TNF-αandleptin) and downregulate the secretion of protective cytokines, such as adiponectin[26]. Some authors suggested that the apolipoprotein E4 variant may have a pathogenic role in psoriasis [27]. The same variant is also associated with certain types of dyslipidemia [28].Arteriosclerosis has many points in common with psoriasis and other inflammatory diseases (eg, rheumatoid arthritis, inflammatory bowel disease and lupus erythematosus).These are related to the immune process and the profile of the mediators and immune cells involved in the pathogenesis of all of these diseases. Inflammatory markers are high at both local and systemic levels. Specifically in psoriasis, the inflammatory process is accompanied by abnormalities in ILs, elevated TNF-α and C-reactive protein playing an important role in the genesis of arteriosclerosis[29-30].

Psychosocial co-morbidities: Psoriasis is not life threatening but is life ruining due to its visibility. Therefore, Psoriasis is associated with a variety of psychological problems, including poor self-esteem, sexual dysfunction, anxiety, depression and suicidal ideation, reported as high as 67% in one study [31]. Since the psychosocial aspect of disease plays an important role in patient's perception of disease severity, quality of life and course of the disease, it is important to include the measures of psychosocial morbidity during assessing the severity of psoriasis.Many

patients experience feelings of social rejection and stigmatization due to the visibility of lesions. The impact on quality of life is accordingly highly variable and is linked to both lesion visibility and age of onset [32].Stigmatization has been shown to be significantly correlated with psychological distress and level of depression [33].
In some studies it is found that the prevalence of depression was significantly higher in patients with psoriasis when compared with general population[34-36].

Metabolic syndrome:

The metabolic syndrome is a constellation of metabolic changes.Metabolic syndrome is a cluster of risk factors. These are associated with increased risk for atherosclerotic cardiovascular disease and type 2 diabetes.National Cholesterol Education Program (ATP III)Criteria for Metabolic Syndrome (2002) is as follows [37].
Three or more of the following criterias:

- Abdominal obesity (waist circumference > 102 cm in men and > 88 cm in women)
- Triglycerides > 150 mg/dL or specific treatment
- HDL-C < 40 mg/dL in men or < 50 mg/dL in women or specific treatment
- BP > 130/85 mm Hg or treatment for hypertension
- Fasting glucose > 100 mg/dL

Recent studies have detected an increased prevalence of metabolic syndrome in patients with psoriasis; irrespective of the definition used.There is increased frequency of the syndrome in psoriasis patients with more severe disease. Most common features of metabolic syndrome among the patients of psoriasis was abdominal obesity followed by hypertriglyceridemia and low level of HDL cholesterol. Women with psoriasis showed a 63% increase risk of future diabetes compared with women without psoriasis[38]. A hospital-based case-control study indicated that metabolic syndrome was significantly more common in psoriasis patients than in controls (30.1% vs 20.6%) [39].
Many studies showed strong relation between obesity and psoriasis. Obesity may be biochemically linked to psoriasis by a common pathophysiology. Both psoriasis and obesity are chronic inflammatory states. Visceral adipocytes secretes multiple cytokines, such as tumor necrosis factor (TNF)-α and adipokines like leptin[40]. Greater concentration of TNF-α in the skin and joints of psoriasis patients in comparison with the skin and joints of unaffected persons also indicates strong relation between obesity and psoriasis[41]. It was observed that a moderate weight loss (i.e. 6% of body weight) increased the responsiveness of obese psoriasis patients to a suboptimal dose of cyclosporine[42]. It was also observed that fixed dose regimen of biologics (eg. Etanercept, adalimumab) may have a compromised efficacy in heavier individuals[43].
Obesity is a relevant risk factor for psoriasis and generally precedes the development of psoriasis. A recent population-based study onpatients with mild to severe psoriasis showed that the risk of obesity was significantly increased in psoriasis patients compared with healthy controls and strongly associated with disease severity[44].

