AN IN – VIVO STUDY ON THE CONCEPT OF APATARPANA IN SANTARPANAJANYA VIKARA WITH SPECIAL REFERENCE TO PRAMEHA

A Thesis

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DOCTOR OF PHILOSOPHY
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Under the board of Ayurveda Studies

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April 2022

CERTIFICATE

It is certified that work entitled "**An in – vivo study on the concept of Apatarpana in Santarpanajanya Vikara with special reference to Prameha"** is an original research work done by Vd. Raja Rajeshwari N M under my supervision for the degree of Doctor of Philosophy in Ayurveda (Samhita Siddhant) to be awarded by Tilak Maharashtra Vidyapeeth, Pune. To best of my knowledge this thesis

- Embodies the work of candidate
- Himself/herself has duly been completed
- Fulfils the requirement of the ordinance related to Ph. D. degree of the TMV
- Up to the standard in respect of both content and language for being referred to the examiner.

Signature of the Supervisor

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Undertaking

I Vd. Raja Rajeshwarin N M is the Ph. D Scholar of the Tilak Maharashtra Vidyapeeth in Samhita Siddhant in the Dept. of Ayurveda subject. Thesis entitled **"An in – vivo study on the concept of Apatarpana in Santarpanajanya Vikara with special reference to Prameha"** under the supervision of Dr Abhijit H. Joshi Solemnly affirm that the thesis submitted by me is my own work. I have not copied it from any source. I have gone through extensive review of literature of the related published / unpublished research works and the use of such references made has been acknowledged in my thesis. The title and the content of research is original. I understand that, in case of any complaint especially plagiarism, regarding my Ph.D. research from any party, I have to go through the enquiry procedure as decided by the Vidyapeeth at any point of time. I understand that, if my Ph.D. thesis (or part of it) is found duplicate at any point of time, my research degree will be withdrawn and in such circumstances, I will be solely responsible and liable for any consequences arises thereby. I will not hold the TMV, Pune responsible and liable in any case.

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Vd. Raja Rajeshwari N M

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ABSTRACT

STRUCTURED ABSTRACT

TITLE – AN IN – VIVO STUDY ON THE CONCEPT OF APATARPANA IN SANTARPANAJANYA VIKARA WITH SPECIAL REFERENCE TO PRAMEHA

BACKGROUND – *Ayurveda* says the factors responsible for disease and health is one. The factors when consumed in a method explained in the text, will be responsible for health and when taken inappropriately leads to disease. Hence treatment principle and causative factors can be basically classified into *Santarpana* and *Apatarpana*. *Santarpana* is the principle which brings nourishment to the *Dhatus* and *Apatarpana* is one which brings depletion of *Dhatus*. The *Apatarpana* is a wide term dealt in different context with different methods of administration of treatment principle which broadly includes – *Rukshana*, *Langhana* and *Swedana*. *Prameha* is one of the *Santarpanajanya Vikara* and the treatment plan includes the use of *Apatarpna* as a part of *Chikitsa*. Even though the word *Apatarpana* is not directly mentioned in *Prameha Chikitsa* but it has been explained in between the lines. The study carried out helps in understanding the *Apatarpana Upakrama* in *Prameha*. One of the ways of adopting *Apatarpana* in *Prameha* has been mentioned in the form of dietary preparations which includes use of whole grains of *Yava* (barley- Hordeum vulgare). Various methods of bio processing of *Yava* (barley- Hordeum vulgare) have been explained in *Ayurveda* literature and one among it was by processing *Yava* (barley- Hordeum vulgare) in *Triphala Kashaya*. *Triphala Kashya* is known to have anti- diabetic property and can hence enhance the bio availability of *Yava* and proving it to be efficacious in *Prameha*.

OBJECTIVES OF THE STUDY

- To critically analyse the concept of *Apatarpana* from *Brihattrayee* and *Laghutrayee*.
- To evaluate the concept of *Apatarpana* in *Prameha* with *Triphala Sadhita Yava* in Streptozotocin (STZ) induced diabetes in Wistar albino rats.

Conceptual study:

Review of the fundamental concepts *Apatarpana* as a *Upakrama* along with *Santarpana* as a *Hetu*, *Prameha Santarpana Janya Vyadhi* and *Triphala Sadhita Yava* as *Apatarpana Upakrama*.

Applied study

Pharmaceutical study: This included drug procurement, authentication and preparation of the *Triphala Sadhita Yava*.

Experimental study: It was carried to study the effect of *Apatarpana* (*Triphala Sadhita* Yava) on *Prameha* by using *Triphala Sadhita Yava* on 24 parameters.

OBSERVATION AND RESULTS

The observations were tabulated and subjected for statistical test and results were computed by Dunnett's multiple comparison't' test using Graph Pad Prism 3. A $p<0.05$ was considered as statistically significant.

CONCLUSION

Conclusions are drawn by the critical study of the concept and in vivo study results.

KEY WORDS: *Apatarpana, Upakrama, Santarpana, Prameha, Yava, Barley, Triphala*

LIST OF TABLES

Abbreviations

Sl no	Abbreviation	Full form
1.	STZ	Streptozotocin
2.	SGOT	Serum Glutamic Oxaloacetic Transaminase
3.	SGPT	Serum Glutamic Pyruvic Transaminase
4.	ALP	Alkaline Phosphatase
5.	TP	Total Protein
6.	ALB	Albumin
7.	GLO	Globulin
8.	T.Bil	Total Bilirubin
9.	D.Bil	Direct Bilirubin
10.	TG	Triglycerides
11.	HbA1c	Glycated hemoglobin

Chapter I

INTRODUCTION

INTRODUCTION

Ayurveda says the factors responsible for disease and health is one. The factors when consumed in a method explained in the text then it will be responsible for health and when taken inappropriately leads to disease.[1] Hence treatment principle and causative factors can be basically classified into *Santarpana* and *Apatarpana*. *Santarpana* is the principle which brings nourishment to the *Dhatus* and *Apatarpana* is one which brings depletion of *Dhatus*.

The *Apatarpana* is a wide term dealt in different context with different methods of administration of treatment principle which broadly includes – *Rukshana*, *Langhana* and *Swedana*.[2] Even though it is widely explained, a comprehensive work exclusively needs to be done to understand its applicability in *Chikitsa*. And also an entire chapter '*Santarpaneeyam Adhyayam'* has been exclusively dealt in *Charaka Samhita* where the diseases caused by *Santarpana* have been explained and the treatment for the same have been discussed. Keeping this chapter as a base, a conceptual study was carried out to understand the concept of *Apatarpana* and to compare it with treatment principles of the diseases explained under *Santarpaneeyam Adhyaya*.

Prameha is one of the *Santarpanajanya Vikara* and the treatment plan includes the use of *Apatarpana* as a part of *Chikitsa*.[3] Even though the word *Apatarpana* is not directly mentioned in *Prameha Chikitsa* but it has been explained in between the lines. The study carried out helps in understanding the *Apatarpana Upakrama* in *Prameha*. One of the ways of adopting *Apatarpana* in *Prameha* has been mentioned in the form of dietary preparations which includes use of whole grains of *Yava* (barley- Hordeum

vulgare). Various methods of bio processing of *Yava* (barley- Hordeum vulgare) have been explained in *Ayurveda* literature and one among it was by processing *Yava* (barley- Hordeum vulgare) in *Triphala Kashaya*. *Triphala Kashya* is known to have anti- diabetic property and can hence enhance the bio availability of *Yava* and proving it to be efficacious in *Prameha*. With this background this study was carried out to assess the anti-diabetic potential of *Yava* (barley – Hordeum vulgare) processed in *Triphala Kashaya*.[4]

For this an experimental study was planned using *Triphala Sadhita Yava* as a method of managing the *Santarpanajanya Vikara Prameha.*

OBJECTIVES

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Chapter II

REVIEW

OF

LITERATURE

REVIEW OF LITERATURE

Concept of *Dosha*

Dosha are the fundamental factors responsible for normal functioning of the body. Hence the understanding of disease and treatment principle is explained in terms of *Dosha*.