The risk of developing diabetes mellitus in the psoriasis cohort appeared independent of BMI. Specifically, the risk for developing diabetes mellitus in psoriatic patients of normal weight, or BMI less than 25, increased twofold, 2.02 when compared with normal weight patients without psoriasis[45]. The prevalence of diabetes reported in some studies varied widely, with rates of between 2.35% & 37.4% in mild to moderate cases of psoriasis and between 7.5% & 41.9% in the cases withsevere psoriasis[46-48].In the study of a cohort of nursesthe risk of diabetes ranged from an hazard ration (HR) of 2.08 (95% CI, 1.60-2.69; adjusted for age) to an hazard ratio of 1.63 (95% CI, 1.25-2.12; adjusted for age, tobacco use, BMI, alcohol and physical activity)[49]. In a study without taking into account,the severity of psoriasis, the prevalence of hypertension ranged from 8.9% to 44.4% (60% in older patients). When severity was taken into account, the prevalence ranged from 15.1% to 32% in patients with mild psoriasis and from 19% to 40.3% in those with moderate to severe psoriasis. A cohort study carried out in the United States reported a hazard ratio of 1.17 (95% CI, 1.06-1.39) for hypertension adjusted for age, tobacco use, alcohol consumption, BMI and physical activity. Similarly some other studies also established that hypertension is an important comorbidity associated with psoriasis[50-54]. Dyslipidemia is an acquired or genetic disorder affecting the metabolism of lipoproteins which gives rise to an increase in total plasma cholesterol, triglyceride levels or both. Diagnostic Criteria for Dyslipidemia, Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults (ATP III), 2002[55].

LipidsValue (mg/dL) Criteria: Total cholesterol < 200 Desirable 200-239 Borderline high ≥ 240 High LDL-C < 100 Optimal 100-129 Normal/slightly high 130-159 Borderline high 160-189 High ≥ 190 Very high HDL-C < 40 Low ≥ 60 High Triglycerides < 150 Normal 150-159 Borderline high 200-499 High ≥ 500 Very high

Some studies showed that the prevalence of dyslipidemia among patients with psoriasis varied widely, ranging from 6.4% to 50.9% in general (severity unspecified), from 5.2% to 29.9% in patients with mild psoriasis and from 6.0% to 29.9% in patients with severe psoriasis. A significant association was also found between psoriasis and dyslipidemia in some other studies[56-62].

COPD (Chronic Obstructive Pulmonary Disease): Increased prevalence of COPD have been detected in patients of psoriasis. A large population based case controlled study showed that the prevalence of COPD was significantly higher in patients of psoriasis[63].

In a study, risk of COPD was compared between patients with psoriasis and a matched reference cohort. The study included 2096 patients with psoriasis and 8384 randomly selected subjects. After adjusting for socio-demographic characteristics and selected co-morbid medical disorders, results showed a hazards ratio (HR) of 2.22 for COPD in the patients ofmild psoriasis (defined as those just getting topical therapy). While patients in a cohort study with severe psoriasis (defined as those under phototherapy or systemic medication) had an HR of 2.81 for COPD. Analysis stratified by patient age and gender showed an adjusted HR for COPD occurring during the 18-month follow-up period to be 2.19 times higher for patients with psoriasis who were > 50

years old than for the same age group of the comparison cohort. There was no significant difference in patients≤ 50 years old. In male subjects, the adjusted HR of COPD during the follow-up period was 2.38 times greater for those with psoriasis than those without; however, there was no significant difference in the female group. This study places a distinct association among male patients with severe psoriasis, especially those over 50 years of age[64].

Malignancies : The patients with severe psoriasis shows increased rate of malignancies. The pateints of psoriasis under systemic therapies have higher incidence of non-melanoma skin cancerand lymphoproliferative diseases[65-67].

In contrast to the association seen with lymphoma, psoriasis does not appear to increase one's risk for primary skin malignancies. Most published studies show equivalence in rates of both melanoma and non-melanoma skin cancerswhen compared with the general population.

In Caucasian patients treated with more than 250 psoralene plus UVA (PUVA) light treatments, the risk of cutaneous squamous cell carcinoma becomes sub-stantially elevated up to 14-fold [68-69]. This incidence is further increased by nearly 7 times if cyclosporine therapy alsoused in patients with significant prior PUVA exposure, leading to the risk of developing cutaneous squamous cell carcinoma to a staggering 100 times that of the general population[70].The obscuring nature of widespread psoriasis itself can lead to challenges in the skin examination, requiring the clinician to remain vigilant in the dermatologic assessment of the psoriasis patient at each visit. Several studies suggest an association between psoriasis and lymphoma with increasing riskfor those severely affected by psoriasis. A population-based studydemonstrated an increased risk of about 33% for developing any kind of lymphoma. The highest relative risks were observed for cutaneous T-cell lymphoma (adjusted relative risk=4.34 (95% CI 2.89-6.52.)[71-73].

Infections: Several micro-organisms have been associated with inductionor exacerbation of psoriasis. The strongest evidence exists for the induction of guttate psoriasis by a tonsillar Streptococcuspyogenes infection[74].This observation has been confirmed by several other studies and some indicate that streptococcal throat infections can also cause exacerbation of chronic plaque psoriasis[75-76].

NAFLD (Non-alcoholic fatty liver disease): It includes spectrum of conditions ranging from simple fatty liver to non-alcoholic steatohepatitis which can give rise to fibrosis, cirrhosis and eventually hepatocellular-carcinoma.NAFLD is now considered as hepatic manifestation of metabolic syndrome. A study showed that patients with chronic plaque psoriasis also had a greater incidence of NAFLD thanobserved in control patients, 47% versus 28%, respectively.Interestingly, those patients with both psoriasis and NAFLD in tandem were judged to have more severe psoriasis than their counterparts with psoriasis alone as measured by the Psoriasis Area and Severity Index[76-78].

Ocular manifestation: Inflammatory conditions of eyes, especially uveitis and keratoconjunctivitissicca are associated with psoriasis.Uveitis is a prominent feature of spondyloarthropathies. Uveitis associated with psoriasis, undifferentiated spondyloarthropathy and inflammatory bowel disease may be less characteristic in its presentation, but with a higher tendency of the posterior pole involvement[79]. In a study, it is found that keratoconjunctivitissiccais most common ocular finding related to psoriatic arthritis[80].

Management: It is important to keep in mind that the comorbidities and drugs used to treat them have an impact on the choice of anti-psoriatic treatment. In addition, comorbidities often preclude the use of traditional systemic agents. Recent studies have demonstrated that patients with pre-existing comorbidities can be safely and effectively treated with biologic therapy. Furthermore, literature is evolving to suggest that better control of psoriasis might decrease cardiovascular mortality and thus, prolonging life span[81].Therefore, to provide appropriate management of psoriasis from an early stage, it is necessary to diagnose the concomitant disease and to prevent and treat any comorbidity found. Such an integrated approach also serves to ensure that the drugs used to treat associated diseases do not interfere with the management of psoriasis and vice versa. Dermatologists should not just recognize and treat the signs and symptoms of psoriasis but should also screen patients to detect the existence of comorbid conditions such as arthritis, metabolic syndrome and cardiovascular diseases etc.In this way a multidisciplinary approach with co-ordination between dermatologists and other specialists is needed for the management because psoriasis is now recognized as a multisystem disorderdue to its systemic nature of inflammation[82].

DISCUSSION: It is well acknowledged that the co-morbidities associated with psoriasis are responsible for increasing rates of morbidity and mortality[83].Exploring the correlations between psoriasis and comorbid disease states is increasingly essential to elucidating the comprehensive pathophysiology of psoriasis. Psoriasis is typically unpredictable in its course, may vary in severity from one episode or flare to another and often recurs throughout an affected person's life.Clinical presentation can vary significantly from one patient to another.Psoriasis is associated with significant comorbidities and affects patient's quality of life. The relationship between psoriasis and comorbidities is likely linked to the underlying chronic inflammatory nature of psoriasis[84].Many of these conditions also have a similar immunologic pathogenesis[85]. Knowledge about the association between a dermatoses and another disease may increase the understanding of the shared pathogenesis of both diseases.TNF-α levels are markedly increased in skin lesions, synovium and serum of patients with psoriasis and these correlate with the severity of the disease. Decreased levels are associated with clinical resolution[86]. The presence of comorbidities in dermatology is of interest for various reasons. From apreventive perspective, a skin disease can be an early marker of systemic disease, and therefore, help in early identification of the patients who are at risk of having other more life-threatening diseases. An association between a skin disease and comorbidities may influence clinical management i.e.

multidisciplinary approach and treatment options. Ideally, those treatments options should be selected whichimprove both conditions simultaneously. However, comorbidities may also be a contra-indication for therapies indicated for the skin disease or drugs used in the treatment of the comorbidity may interact with the dermatologic therapy. It also appears necessary to adopt treatment regimens that not only provide early clearing of the involved skin but also provide persistently low inflammatory activity. For effective management of psoriasis and related co-morbidities, an integrated approach targeting both cutaneous and systemic inflammation may be beneficial and management strategies to improve overall condition of the patient should be encouraged to reduce the disease burden[87].Treating psoriasis and the associated co-morbid conditions aggressively from the beginning will definitely improve the quality of life.

CONCLUSION: The relationship between psoriasis and associated diseases has drawn particular interest in recent years. The awareness of comorbidities associated with psoriasis has led to a paradigm shift in the understanding of the disease and its management.The present review highlights the association of psoriasis with co-morbid conditions and role of dermatologist, physicians and other allied specialists in the effective management and drawing up an effective treatment plan.This will minimize co-medication, prevent overlap and improve compliance and thus improving the standards of care for the patients of psoriasis.