Doshaguna[5]:

Table No. 1 *Guna of Vata*

Sl no	*Guna*	*Charaka Samhita*	*Ashtanga Hridaya*	*Ashtanga Sangraha*	*Sushruta Samhita*
1.	*Ruksha*	+	+	+	+
2.	*Sheeta*	+	+	+	+
3.	*Laghu*	+	+	+	+
4.	*Sukshma*	+	-	+	+
5.	*Chala*	+	-	+	+
6.	*Vishada*	+	-	-	+
7.	*Khara*	+	+	+	+

Table No. 2 *Guna of Pitta*

Sl no	*Guna*	*Charaka Samhita*	*Ashtanga Hridya*	*Ashtanga Sangraha*	*Sushruta Samhita*
1.	*Sasneham*	+	+	+	-
2.	*Ushnam*	+	+	+	+
3.	*Tikshnam*	+	+	+	+
4.	*Dravam*	+	+	+	+
5.	*Amlam*	+	-	-	-

6.	*Saram*	+	+	+	
7.	*Katu*	+	-	-	+
8.	*Laghu*	-	+	+	-
9.	*Visram*	-	+	+	-

Table No. 3 *Guna of Kapha*

Sl no	*Guna*	*Charaka Samhita*	*Ashtanga Hridaya*	*Ashtanga Sangraha*	*Sushruta Samhita*
1.	*Guru*	+	+	+	+
2.	*Sheeta*	+	+	+	+
3.	*Mrdu*	+	-	-	-
4.	*Snigdha*	+	+	+	+
5.	*Madhura*	+	+	+	-
6.	*Sthira*	+	+	+	-
7.	*Picchila*	+	-	-	+
8.	*Manda*	-	+	+	-
9.	*Shlakshana*	-	+	+	-
10.	*Mrutsna*	-	+	+	-

Contradictory and Similarity of *Guna* and Mutual co-existence of *Dosha*

1. *Dosha* innately have the *Dushanaswabhava*, therefore possess the inherent capability to cause *Dushti* and *Dosha* have *Guna* which are mutually contradictory. But it is seen that despite this fact, they do not counteract each other even during *Sama Avastha* and also the deranged *Dosha* cannot be counteracted by the other two *Dosha* which are in *Prakrata* state because they do not have the capacity to do so.

2. There are some *Gunas* which are common to one another. For example, *Sheeta* is common to *Vata* and *Kapha*, *Amla* and *Lavana* are common to *Pitta* and *Kapha*, *Katu* and *Laghu* are common to *Vata* and *Pitta*, and so on. That is, there are causative factors of *Dushti* which are common to more than one *Dosha*. Such factors behave as common *Hetu* in causing vitiation of more than one *Dosha* simultaneously, thus leading to *Sansarga/ Sannipata* diseases[6].

Tridosha Involvement in Diseases

Diseases are innumerable eventhough the *Dosha* are only three because of the variation in the *Prakruti* (*Dosha*), *Adhishthana* (*Dushya*), *Linga* (Clinical presentation) and *Ayatana* (causative factors).[7]

Samanya Vishesha Siddhanta

Treatment principles in Ayurveda are based on the fundamental doctrine of *Samanya* and *Vishesha*. *Dosha* and *Dhatu Vaishamya* are brought back to *Samya* by using the effects exerted by *Dravya / Aushadha* by virtue of *Samanya* and *Vishesha*.

Samanya can be inferred wherever there is an increase of *Guna* or *Karma* and *Vishesha* can be inferred wherever there is a decrease. The mechanism of increase or decrease of the *Guna* or *Karma* in the bodily *Dhatu* can be attributed to *Samanya* or *Vishesha* respectively.

Understanding *Samanya*

It has been observed that the mere existence of similarity does not yield increase, such as the *Amla Rasa* in *Amalaka* does not in any way contribute to increase of *Pittam*. This phenomenon can be understood by the analysis of other qualities present in *Amalaka*, one being *Sheetaguna*. This, being contradictory to the *Ushnaguna* of *Pittam*, leads *Amalaka* to pacify *Pittam*, despite the presence of a similar quality. This counteracting factor is considered as the *Virodhikarana*. On the other hand, it has also been observed that *Ghrtam* leads to the increase of *Agni* and *Medha*, despite not having *Samana* qualities of neither *Agni* nor *Medha*. This must be attributed to the *Prabhava* of *Ghrtam*. Therefore, it must be inferred that while the resultant increase can be attributed to *Samanya*, the existence of *Samanya* itself shall not yield a definite increase.

Understanding *Vishesha*

The observed decrease of bodily *Guna* or *Karma* is attributed to *Vishesha*. The mechanism of decrease can be inferred by means of *Vishesha*. That which is dissimilar/ distinct/ opposite can be understood as *Vishesha*. In the presence of dissimilar or opposite qualities, the decrease can be caused only when there are no counteracting factors contributing to increase. An example for this is *Mandaka* and *Nikucha*, which, even in the presence of *Snigdhadiguna* lead to increase of *Vata*, because of *Apathyaprabhava*. Therefore, *Virodhikarana* are appreciable in the case of *Vishesha* as well.

Vishesha can be of two kinds – *Viruddhavishesha* (stark opposite) and *Aviruddhavishesha* (merely dissimilar). *Viruddhavishesha* is a sure factor for decrease due to the presence of opposite qualities. Meanwhile, *Aviruddhavishesha* can be attributed to decrease only due to the observation that it does not contribute to increase. With respect to the bodily *Dhatu*, as they are being deteriorated in every smallest frame of time, they require constant supplementation, which is nothing but increase, attributed to *Samanaguna*. When the supplementation does not take place due to unmatched

qualities (dissimilar/ *Aviruddhavishesha*), there is no net increase and as a result it is still considered as decrease. Therefore, having dissimilar qualities is also considered as a cause for decrease.

Pravrttihubhayasyatu

While stating the law/ application of this, an important clause is given by *Charaka* as *Pravrttihubhayasyatu*. This indicates that *Samanya* and *Vishesha* are not exclusive occurrences with respect to time. That is, for every occurrence of *Samanya*, there is a corresponding consequent occurrence of *Vishesha* at the same time. Increase of one *Guna* in the body leads to the consequent decrease of the *Guna* opposite to it. So, continuous application of a single *Upakrama* cannot be followed as it can in turn lead to derangement of *Dhatu Samya* and defeat the purpose of *Chikitsa*[8] (*Cha. Su*. 1/ 45, *Chakrapani*)

Upakrama of *Sansarga* and *Sannipata* Conditions

The *Dosha* which has undergone the maximum derangement has to be determined and treated first, using the *Upakrama* mentioned for itself.

1. In *Vata Pitta Sansarga*, the treatment should comprise of *Aushadha* of *Vata* and *Aushadha* for *Pitta*, simultaneously remaining mutually non-contradictory. The principle of treatment which is explained in *Greeshmaritucharya* needs to be followed where the use of *Madhura Rasa*, etc. which subside *Vata Pitta* should be advised and use of *Patu, Katu, Amla, Vyayama*, etc. which increase *Vata Pitta* are contraindicated. This principle is used because *Vata* has *Yogavaitva.* When *Vata* is associated with *Pittam*, then it produces symptoms such as *Daha*. But when *Pittam* remains in the physiological state, *Dahadi* symptoms cannot be produced. And during *Greeshmaritusheeta* is not the only *Guna* incorporated in *Greeshmaritucharya*. *Snigdhadravya* are also indicated. Therefore, *Greeshmaritucharya* can never directly contribute to increase *Vata*.

2. In *Sansarga* of *Kapha-maruta*, the approach of treatment can be considered to be similar to *Vasantaritucharya* like use of *Tikshnaihvamananasyadyaih*, etc. During *Vasantaritu*, the association of *Vata* enables the derangement of *Kapha*, due to the *Yogavahi* property of *Vata*. Therefore, it is only logical to apply the treatment which has opposite nature to *Kapha*, such as *Vamana* and *Nasya*. Therefore, they cannot result in increase of *Vata*. Thereby, it is to be understood that *Pitta Chikitsa* is to be given for *Vata* associated with *Pittam, Kapha Chikitsa* for *Vata* associated with *Kapha*.

3. In conditions of *Sansarga* of *Kapha* and *Pittam*, the approach of treatment can be considered to be similar to *Sharadritucharya*.

4. In case of *Sannipata Chikitsa*. Here, *Varsharitucharya* is to be adopted, as during *Varsharitu*, there is simultaneous derangement of all the *Dosha* as per the lines of *"Bhajetsadharanamsarvam…"*[9].

Status of *Dosha* in Planning the *Upakrama*

Cayaavastha: The *Dosha* which is disturbed in its own *Sthana* is *Caya*
Kopa: The *Dosha* which causes disturbances in other *Sthana* is *Kopa*.

Dosha Gati

The *Dosha* in *Kupitaavastha* spreads throughout the body right from the sole of the foot to the head, like flowing water. Due to other specific reasons, the *Dosha* that has spread can also undergo gradual decrease, again, like flood. Just like how the flow of water, when strong, can spread everywhere, the *Kupitadosha* spreads all over the body. But when there may be reasons for it to reduce the flow, the water gradually retreats and decreases the flow[10].

The flow of *Doshas* from the *Koshta* to the *Shakha* can be through four factors as follows:

1. *Vyayama* – the combination of fatigue, exertion and heat generated in the body due to physical exercise causes upward movement of *Vata* which expels the *Shithila* and *Cala Dosha* out of it own *Sthana* haphazardly throughout the body towards the *Shakha*.
2. *Ushmanahtaikshnya* – coming in contact with heat such as *Atapa*, causes the *Dosha* to attain a state of *Vilayana*. Simultaneously the heat causes the *Srotomukha* to get activated and stimulated thereby enabling easy movement of the *Dosha* from *Koshta* to the *Shakha*.
3. *Ahitacarana* – indulging in *Ahitaahara* and *Vihara* causes direct increase in the quantity of *Dosha* present in the *Koshta*. There, it undergoes voluminous increase and fills up the *Srotas* from the *Koshta* which is at a depressed level to the *Shakha*, which is at an elevated level, just like how a flood rises from a water body due to excessive rain.
4. *Drutatvatmarutasya* – another contributing factor for the movement of *Dosha* is the speed of *Vata*. With increased speed, there is further movement.

The flow of *Dosha* from the *Shakha* to the *Koshta* can be through five factors as follows:

1. *Srotomukhavishodhana* – the *Dosha* situated at the elevated level moves back in to the *Koshta*, when their pathway is unobstructed. This is achieved by clearing the *Srotomukha*.
2. *Vrddhi* – causing a quantitative increase in the *Dosha* causes their own *Srotas* to be completely filled, thereby leaving the *Koshta*.

3. *Abhishyandana – Dosha* attain a specific state of liquidity caused due to external heat such as *Atapa* and also dietary items such as *Kshira*, *Dadhi*, etc.
4. *Paka* – when *Dosha* undergo *Paka*, their movement becomes unobstructed towards the *Koshta*.
5. *Vayohnigraha* – this is an inevitable factor to enable the other 4 above.

The *Dosha* when situated in the *Koshta* are not capable of generating diseases. *Dosha* present all over the body remain in an inactive state due to less *Shakti*. On occurrence of *Hetu*, there occurs consequent derangement of the *Dosha* and these *Hetu* in the form of *Kaladi*. *Kaladi* indicates *Kala, Desha* and other *Apathya* which have similar *Guna* to the *Dosha*. Occurrence of such factors will impart more *Balam* to the *Dosha*, i.e., they impart more disease-causing ability to the *Dosha*. Thus, they are awakened from a dormant, inactive state to a pathogenic state. They incur the ability to move to other *Sthana* as well, apart from increase, from pre-existing *Koshta* to *Shakha*, or vice-versa[11].

Based on the *Taratama Bhava* of *Dosha*

1. When there is a stage of derangement of all the *Dosha*, but in different stages of vitiation, the physician must assess the level of vitiation of each *Dosha* and identify the one which has attained the maximum progress, and the treatment must be predominant of the *Upakrama* of that *Dosha*. At the same time, it must be designed in such a way that the other two *Dosha* remain unaggravated.

2. When there is a stage of derangement of all the *Dosha*, but in same stages of vitiation there are different approaches of treatment principle needs to be followed:
    a. The priority of treatment must be first given to *Vata*, followed by *Pittam* and lastly, *Kapha*. This is because, the inherent *Balam* of *Pittam* is more than *Kapha*. *Vatadosha* has more *Balam* as compared to *Pittam*. Substantiation to this order is given by quoting *Parasharshi – Vata*, being the initiator of all movement, is compared to the commander of an army. Here, *Pittam* and *Kapha* are compared to the troop of the army. When the commander of an

army is conquered over, the entire army surrenders. Thus, once *Vata* is pacified, it is easier to manage *Pittam* and *Kapha*, thereby justifying the priority given to *Vata*.

b. Based on *Sthanam*, the priority is given to *Shleshma* first, followed by *Pittam* and lastly, *Vata*. The logic behind it is given as follows. Aggravated *Shleshma* exhibits symptoms such as *Aruchi*, which hampers the intake of food, drinks and medicine. This might disable the physician to administer diet or medicine, due to which the *Dosha* imbalance remains untreated. Therefore, aggravated *Kapha* is compared to a bolt/ latch/ door to the *Mahasrota*s, which is considered as the entry point to the body. When the entry point is closed, the internal pathology cannot be accessed and treated. Therefore, priority is given to *Kapha*. On the other hand, *Pittam* acts in association with *Vata* and is *Ashukari* in nature. Therefore, the second position in the order of priority belongs to *Pittam*. When the two *Doshas* are under control, *Vata* becomes manageable.
c. Based on the role of the *Dosha* in contributing to the pathology, another order has been opinionated by *Sushruta*. *Pittam*has to be treated first in conditions such as *Atisara* and *Jwara*, followed by *Kapha*, and lastly, *Vata*. The reason for this is as follows – in *Jwara*, the prime symptom in *Santapa,* which is generated by the virtue of *Pittam*. In *Atisara,* the *Vit* is eliminated in liquid form due to the attainment of *Mrdykohthata*, both of which is the action of *Pittam*. The associated symptoms such as *Gaurava* is due to the action of *Kapha,* which gives it second position in the order or priority. When both *Pittam* and *Kapha* are pacified, there is resultant *Vrddhi* of *Vayu* which can then be directly managed using its *Chikitsa*. Therefore, such an order remains justified.

d. Yet another opinion has been enlisted in the order of priority, which is *Kapha*, followed by *Pittam* and lastly, *Vata*. This is reasoned as follows – *Jwara* and

Atisara, are diseases resulting from the *Utklesha* of *Amashaya*. Therefore, adopting *Pittaharachikitsa* such as *sheeta* shall be *Apathya* to the *Sthanam*.