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Tonsillectomy under local anesthesia: A safe and effective alternative

Dr. Rajesh Kumar*Dr. RK Singh Dr Amber Kesarwani****

Abstract: Tonsillectomy under local anesthesia is a safe and effective alternative to general anesthesia in the healthy, cooperative teenager or adult patient. This retrospective analysis involved 64 local tonsillectomies performed over the last one year from January 2014 to December 2014 in the minor operative room using the local anesthesia under sedation (intravenous). Operation was performed by the residents under training as well as the consultant. Blood loss, morbidity, complication and patients satisfaction were reviewed and compared with tonsillectomy under general anesthesia. The average blood loss was 42 ml in local tonsillectomy group with no cases of post operative hemorrhage, compared with 198 ml in general anesthesia group with 2 cases of post operative hemorrhage. There was one major complication related to post operative antibiotic use in the local anesthesia group, and follow up interview revealed that patients were satisfied with the procedure and would recommend and choose local anesthesia again. We conclude that local tonsillectomy have higher patients acceptance and are associated with minimal morbidity and complication. Moreover, they are cost effective.

Key words: Tonsillectomy, Local anesthesia, cost.

Introduction: Tonsillectomy is presently one of the most frequently performed surgery in the United States (1). Under most circumstances, general anesthesia is safe, and is invariably necessary in children. Adult tonsillectomies, although less frequent, can be performed in local anesthesia or general anesthesia. Unfortunately, most practicing Otolaryngologists have not been effectively trained in the technique of using local anesthesia for tonsillectomy and, therefore, do not feel comfortable with the procedure. In contrast, most surgeons and patients accepts local anesthesia for procedure such as rhinoplasty or rhytidectomy. Most of the Otolaryngologists reject the idea of tonsillectomy under local anesthesia for fear of patient's discomfort and consequent dissatisfaction. Moreover, there is an unnecessary concern over airway and hemostatic control with local tonsillectomy.

In this study we analyzed 64 consecutive tonsillectomies performed under local anesthesia in minor operative room. The safety and effectiveness of local anesthesia in selected healthy, cooperative teenager and adult patients is demonstrated. Surgical technique and patient's satisfaction are noted and compared with a group of adult patients who had undergone tonsillectomy under general anesthesia.

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Materials and Methods: This study comprises of patients presenting in Outpatient Department of Otolaryngology who underwent tonsillectomy under local anesthesia at Sir Sunderlal Hospital, Institute of Medical Sciences, Banaras Hindu University during last one year time period from January 2014 to December 2014. All routine investigations were done in patients preoperatively. The age and sex matched patients were divided into two groups. Group 1 comprises of 64 patients who underwent tonsillectomy under local anesthesia and Group 2 comprises of 25 patients who underwent tonsillectomy under general anesthesia. A comparative analysis of both the group (64 tonsillectomy under local anesthesia and 25 consecutive adult tonsillectomy under general anesthesia) were done. Medical records of patients were reviewed, and the surgical indications, type of anesthesia, surgical technique, operative surgeon, blood loss, operative and post operative complications, treatment outcome were recorded. In addition to routine follow up, telephone interview with local anesthesia group assessed in respect to overall patient's satisfaction, specific complaints, and whether the patient is willing to choose the local anesthesia again either for themselves or for a friend or relative. Total cost of the procedure was calculated including operating room charges and surgical and anesthesia fees. A statistical comparison of the operative blood loss was performed using Student's t-test.

Observation and Results: Total 73 tonsillectomy were performed under local anesthesia in minor operation room during the study period. Nine patients were excluded from the from the study due to inadequate follow up information. Of the 64 cases analyzed, patient age range from 13 to 39 years comprising 32 male and 32 female.
A comparison group of 25 consecutive adult tonsillectomies under general anesthesia, had an age range of 17 to 47 year comprising 15 male and 10 female.

Table 1 is the summary of findings which we observed in our study. Results of local tonsillectomy were tabulated.

Table 1 Tonsillectomy under local anesthesia versus general anesthesia

	Local anesthesia	General anesthesia
Total cases	64	25
Average age	23.7	25.4
Range	14-39	17-47
Average operative blood loss(ml)	42	198
Range of blood loss (ml)	5-200	25-600
Patient acceptance	43/44	---
Complication(total)	2	3
Surgical complication	0	2
Other factors	2	1
Cost	3500	5800