Based on the *Sthanika* and *Agantudosha*

In conditions where the *Dosha* is present in the *Sthana* of other *Dosha*, the treatment must be in line with the *Sthanidosha*. This is allowed only if the occupant is possessing comparatively less *Balam* to the *Sthani*. When the occupant possesses higher *Balam* then the *Chikitsa* must be in line with it only. This occurs when the occupant *Dosha* is aggravated and gains more *Balam* by its own causative factors. This linear rule is counterfeited with exceptional cases as follows:

1. During conditions where the *Agantudosha* is stronger than the *Sthanadosha*, the *Chikitsa* must be in line with the *Agantudosha*. After successfully managing the *Agantudosha*, the *Sthanidosha* must also be pacified in such a way that it does not give rise to diseases of its own origin.

2. On the other hand, when the *Agantudosha* is less strong than the *Sthanidosha*, the treatment must not be considered complete with the mere management of *Sthanidosha*. *Agantudosha* must also be pacified[12].

ORDER OF PREFERENCE OF *RASA* FOR *DOSHA*

In case of increased *Vata*, the choice of *Rasa* are *Patu*, *Amla*, *Madhura*, in that order. The *Rasa* of highest preference in case of increased *Pitta* is *Tikta*, followed by *Madhura* and then *Kashaya*. Similarly, for increased *Kapha*, the *Rasa* of choice is *Katu*, followed by *Tikta* and *Kashaya*[13].

Sama Niramata of *Dosha*:

After explaining about the *Upakrama* of *Dosha* and their status, the *Upakrama* of *Samadosha* is explained. In order to identify the status of *Samadosha*, the *Lakshanas* are enlisted below:

1. *Srotorodha*: obstruction in the pathway of *Dosha* and *Dhatu*
2. *Balabhransha*: decrease in *Bala*
3. *Gaurava*: feeling of heaviness in the body
4. *Anilamudhata*: hindrances in the path of *Vata*
5. *Alasyam*: feeling of inertia
6. *Apakti:* disturbance in metabolism
7. *Nishtiva*: excessive salivation
8. *Malasanga:* improper expulsion of excretory products
9. *Aruchi:* disinterest in food
10. *Klama*: fatigue

The state of *Niramadosha* is identified by opposite *Lakshana* such as

1. *Srotahshuddhi*
2. *Samyakbala*
3. *Laghava*
4. *Sanilasyasamyaksancharah*
5. *Analasya*
6. *Samyakpakti*
7. Absence of *Nishtiva*
8. Absence of *Malasanga*
9. *Samyakruchi*
10. Absence of *Klama*[14]

Digestion and Metabolism in Ayurveda *Aharapakakrama*

The fate of the food contents on reaching the *Koshta* gets acted upon by *Agni* to get converted into absorbable form to nourish the *Dhatu*. The process of digestion begins right in the mouth.The process of deglutition and the further movement of the food in the *Kantha* (*Adana Karma* indicates the function of carrying/ bringing the food to the *Koshta*) is because of *Pranavata*. This food which is consumed is broken down into smaller particles, becomes moistened and soft because of the liquid content which is consumed by the food. The *Agni* which is being stimulated by *Samanavata* which is in its normal state digests the food appropriately when it is taken in time and right method and thereby eventually leads to longevity. As *Udeerna Agni* is responsible for metabolising food contents the food should be consumed during the time of hunger. '*Pavanodvaha'* is used as a descriptive term for *Agni* as the *Agni* is kindled with the help of *Samanavata*, which is present in proximity to the *Agni*. When this *Samanavata* is functioning normally as per physiology, it kindles the *Agni* like the role of air being blown to kindle the fire. It also helps to maintain the fire from being extinguished, therefore, prevents *Vaishamyata*. Deranged functioning of *Samanavata* thereby causes consequent inconsistency in the functioning of *Agni* as well. When the food is taken in '*Samam'* and '*Samyak'* manner, the *Agni* metabolises the food and thereby contributes to the *Vriddhi* of *Ayu*. *Sama* indicates the quantity of food taken and '*Samyak'* is used as a descriptive term for food, wherein, it indicates the quantity of food consumed or can also be used to indicate the appropriate use of *Prakrti, Karana, Samyoga, Rashi, Desha, Kala, Upayogasanstha* and *Upayokta Vidhana. Vivrddhi* of *Ayu* consists of enabling the appropriate *Sanyoga* of the constituents of *Ayu* such as *Sharira, Indriya, Sattva* and *Atma*, and thereby prolonging it. The conversion of *Anna* into *Ahara Rasa* and *Mala* is done in *Amashaya* and the process is carried out by the *Agni* located below the *Amashaya*. To understand this mechanism a simile of rice being cooked in a vessel with the fire from below is given. For the digestion and metabolism to occur properly the *Aharaparinamakara Bhavas* must be appropriate.

The food which is composed of *Shadrasa* which is indicative of aptly consumed food immediately after consumption undergoes digestion and this stage of digestion is called *Avasthapaka*. This happens in three stages

1. *Madhura Avasthapaka*: This process takes place in the *Amashaya* which is the *Sthana* of *Kapha*. This by nature can extract the *Madhura Pradhana Rasa* in the *Ahara* which leads to the formation of *Kapha*. So, the food transforms into *Madhura Rasa* and leads to the formation of *Phenabhutakapha*. If the person consumes *Madhura Anuguna Rasa Ahara* then there will be formation of *Bahushleshma*.
2. *Amla Avasthapaka*: followed by the food undergoes *Amla Bhava* and in this state, food will be in *Vidagdhaavastha* and it leads to formation *Pitta* which is *Aghana*. These two stages take place in *Amashaya*. By this stage, the food is divided into *Ahara Rasa* and *Mala*. This also indicates that *PittaSthanasambandha* with the word *Vidagdhaahara* which further helps in understanding the pathology.
3. *Katu Avasthapaka*: Food is now propelled into *Pakvashaya* by *Vata* where the third stage of *Avasthapaka* takes place. *Vanhi* which is located in *Amashaya* now does the *Shoshana* of *Mala* and it now becomes *Paripinditaavastha* of *Mala* results in *Katubhava* which results in the formation of *Vayu.*

Relation between *Avasthapaka* and *Vipaka*

In the stage of *Avasthapaka*, the food comprising of six *Rasa* attains an overall predominance of *Kapha*, followed by *Pitta* and at last *Vata*. If so, then is it contradicting to the verse that explains about *Vipaka*? Does the *Avasthapaka* have an effect on the *Vipaka*? No, it is not contradictory. The *Avasthapaka* does not affect the *Nishthapaka*, because of the following reason. During the initial stage of digestion, each *Rasa* exerts it own effect, along with which *Avasthapaka* also occurs. If the food taken consists of *Madhura, Tikta,* etc.*rasa*, then each *Rasa* exerts its own effect, along with *Avasthapaka*. The difference lies in the following condition - During the 1st*Avasthapaka*, i.e., *Madhura*, the generation of more quantity of *Shleshma* is seen, if the food contains more of *Shleshma-Janaka Rasa*. On the other hand, if the food contains more of *Katu-Tikta,*

etc.*Rasa* which are opposite to *Shleshma*, then it leads to the generation of less quantity of *Kapha*. The similar logic is to be applied for *Pitta-Janakaavasthapaka* as well. The amount of *Dosha* which is produced depends on the food consumed. If the person consumes *Rasas* which are *Anuguna* (have an effect of generating *Kapha*) then more *Kapha* will be produced and vice versa. Therefore, the food which by nature undergo delayed digestion at the level of *Amla Avasthapaka* will produce more *Pitta* than others. This is the base for deciding the *Vidahiahara* which is one of the *Hetu* for *Pitta*. Even though, these *Avasthapaka* do not interfere in the action of *Vipaka* as they occur at different timeline but still the effect generated *Vipaka* with respect to *Dosha* may get altered like – the *Dosha* produced at *Avasthapaka* may be increased or decreased based on *Doshic* effect of *Vipaka*. Hence the knowledge of *Vipaka* and its effect on *Rasa* is essential.

The *Prasadabhaga* (essential and nutritive part) and *Malabhaga* (non-essential part) of *Ahara* which is called *Rasa* and *Kittam* respectively are formed. From *Kitta* the *Malas* like – *Mootra, Purisha, Vata*, are formed.

Action of *Dhatvagni*:

The *Ahara Rasa* carries each of the *Poshakaamsha* of each of the *Dhatus* which were formed at the time of the birth and constantly need to be nourished. This *Ahara Rasa* is now further digested by the *Dhatvagni* resulting in the *Dhatu Prasadabhaga* and *Malabhaga*. The *Dhatu Prasadabhaga* includes *Dhatus* like – *Rasa, Rudhira, Mamsa, Meda, Asthi, Majja, Shukra, Ojas, Panchaindriyadravya* and various *Avayava* like *Sharira Sandhi Bandha* and *Piccha* dietc are nourished. From the *Kittamsha* (Non – essential part of the food) *Mutra, Purisha* and *Vayu* will be formed. From the *Mala Dhatu Mala* like – *Shleshma, Pitta, Karna, Nakha, Akshi, Asya, Lomakoopa, Prajanana Mala, Sveda, Kesha, Shmashru, Nakha* and other *Avayava* are nourished.The *Panchendriya Dravya* in the form of *Pruthviadi* also gets nourished which are responsible for the existence of the respective *Indriya*. The food which is *Ishta* (liked) and *Hita* (good for health) in terms of the five senses respectively nourish the *Gandhadiindriyadravya*. *Sharira Bandha* refers to those structures that bind the tissues of the body like *Snayushira, Artava, Stanya,* etc[15].

Nyayas to understand this mechanism

Kedarakulya Nyaya

A part of the *Ahara Rasa* which is derived from the digestion of the food gets converted into the *Dhatu Roopa Rasa* and rest of it moves on to the nourishment of the next *Dhatu Shonita* and during this process acquires the properties of *Shonita* in terms of *Gandha* and *Varna* and a part of this that is similar to that of *Shonita* nourishes it and rest part of it moves on to the next *Dhatu*. The same continues with the rest of the *Dhatu*. The same has been justified by *Hareeta* as the *Ahara Rasa* undergoes changes in its colours from the time it moves from *Rasa Dhatu* till it reaches *Rakta* and this whole process takes a time of seven days. In this way for the formation of the entire *Dhatu* it takes a time of one month. A similar reference is obtained in *Suhsruta Samhita* also. The *Ahara Rasa* stays in each *Dhatu* for 3000 *Kala*.

Khale Kapota Nyaya

The *Ahara Rasa* travels in separate distinctive paths to nourish each of these *Dhatu* and the specific parts of *Ahara Rasa* which nourish specific *Dhatu* do not come in contact with other *Dhatu*. The order in which the nourishment takes place depends on the location of *Dhatu* and distance to be travelled. The *Uttorottara Dhatu* are farther and also *Srotas* through which the *Ahara Rasa* has to travel become more *Sookshma* and *Deergha* and hence there is delay in their nourishment. Hence *Ahara* Rasa containing the part which nourishes *Rasa Dhatu* travels through *Rasavahasrotas* and does nourishment. The *Ahara Rasa* which contains the part which nourishes *Rakta Dhatu* travels through *Raktaposhakamarga* and because it is farther and the *Srotas* are *Deergha* and *Sookshma* it takes more time than *Rasa Dhatu* nourishment. In the same way other *Dhatu* also need to be understood.

Nullyfying *Ksheeradadhi Nyaya* and Justification of *Kedarakulya Nyaya* and *Khalekapota Nyaya*

- If *Parinamavada* is accepted, then *Vrushyaprayoga* by entering the cycle of *Dhatu Parinamana* nourishes the *Shukra* after the formation of *Shonitadi Dhatus* which delays the process of *Shukrautpatti*. But in practice *Ksheeradi* is *Sadyavrushyakara*.
- As per *Khalekapota Paksha* the *Ahara Rasa* derived from the *Vrushyadravyas* because of their *Prabhava* reach *Shukra Dhatu* very quickly and therefore the nourishment of *Shukra Dhatu* is also quicker.
- If *Parinama Vada* is accepted then it is has to be accepted that if there is *Rasa Dhatu Dushti* then the eventual products like *Shonita* and other *Dhatus* will also undergo *Dushti* as they are derived from *Dushta Karana*.
- As per *KhaleKapota Paksha* if the part of *Ahara Rasa* which nourishes *Rasa Dhatu* is *Dhooshita* then only that *Dhatu* will be vitiated and not other because they are derived from different elements of *Ahara Rasa* which may not be affected.
- If *Parinama Paksha* is accepeted then in ***Medovrddhi***, *Asthi* which is derived from *Meda* also should be abundant in quantity but it is not seen instead there is other *Dhatu Kshaya*. In this way *Parinama Paksha* has many fallacies hence it is not acceptable. Hence the theory where every *Dhatu* undergoes transformation to yield the consecutive *Dhatu* is unacceptable. If this is accepeted then a person with 3–4 *Upavasa* person would die or with *Upavasa* of a month body would be containing only *Shukra* and nothing else. This is illogical.
- *Khalekapota Nyaya* and *Kedarakulya Nyaya* have equal weight in their argument.
- Effect of *Vrushyadravya* by *Kedarakulya Nyaya*: because of *Prabhavavrushyadravya* reach *Shukra Dhatu* by bypassing the *Raktadi Dhatu* and show the *Vrushyaprabhava* just like how *Pravbhava* works in *Khalekapota Paksha*.
- Concept of Specific *Dhatu Dushti*: it is because of *Dushti* in the specific part of *Ahara Rasa* which is intended to nourish specific *Dhatu* leading to *Dushti* in that specific part[16].

Action of *Bhutagni*

Bhutagni – Akashabhutagni, Vayubhutagni, Tejobhutagni, Jalabhutagni and *Prithvibhutagni* are contained in their respective *Dravya* (food). They are activated when the *Agni* present in the body is kindled and stimulated. Thereby, they each metabolise the respective contents based on their specific *Guna*. From *Sutrasthana*, this talks about the fate of food content on encountering Agni. Here, the *Bhutagni* present in the food is activated with the help of *Jatharagni* and thereby enables digestion by recognizing their respective *Gunas*. Although it is implied that the *Dravya* undergoes digestion with the *Bhutagni*, it is to be inferred that, due to the digestion of the *Parthivadidraya*, the *Parthivadiguna* are generated. This lays emphasis on the importance of understanding digestion at the level of *Guna*, through the phrase "*Parthivadinaharagunanjanayanti*" thereby, after the action of *Agni*, the *Guna* remains, and not the *Dravya*. The *Dravya* is acted upon by the *Agni* to generate *Guna*. The word *Aharagunan* can also be split as *Ahara* and *Guna*, so that the word *Ahara* can indicate '*Dravya*. Therefore, *Jatharagni* is responsible for overall digestion and metabolism of food contents and the subsequent generation of *Ahara Rasa* and *Vipaka*, while the *Bhutagni* performs the function of metabolising based on the *Guna* and thereby giving rise to an increase in those *Guna*. This is reflected in the following statement – this action of *Bhutagni* is also seen in the *Dhatu,* when the nutrients move from one *Dhatu* to another. This is because, five *Bhuta* are witnessed in the *Dhatu* as well. Here, the action of both *Dhatvagni* and *Bhutagni* are seen, like the action of *Jatharagni* and *Bhutagni*. The *Parthivabhutagni* enables the transformation of its own specific respective *Dravya* and *Guna* in the body and gives nourishment to that very *Dravya* and *Guna*. By the action of five *Bhutagni* on their respective *Bhoutikaaharaguna* and they will be nourished.