The average operative blood loss for the Group 1 patients tonsillectomy under local anesthesia was 42 ml, with range of 5 to 200 ml. While in Group 2 patients tonsillectomy under general anesthesia, significantly greater average blood loss 198ml was recorded, with a range of 25 to 600ml($P<0.05$). There was one major and one minor complication noted in the Group 1 , both related to the use of medications. In one patient, following local tonsillectomy, diarrhea was noted after four dose of intrvenous Amoxicillin with clavulanic acid, while One local tonsillectomy patient required recourse to general anesthesia. This patient was probably over sedated with intravenous narcotics, and consequently became uncooperative and hysterical. The patient was intubated after removal of first tonsil and contralateral tonsil was removed under general anesthesia without further difficulty.
43(67%) of local tonsillectomy patients were contacted by phone for follow up interviews. 21 patients were excluded because they could not contacted. all local tonsillectomy patients reported total resolution of the symptoms. the only patient who expressed dissatisfaction with the choice of local anesthesia was a 39 years old man who had initially requested general anesthesia, but later reluctantly agreed to local anesthesia.
The average cost of tonsillectomy under local anesthesia was Rs 3500 and under general anesthesia was Rs 5800.

Technique: Patients were premedicated 30 minutes prior to surgery with intra muscular injection of Atropine. A intravenous sedation is started in the operation room, and the patient is monitored with standard electrocardiogram and frequent blood pressure determinations.
Patient is positioned 30 degree upright and 10% lidocaine spray is sprayed in oral cavity and oropharynx. Anterior pillar, posterior pillar, upper and lower pole with pericapsular area and finally the base of tongue is injected bilaterally (2) with 1:100000 lidocaine with epinephrine using a long angulated needle.
The tonsil is then grasped with curved clamp and pulled medially. The inferior margins of the anterior and posterior pillars are incised with a number 12 blade. If the anesthetic has been placed correctly in the peritonsillar space , the dissection of tonsil is easily accomplished with blunt dissection using a dissector and the index finger or scissors. Tonsillar snare is placed over the clamp and the tonsil is snared at the base of the tongue. tonsillar sponges are placed in the tonsillar fossa , and bleeding, which is usually minimal, can be controlled with the electrocautery. The patient is observed and allowed to recover in the post operative recovery room for minimum of 1 hour and/or liquid are tolerated. Post operative antibiotica and analgesics are provided, and patient is admitted overnight.

Discussion: The results of this study demonstrate that using local anesthesia for tonsillectomies in selected, cooperative teenage and adult patients is a safe and practical alternative to the use of general anesthesia. The technique is cost effective and can be easily taught to residents during their training, and associated with the low incidence of complications. In this study, it was found that local tonsillectomies took less time to perform, were cost effective, were associated with

less perioperative blood loss, and were associated with a low incidence of complications.(3,4). The patients under going tonsillectomy under local anesthesia has less intraoperative blood loss in comparison to tonsillectomy under general anesthesia, this may be related to number of factors, including the use of epinepherine in local lignocaine infiltration (5), the upright position of the patient. When one adds the theoretical risk associated with general anesthesia(6), it become clear that , overall, tonsillectomies using local anesthesia are a preferable alternative in cooperative teenager and adult.

Conclusion: This series strongly attests to the efficacy and safety of local tonsillectomy with a low complication rate and low perioperative blood loss when compared with the patients undergoing the same procedure under general anesthesia. Moreover, in the financially conscious era, it is important to emphasize the cost effectiveness of local tonsillectomies.

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APPEAL

All the life members who had already paid Rs. 500.00 as Life Membership fee are requested to send a DD of Rs. 500.00 in favor of A.A.I.M. payable at Varanasi for purchase of Land of office of Association (C.C.) at Varanasi. The members who will donate Rs. 1001.00 or more will be presented a certificate and their name will be published in the Journal with their Photographs. Due to increase in Postal Charges the Journal will be send only to those members who will send Rs. 100.00 as Postal Charges by M.O./ D.D. in favor of *Sangyaharan Shodh*.


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ROFIX-OX Cefixime + Ofloxacin
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ROULET-DSR Rabeprazole Sodium + Domperidone
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FOZAC Cefoperazone Sodium + Sulbactam
MIZIT Azithromycin
MPROZ Methyl Prednisolone Sod. Succinate
MPROZ-A Methyl Prednisolone Sod. Acetate
PIPZAR Piperacillin + Tazobactam
ROCEF 250/500/1GM Ceftriaxone
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AAIMCON-2015
&
Annual Scientific Seminar
April 10th to 11th, 2015
Rajiv Gandhi
Govt. Post Graduate Ayurvedic College
Rajiv Gandhi Govt. Post Graduate Ayurvedic College,
Paprola, Kangra (H.P.) Pin - 176115

Dear Colleagues

On behalf of the organising committee, we feel honoured in inviting you to join us for the 17th Annual National Conference of the Association of Anasethetists of Indian Medicine & Annual Scientific Seminar of the College, to be held at Rajiv Gandhi Govt. Post Graduate Ayurvedic College Paprola from 10th-11th April 2015. It will be our endeavour to update you with recent developments and new modalities in the field of Anaesthesia, Critical Care and Pain management. The annual scientific seminar being multi-disciplinary would give young researchers both present and past of this college of all the disciplines an opportunity to put forward their experiences in the field of research. The aim of the present conference is to disseminate the knowledge of research based activities among the participants.

A galaxy of prominent speakers will share their experiences and update the delegates with the latest developments in Sangyaharan. This is an effort to propagate the spirit of knowledge, research and presentation among young scientists of Ayurveda and many will be encouraged to present their experiences and work in the scientific sessions of the conference.

We are eagerly awaiting your arrival at Rajiv Gandhi Govt. Post Graduate Ayurvedic College Paprola. Your presence and active participation will result in success of the conferences.

With warm regards:

Prof. Y.K.Sharma — Dr. Anil Dutt

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Original research/review papers related to the theme of the conference are invited for presentation purposes from Teaches /Researchers/Scientists/P.G.Students. Soft copy of full papers Strictly Typed in time New Roman (English), Kruti Dev 010 (Hindi/Sanskrit) in double space and 12 font size, (Header and footer not allowed) and abstracts (not exceeding 200 words) should be sent by mail to Prof. Sanjeev Sharma Chairman Scientific Committee AAIMCON-2015 and Annual Scientific Seminar, R.G.Govt.P.G.Ayurvedic College Paprola H.P. on his e-mail-profsanjeevhp@gmail.com upto 25th March 2015.A Souvenir of selected paper & abstracts will also be published. The name of the author and co-author of Paper should appear at right upper corner of the first page.

About institute:

Rajiv Gandhi Govt. Post Graduate Ayurvedic College- Paprola is situated in Paprola town (about 700 metres from main town on Andretta Link road). This is a leading post graduate Institute of Ayurveda in India, running UG and PG (11 subjects) courses. College is having very good complex enriched by the natural flora. It is also having its own Hospital and Pharmacy in the campus.

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Paprola is a small and beautiful town of Distt. Kangra (H.P.), situated on the bank of river Binwa which divides Paprola and Baijnath (twin towns). Surrounding green hills of the town are picturesque and rich in flora having many important medicinal plants.

Connections:-

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Climate:-

The climate of Paprola in April will be pleasant. But on rainy day it becomes cold so light woollen clothes may be required (18^0C to 25^0C)

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Category	Upto 15th March	Upto 1st April	Spot
Reception Committee	2500/-	2500/-	2500/-
Delegates	1000/-	1500/-	2000/-
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Organizing Secretary

Effect of Shallaki Niryasa in Sandhi Vat

JK Choubey* **DN Pande****

Abstract: Joint pain is one of the commonest disorders broadly coming under vatavyadhi and affects to the skeletal system at the geriatric age group.Nowaday's joint disorder is one of the main cause of distress after age of third decade. Though medical science provides powerful analgesic drugs and new surgical tools available today still this disease remains a challenge for the research scholars a thorough study was planned to evaluate the efficacy of ShallakiNiryasa on 50 patients.

Keyword: Agni Karma, Shallaki, Niryasa, Pain.

Introduction: Ayurveda, the science of life, has its own methodology and hypothesis tomanage all type of diseases. Joint pain is one of the commonest disorders broadlycoming under vatavyadhiand affects to the skeletal system at thegeriatric age group. This age group is more vulnerable to dhatuksaya and slowly loosestheir own function. Ultimately vatadosabecomesprovocated due to Ksaya or Avaranaprakriya and produces various typesof vatikvyadhies. To overcome such type of degenerative process andsymptomatology various research work are going on to find out newremedy in surgical as well as medical system since ancient period.Now a day's joint disorder is one of the main cause of distress afterage of third decade.Though medical science provides powerful analgesicdrugs and new surgical tools available today. Due to more side effects ofanalgesic drugs and complication of surgical procedure, this disease isremaining a challenge for the research scholar. The clinical study was carried out by giving patients with 500 mg Niryasa of Shallaki 500 mg TID orally.

Examination and Assessment:

- After the registration of the patient, the detailed history was taken and complete physical examination was performed. All findings were noted down in a set proforma, if he/she fulfilled the conditions of inclusion criteria.
- Particulars of the patient including age, sex, occupation, socio-economic status, religion, dietic habits etc.
- Chief complaints with duration of symptoms, their commencement, history of present illness including history of trauma, straining and nature of pain.
- History of past illness, particularly regarding trauma/straining of affected part.