Based on the requirement of the body in accordance with age and constitution of the individual the degree of nourishment is done by the body. In this way after undergoing proper digestion the *Ahara Rasa* which gets formed in the required quantity and neither less nor more travels through the *Dhamani* and undergoes further digestion at the level of the *Dhatus* resulting in *Sama Dhatu* and *Sama Mala* follow the healthy (*Sama Dhatohoashrayasya*) state pre–existing in the *Sharira* or *Ashraya* (Healthy state pre-

existing and kind of nutrients). *Malas* also have been named as *Dhatu* as they sustain the body when they are present in the required range. This entire process of digestion is always continuous just like a wheel which never stops but the pace with which continues depends on the efficiency of all the factors involved in it.

Due to any pathological conditions (*Nimitta*) other than *Arishtalakshana*, if there is any *Vruddhi* or *Kshaya* in the *Dhatus* is seen it will be managed by *Ahara Rasa* itself. Any kind of the *Kshaya* or *Vruddhi* of the *Prakruta Dhatu* for the sake of *Samya* is not accepted. This kind of *Samya* leads to *Roga*. The same rule applies for the *Mala* also. The *Vruddhi* or *Kshaya* of *Mala* must be balanced with *Ahara* itself and the *Samya* which leads to *Arogya* has to be maintained with proper *Mana* of *Mala*. If there is *Vruddhi* of *Mala* then one has to follow –

- *Utsarga – Shodhana*
- *Sheetoshnaparyaya Chikitsa – Gunaviparyaya Chikitsa* has to be followed
- *Nidanaparivarjana*[15-16]

Ahara Rasa Vs *Rasa Dhatu*

If a distinct existence of *Ahara Rasa* is accepted other than *Rasa Dhatu* then what is *Sthana* and *Pramana* of it as there is no description of that in the text?

As there is no definite quantity of *Ahara Rasa* being formed and it constantly increases and decreases as the quantity of the food also varies hence the definite quantity has not been mentioned in the text.

The *Sthana* of *Ahara Rasa* is *Dhamani* which are related to these *Dhatus* and therefore there is no separate *Sthana* mentioned for it other than these *Dhatus*.

The following criteria need to be fulfilled for the nourishment of the body leading to proper growth and development.

1. The *Ahara Rasa* which is formed should get transported from one *Dhatu* to another without any interruption. If there is any obstacle for the transportation of the *Ahara Rasa* due to any pathological condition then it does not result in proper nourishment and thereby affecting the growth and development.
2. The *Agni* present in each *Dhatu* should be functioning properly. Even if anyone of *Dhatu Poshaka Agni* gets hampered then the entire process of digestion and metabolism gets hampered.
3. The *Vyanavata* which is responsible for carrying the *Ahara Rasa* containing *Dosha* and the *Poshakaamsha* of each *Dhatus* to each of these *Dhatuvagni* should be functioning properly. If the function of the *Vyanavata* gets affected then the process of digestion and metabolism gets disturbed. If the movement of *Vyanavata* gets hampered because of fault in any of the *Srotas* then the *Dosha* which are being carried by *Vyanavata* get stagnated and lead to the diseases which are mentioned as *Pradoshajavyadhi* for that particular *Dhatu*.
4. The *Srotas* through which the *Ahara Rasa* is carried to each of these *Dhatvagni* should be functioning properly if not then it affects the digestion and metabolism. These *Srotomukhaayana* is specific to each *Dhatu* and has the respective name. These *Srotas* carry the specific nutrients to the specific *Dhatus* and nourish them. And in a healthy individual the amount of nutrients provided depends on the requirement of *Dhatu*. But in diseased condition this mechanism does not exist and hence it has to be corrected.
5. The Spiritual health of the individual should be good to get these benefits.

By the disturbance in these factors, the *Dhatus* do not get nourished and therefore the growth and development also gets affected. Body is the by-product of the food. All the *Dhatus* which sustain the body are resultant of the *Ahara Rasa* and therefore the constant conversion of *Ahara Rasa* into the *Dhatu* which are under constant depletion is the only way to maintain health and there is no other alternative to it.

The general causative factors for the disturbance of *Agni* and the cause for occurrence of various diseases are enlisted here. *Kala* indicates *Nityagakala*. *Deshavaishamyam* indicates disturbed *Desha*, which has been explained in *Janapadodhwansaniyavimanam*. - *Ritu* indicates seasons like *Shishira*, etc *Kalavaishamya* implies that all the *Ritus* have been deranged, but *Rituvaishamya* implies the derangement of only one or two *Ritushuktatva* indicates a sour quality the undigested food is compared to *Visha*, as *Visha* can lead to several diseases and complications. *Abhojana* – being devoid of food, *Ajirnabhojana* – consuming food before the previous meal is digested, *Atibojana* – consuming higher quantity of food than required, *Vishamashana* – consuming inappropriate quantity of food (more quantity or less) at erratic timings, consuming non-conducive, *Guru, Sheeta, Atiruksha,* and vitiated food, complications arising due to inappropriate administration of *Virechana, Vamana, Snehapana*, chronic diseases, badly afflicted *Desha*, derangement of all or some seasons and holding of natural urges are the causes of vitiation of *Agni*. Such deranged *Agni* becomes incapable of digesting even light food, and therefore the food contents become only partially digested. This undigested content attains a state of sourness and act like *Visha*, which gives rise to several diseases and complications[16].

Concept of *Santarapneeyamadhyayam*

The *Shadupakramas* which have been classified into two namely *Santarpana* and *Apatarpana* which will be used in two areas and hence the chapter has been named as *Santarapneeyamadhayayam*.

Derivation of the word *Santarpana*:

The one which brings *Sam–Tarpana* is called *Santarpana*.

The word *Santarpana* is derived from the word "*Trupa*" which indicates both *Sandeepana* and *Preenana*.

Santarpanahetu:

1. Diets which are prominent of qualities like
    - *Madhurarasa*
    - *Snigdha*
    - *Guru*
    - *Picchila*
2. *Navanna* (crops which have not crossed one year after harvesting)
3. *Navamadya*
4. *Mamsa* derived from animals belonging to the category of *Anupa*
5. *Mamsa* derived from animals belonging to the category of *Varija*
6. *Gorasa* (products of milk)
7. *Goudika* (products of jaggery)
8. *Pishtanna* (preparations involving flour)
9. *Cheshtadweshi*: One who does not like any sort of physical activity
10. *Divasvapna*: One who has the habit of sleeping in the day time
11. *Shayya Asana Sukha Rata*: One who always has the tendency towards physical comfort like laying down or sitting for long hours.

Table No. 4 List of dietary Items and their effect on *Dosha*

Sl No.	Dietary Item	Properties	Effect on *Dosha*
1.	*Navanna* (crops which have not crossed one year after harvesting)	*Ruksha Guru*	-
2.	*NavaMadya*	*Guru*	*Tridhosha Karaka*
3.	*Mamsa* derived from animals belonging to the category of *Anupa*	*Guru Ushna Snigdha*	*Kapha Pitta Vardhana Vatahara*
4.	*Mamsa* derived from animals belonging to the category of *Varija*	*Guru Ushna Snigdha*	*Kapha Pitta Vardhana Vatahara*
5.	*Gorasa* (products of milk)	*Guru*	*Kapha Vardhaka*

6.	*Goudika* (products of jaggery)	*Snigdha Sheeta Sara*	*Kapha Vardhaka*
7.	*Pishtanna* (preparations involving flour)	*Guru*	*Tridoshakara*

Table No. 5 Lifestyle / Activities and their effect on *Dosha*

Sl No.	Lifestyle / Activities	Effect on *Dosha*	Effect on *Dhatu*
1.	*Cheshtadweshi*: One who does not like any sort of physical activity	*Kapha Kara*	*Meda*
2.	*Divasvapna*	*Kapha Pitta Kara*	*Meda*
3.	*Shayya Rata*	*Kapha Kara*	*Meda*
4.	*Sukha Rata*	*Kapha Kara*	*Meda*
5.	*Asana Rata*	*Kapha Kara*	*Meda*

Santarpanajanyavyadhi*: Diseases and Conditions occurring because of *Santarpana

1. *Prameha*
2. *Pidaka*
3. *Kotha*
4. *Kandu*
5. *Pandvamaya*
6. *Jvara*

7. *Kushta*
8. *Amapradosha*
9. *Mutrakrchra*
10. *Arochaka*
11. *Tandra*
12. *Klaibya*
13. *Atisthoulya*
14. *Alasya*
15. *Gurugatrata*
16. *Indriyalepa*
17. *Srotolepa*
18. *Buddhi Moha*
19. *Prameelaka*
20. *Shopha*[17]

Samprapti of Santarpana Janya Vyadhi

Pandu

In take of *Nidana* causes vitiation of *Doshas*, predominant with *Pitta Gunas*, throughout the *Dhatus*. Due to this, the *Dhatus* lose integrity and function, leading to generation of *Shaithilya* and *Gaurava* (inability to carry out daily activities). With further progress in the *Samprapti*, there is further *Dosha Dushya Sammurchhana* into deeper *Dhatus*. Due to compromised nutrition in the *Dhatus*, *Ojas* is also not formed, thereby leading to a severe decrease in *Varna*, *Bala*, *Sneha* and other qualities of *Ojas*. As a result, the reduction of *Sneha Gunas* responsible for integrity of the *Dhatus* causes decrease in the *Saarata*, *Rakta* and *Medas* leading to decrease in the functioning of *Indriya* and *Vaivarnya*. There is extensive derangement of *Dosha* and *Dushya* in *Pandu*, leading to a complete derangement in the function of the *Dhatus* and *Indriyas*.

Here, the *Pittavrddhi* does not cause subsequent R*akta Vrddhi* as expected due to *Ashraya Ashrayi Bhava*, because the *Raktaposhaka Rasa Dhatu* is disturbed by *Pittam*, due to which there is compromise in the nourishment to *Rakta*, thereby leading to *Raktakshaya*. Similarly, since there is derangement in the *Pitta Guna*, there is also a reduction in its function which causes *Vikrta Varna*[18].

Jwara

Jwara can occur if and only if the *Prakupitavata* localises in the *Amashaya* and affects the *Agni*. The word '*Yada*' is indicative of this case. The *Dushta Dosha* which is potentially responsible for the causation of *Jwara* invariably causes *Dushti* of the *Amashaya*. Despite the fact that *Amashaya* is the *Sthana* for *Agni* and the disturbance of it reflects on the *Agni* as well, the phrase *Ushmanasahamishribhuya* is mentioned to show that the *Grahanirupavahnisthana* need not always be involved in case of the disturbance of merely the *Amashaya*. *Amashaya* has to be understood as the *Ekadesha Vahnisthana*. *Rasanamanam* can also mean the *Madhuradi Rasa* in the *Ahara*, therefore it is described as *Aharaparinamadhatu*, which is the result of *Ahara Parinama*. But all the seven *Dhatu* are a result of *Ahara Parinama*, which is why the word *Adya* is used to indicate the first *Dhatu* formed out of *Ahara Parinama*.

With the phrase *Ushmanam Nirasya*, it can be understood that *Agni* is displaced from its *Sthana* of function, but despite this the word *Paktisthanat* is used to indicate that the *Agni* is completely displaced from its site of function. It is rendered powerless due to the displacement of its *Ushmata*. Thereby, *Pittam*, which has the property of burning similar to the *Bahyavahni*, is excluded.

This is the reason that *Agnimandya* is seen in *Jwara*. The factor responsible for the displacement of *Agni* from its site of function is *Vata*, which is deranged and thereby ceases to act as a mere kindler, but completely extingushes it and carries the heat throughout the body[19].

Kushtha

The loss of integrity of the *Tvak Aadi Dhatu* becomes a favourable condition for the *Dosha* to take shelter and undergo derangement. Therefore, they attain *Sthirata*, i.e., they settle down in the other *Sthana* to cause *Kusihta*. It has to be understood that

circulating *Doshas* are incapable of generating *Kushta*. Intake of *Nidana* causes the *Prakopa* of *Pitta* and *Shleshma*. This causes derangement in the functioning of *Vata,* which leads to its increase. These three *Doshas* thereby circulate through the *Siras* in the *Shaakha,* etc. (*Tiryak*) and reach the *Bahyamarga*. Thereby they circulate to the entire body. They may settle down at certain places near the *Tvak,* to cause skin lesions such as *Mandala*. If not treated, they cause the derangement of the deeper *Dhatu*. They move from external pathways to the internal pathways[20].

Mutrakrcchram

Doshas due to their own specific *Hetu* undergo *Prakopa,* all together or individually in the *Vasti*. Therefore they cause the derangement in the pathway of urine leading to obstruction in urine[21].

Tandra

Due to increased *Tamoguna* and lack of focus and awareness, *Tandra* is characterized by Inability to process the *Indriyartha*, *Gauravam* – heaviness of the body, *Jrmbhanam* – yawning *Klama* – fatigue, caused without any physical activity, *Nidrartasya*– drowsiness[22]

Buddheh mohah

Doshas undergo *Prakopa* altogether or one by one in *Raktavaha, Rasavaha* and *Sangnyavahasrotas*. The cause of this is as follows:

1. *Malina Ahara Sheela* – not having good eating habits.
2. *Rajo Moha Avrta* – increase of *Rajo Guna* causing confusions and lack of awareness.

The *Doshas* settle down therein and cause the obstruction of these *Srotamsi* to lead to *Vyadhi* such as *Mada, Mrcchha* and *Sanyasa,* which are increasingly severe[23].

Treatment Principle for *Santarpanajanyavyadhi*:

In the above said conditions *Vamana* therapy is said to be suitable along with *Virechana, Vyayama, Upavasa, Raktamokshana, Dhoomapana Svedana* and *Churna Pradeha*. One should also advise intake of *Ruksha Anna*. All the formulations which are said to subside *Kandukotha* are also beneficial.

Formulations

1. *Hareetakichurna* along with *Madhu* in the form of *Lehana*.
2. The *Kwatha* of *Triphala, Aragvadha, Patha, Saptaparna, Vatsaka, Musta, Madana, Nimbi*. This is said to subside the diseases like *Meha* etc. which occur because of *Santarpana* when used continuously with a due consideration to the *Matra* and *Kala*.
3. The *Kwatha* of *Musta, Aragvadha, Patha, Triphala, Devadaru, Shvadamshtra, Khadira, Nimba, Haridra, Vatsakatvak* should be administered in empty stomach in the early morning to get rid of the diseases occurring because of *Santarpana*. The same drugs can be used for the purpose of *Udvartana, Udgharshana, Snana*, etc. to get rid of the diseases pertaining to skin. Based on the condition the same drugs can be processed with *Sneha* and administered in diseases pertaining to skin.
4. *Kushtha, Gomedaka, Hingu, Krounchasthi, Tryooshana, Vaca, Vrushaka, Ela, Shvadamshtra, Kharahva, Ashmabhedaka* when administered with either *Takra* or *Dadhimanda, Badaramla* can subside diseases like *Mutrakruchra* and *Prameha*.
5. The regular use of *Hareetakichurna* along with *Takra* or *Triphalachurna* with *Takra* or regular use of *Arishta* will subside diseases like *Meha*, etc.
6. The *Mantha* prepared with *Tryooshana, Triphala, Vidanga, Ajamoda* and *Madhu* is beneficial.
7. The *Saktu* or *Taila* prepared with the above said drugs and consumed with *Agarukvatha* is beneficial.
8. The *Churna* of *Vyosha, Vidanga, Shigru, Tripahala, Katurohini, Brihati, Kanthakari, Haridra, Daruharidra, Patha, Ativisha, Sthira, Hingu, Kebukamoola, Yavani, Dhanyaka, Chitraka, Souvarchala, Ajaji* and *Hapusha* is mixed with equal quantities

of *Taila, Ghrta, Kshoudra* and 16 parts of *Saktu* and should consume. Diseases occurring because of *Santarpana* will get subsided by this along with diseases like – *Prameha, Moodhavata, Kushtha, Arshas, Kamala, Pleeha, Panduroga, Shopha, Mootrakruchra, Arochaka, Hridroga, Rajayakshma, Kasa, Shvasa, Galagraha, Krimiroga, Grihanidosha, Shvitra, Atisthoulya*. It helps in improving *Agni*, *Smruti*, and *Buddhi*.