***Research Scholar**Professor, Department of Sangyaharan, Faculty of Ayurveda, IMS, BHU, Varanasi.**

Selection of patients:

All the patients attending SangyaharanVedanahar clinic suffering from Aamvata, Kativata, Gridhrasiespecially Sandhivatwere selected for this study.

Inclusion criteria:

- Patients having typical clinical features pertaining to above condition.
- Patients willing to undergo trial.
- Patients between age group 20-70 years, of either sex.

Exclusion criteria:

- Patients below 20 years and above 70 years of age.
- Patients not willing to undergo trial.
- Patient suffering from diabetes mellitus, tubercular arthritis,rheumatoid arthritis etc.
- Patients of PaittikPrakriti, AlpaSatva, AvarSahanam, Pregnant woman.

Laboratory Investigations:

Blood investigation Hb, TLC, DLC, ESR.
FBS, BU, S. Creatinine, S. Uric acid,
R.A. Factor.
HIV, HbsAg
X-ray of the affected part of the body.

Physical Examination

General Examination- About the Prakriti, Satva, Sara, Samhanana, general appearance, weight, pulse, B.P., respiration rate of the patient.

Local Examination:

Inspection - Attitude
Swelling
Deformity
Texture or colour of skin
Presence of any wound, ulcer, spots, scars, sinuses,
Wasting, muscle spasm etc.

Palpation - Local temperature Skin texture
Muscle spasm
Swelling
Tenderness
Crepitus

Movements - Range of movements were assessed clinically.

Criteria for Assessment

Improvement in the patient has been assessed mainly on the basis of relief in the cardinal signs and symptoms. To assess the effect of therapy objectively, all the signs and symptoms were given scoring depending on their severity as below:

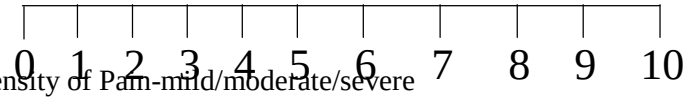
- Pain
- Radiation of pain
- Tenderness
- Ability to do daily routine work

Change in the range of movement

1. Pain (Ruja)

B) Visual Analogue scale – 0 to 10

0 = no pain
1 - 3 = mild pain
4 - 7 = moderate pain
8 - 10 = severe pain



B) Intensity of Pain-mild/moderate/severe

a) No Pain 0
b) No Pain at rest but pain occurs afterphysical work 1
c) Pain also present at rest but mild 2
d) Pain also present at rest but moderate 3
e) Pain also present at rest but severe 4

2. Pricking sensation (Toda)

a) No pricking sensation 0
b) Occasional pricking sensation 1
c) Constant mild pricking sensation 2
d) Constant moderate pricking sensation 3
e) Constant severe pricking sensation 4

3.	**Unable to do daily routine work by affected part (Daurbalyata)**		
	a)	Can actively do all the routine work	0
	b)	Can do daily routine work but have to take rest intermittently	1
	c)	Can do daily routine work but have to take rest veryoftenly	2
	d)	Can't do daily routine work	3
	Karnofsky performance scale		
	a)	Normal activity with no special care	1
	b)	Unable to work but able to live at home	2
	c)	Needs hospital care	3
4.	**Radiation of pain**		
	a)	No radiation of pain	0
	b)	Pain radiates up to thigh	1
	c)	Pain radiates up to knee joint	2
	d)	Pain radiates up to leg	3
	e)	Pain radiates up to ankle	4
	f)	Pain radiates up to foot	5
5.	**Tenderness**		
	a)	No pain on palpation	0
	b)	Pain occurs on deep palpation	1
	c)	Pain occurs on light palpation	2
	d)	Patient does not allow to touch the affected part	3

ShallakiNiryasa is a traditional remedy in Ayurvedic medicine used in India for variety of inflammatory diseases including rheumatoid arthritis, osteoarthritis & cervical spondylosis. The main constituents of the gum resin are boswellic acids and other compounds such as volatile oils, terpinols, arabillosa, xylose, galactose, uronic acids, and phlobaphenes. The known pharmacological effects of olibanum are anti-inflammatory analgesic, immunomodutation, hepatoproteclive and antimicrobic. ShallakiNiryasa has been shown to possess anti-inflammatory and analgesic property.

Grouping of patients:

AGE, WEIGHT AND HEIGHT

Table 1: Mean age, mean weight and mean height of the patients for the trial group.

Trial group	Age (years) Mean ï SD	Weight (Kg) Mean ï SD	Height (cm) Mean ï SD
Shallaki	44.68 ± 14.19	55.90 ± 4.98	159.20 ± 7.25

2. EFFECT ON PULSE:

Table 2: The effect on pulse per minute before treatment and after treatment

		Pulse
Pulse Before Treatment Mean ⟨ SD		78.06 ⟨ 5.78
Pulse After treatment Mean ⟨ SD		77.27 ⟨ 5.09
Comparison within the group	t value	0.999
	p-value	> 0.05
REMARK		NS

No statistical change was observed in pulse rate as observed at before treatment vs. after treatment.

3. EFFECT ON OXYGEN SATURATION:

Table 3: The effect of shallaki on oxygen saturation level before treatment and after treatment

		Oxygen saturation
SPO_2 Before Treatment Mean ⟨ SD		98.08⟨ 0.966
SPO_2 After treatment Mean ⟨ SD		97.88⟨ 1.081
Comparison within the group	t value	1.520
	p-value	> 0.05
REMARK		NS

Oxygen saturation level was statistically insignificant in this groupas observed at before treatment vs. after treatment

4. EFFECT ON MEAN BLOOD PRESSURE (MBP):

Table 4: The effect of shallaki on Mean Blood pressure before treatment and after treatment

		Mean blood pressure
MBP Before Treatment Mean ⟨ SD		93.44⟨ 7.96
MBP After treatment Mean ⟨ SD		93.40⟨ 7.14
Comparison within the group	t value	0.150
	p-value	> 0.05
REMARK		NS

Changes in Mean blood pressure was statistically non significant as observed at before treatment vs. after treatment.

5. EFFECT ON RESPIRATORY RATE (RR) :

Table 5: The effect of respiratory rate per minute before treatment and after treatment

GROUP		GROUP B (Shallaki)
RR Before Treatment Mean ± SD		18.02± 1.34
RR After treatment Mean ± SD		17.28± 1.52
Comparison within the group	t value	2.84
	p-value	< 0.01
REMARK		HS

Changes in Respiratory rate is statistically highly significant as observed at before treatment vs. after treatment.

6. EFFECT ON VISUAL ANALOGUE SCALE (VAS)

Table 6: The effect of shallaki on visual analogue scale before treatment and after treatment

		VAS
VAS Before Treatment Mean ± SD		7.76± 0.847
VAS After treatment Mean ± SD		2.62± 2.14
Comparison within the group	t value	16.07
	p-value	< 0.001
REMARK		HS

Changes in visual analogue scale wasstatistically highly significant as observed at before treatment vs. after treatment.

7, EFFECT ON KARNOFSKY SCALE (KSKY) -

Table 7: The effect of shallaki on Karnofsky pain scale before treatment and after treatment

		KARNOFSKY SCALE (KSKY)
KSKY Before Treatment Mean ± SD		0.98± 0.143
KSKY After treatment Mean ± SD		0.08± 0.277
Comparison within the group	t value	20.55
	p-value	< 0.001
REMARK		HS

Changes in Karnofsky pain scale is highly significant as observed in before treatment vs. after treatment.

8. EFFECT ON PRICKING SENSATION:

Table 8: The effect of shallaki on pricking sensation before treatment and after treatment

		Pricking sensation
Before Treatment Mean ± SD		1.64± 0.82
After treatment Mean ± SD		.58± 1.071
Comparison within the group	t value	8.212
	p-value	< 0.001
REMARK		HS

Changes in pricking sensation is highly significant as observed at before treatment vs. after treatment.

9. EFFECT ON PAIN RADIATION :

Table 9: The effect of shallakion radiation of pain before treatment and after treatment

		Pain radiation
Before Treatment Mean ± SD		1.52± 1.31
After treatment Mean ± SD		0.34± 0.479
Comparison within the group	t value	7.99
	p-value	< 0.001
REMARK		HS

Changes in radiation of pain is highly significant as observed at before treatment vs. after treatment.

10. EFFECT ON TENDERNESS:

Table 10: The effect of shallakion tenderness before treatment and after treatment

		GROUP B (Shallaki)
Before Treatment Mean ± SD		1.02± 0.247
After treatment Mean ± SD		0.18± 0.388
Comparison within the group	t value	14.08
	p-value	< 0.001
REMARK		HS

Changes in tenderness is highly significant as observed at before treatment vs. after treatment.

Conclusion:

Pulse rate, oxygen saturation and mean blood pressure showed statistically insignificant changes but regarding respiratory rate showedstatistically significant change.

Changes in Visual analogue scale, Karnofsky pain scale, Pricking sensation, Radiation of pain and tenderness is highly significant as observed at before treatment vs. after treatment.

On the basis of the above observations it can be attributed that Niryasa of shallakican be used for the management of pain in musculoskeletal disorders. The Shoolprashaman property of Niryasa of Shallakiis well established in Ayurveda and is being used in many painful conditions is also justified here.

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