9. The individual who does exercise daily and consumes *Yava* and *Godhuma* daily will be devoid of the disturbances caused by *Santarpana* and will get rid of *Sthoulya*[17].

Table No. 6 *Guna Karma* of the drugs used in *Santarpana Janya Vyadhi Chikitsa*

Sl No.	***Drug***	***Rasa***	***Guna***	***Veerya***	***Vipaka***	***Doshaghnata***	***Karma***
1.	*Hareetaki* Terminalia chebula	*Kashaya Pradhana*	*Laghu Ruksha*	*Ushna*	*Madhura*	*Tridosha shamaka*	*Rasayana, Medhya, Anulomana, Pramehahara,Vranahara, Vayasthapaka*
2.	*Madhu*	*Kashaya Madhura*	*Guru Ruksha Yogavahi*	*Sheeta*	*Madhura*	*Vatakara Rakta, Pitta Kapha Hara*	*Deepana, Varnya, Swarya, Lekhana, Grahi Vrana Ropana*
3.	*Triphala*	-	*Sara*	-	-	*Kapha Pitta Shamaka*	*Mehaghni, Kushtaghna, Deepana, Chakshushya Rasayana*
4.	*Aragvadha* Cassia fistula *Phalamajja* / Fruit pulp	*Madhura Tikta*	*Guru Snigdha*	*Ushna*	*Madhura*	*Vatapitta Shamaka Pittakapha Samshodhaka*	*Sramsana, Krimighna Pramehaghna Jwaraghna Kushtaghna*

5.	*Patha* Cissampelos pareira	*Tikta*	*Laghu Teekshna*	*Ushna*	*Katu*	*Vata Kapha Shamaka*	*Jwarahara*
6.	*Saptaparna* Alstonia scholaris	*Tikta Kashaya*	*Laghu Snigdha Sara*	*Ushna*	*Katu*	*Kapha Pitta Shamaka*	*Kushtaghna,*
7.	*Vatsaka Twak* Holerrhena antidysenterica	*Tikta Kashaya*	*Laghu Ruksha*	*Sheeta*	*Katu*	*Pitta Kapha Shamaka*	*Sthambhaka Arshoghna Trishna Nigrahana Ama Pachaka*
8.	*Musta* Cyperus rotundus	*Tikta Kashaya Katu*	*Laghu Ruksha*	*Sheeta*	*Katu*	*Pitta Kapha Shamaka*	*Sheeta grahi Jwarahara Trishnahara Deepana Pachana*
9.	*Madana Randia Dumetorum*	*Tikta Madhura Katu Kashaya*	*Laghu Ruksha*	*Ushna*	*Katu*	*Kapha Vata Shamaka Pitta Saraka*	*Vamaka Lekhana Vranahara Vidhradihara Kushtaghna Jwarahara Shophahara*
10.	*Nimba* Azardirachta indica	*Tikta Kashaya*	*Laghu Ruksha*	*Sheeta*	*Katu*	*Pitta Kapha Shamaka*	*Trishnahara Mehahara Krimighna Kushtaghna Shramahara Jwaraghna*
11.	*Devadaru* Cedrus *deodara*	*Tikta Katu Kashaya*	*Snigdha Laghu*	*Ushna*	*Katu*	*Vata Kapha Shamaka*	*Vibandha Hara Tandrahara Jwarahara Mehahara Arsha Kandu*
12.	*Shvadamshtra* Tribulus terristris	*Madhura*	*Guru Snigdha*	*Sheeta*	*Madhura*	*Tridosha Shamaka*	*Shotahara Mutrala Basti Shodhaka Balya Deepana*

							Pramehahara
13.	*Khadira* Acacia Catechu	*Tikta Kashaya*	*Laghu Ruksha*	*Sheeta*	*Katu*	*Kapha Pitta Shamaka*	*Kushtaghna Kandughna Dantya Medohara Pramehahara Pachana Jwaraghna Shothahara*
14.	*Haridra* Curcuma longa	*Tikta Katu*	*Ruksha Laghu*	*Ushna*	*Katu*	*Kapha Pitta Shamaka*	*Twachya Pramehahara Shotha hara Vishaghna*
15.	*Vatsakatvak*	*Tikta Kashaya*	*Laghu Ruksha*	*Sheeta*	*Katu*	*Pitta kapha shamaka*	*Sthambhaka Arshoghna trishna nigrahana ama pachaka*
16.	*Kushtha*	*Tikta Katu Madhura*	*Laghu Ruksha Teekshna*	*Ushna*	*Katu*	*Kapha Vata Shamaka*	*Kushtaghna Vatarakt Nashaka Vishaghna Visarpahara Kasahara*
17.	*Hingu*	*Katu Tikta*	*Laghu Tikshna*	*Ushna*	*Katu*	*Vata Kapha Shamaka*	*Pachana Ruchya Krimighna Swasahara Chakshushya*
18.	*Tryooshana*	*Katu*		*Ushna*	*Katu*	*Vata Kapha Shamaka*	*Deepana Shulahara Medorogaghna Pramehaghna*
19.	*Vaca*	*Katu Tikta*	*Laghu Tikshna*	*Ushna*	*Katu*	*Kapha Vata Shamaka*	*Medhya Lekhana Mutra Vishodhaka Bhutaghna Kantya*

							Pachana
20.	*Vrushaka*	*Tikta Kashaya*	*Laghu Ruksha*	*Sheeta*	*Katu*	*Kapha Pitta Shamaka*	*Swasahara, Kasahara Swarya Rakta Pittahara Mehahara Trishna Shamaka Kamalahara Jwaraghna*
21.	*Ela*	*Katu*	*Laghu Ruksha*	*Ushna*	*Katu*	*Vata Kapha Shamaka*	*Rochaka Trishnahara Swasahara Kandughna*
22.	*Kharahva*	*Katu Tikta*	*Laghu Ruksha Teekshna*	*Ushna*	*Katu*	*Kapha Vatahara*	*Deepana Ruchikara Bastirujahara Hikka Nigrahana Chardi Nigrahana*
23.	*Ashmabhedaka*	*Kashaya Tikta*	*Snigdha*	*Sheeta*	*Katu*	*Tridosha Shamaka*	*Ashmarighna Bastishodhaka Pramehahara Mutrala Dahahara*
24.	*Vidanga*	*Katu Kashaya*	*Laghu Ruksha Tikshna*	*Ushna*	*Katu*	*Kapha Vata Shamaka*	*Ruchya Vishaghna Medohara Mehahara Rasayana*
25.	*Ajamoda*	*Katu Tikta*	*Laghu Ruksha Tikshna*	*Ushna*	*Katu*	*Kapha Vata Shamaka*	*Deepana Ruchikara Bastirujahara Balya*
26.	*Agaru*	*Katu Tikta*	*Teekshna Laghu Snigdha*	*Ushna*	*Katu*	*Tridoshahara*	*Sheetapanayana Twachya Karna rogahara*

27.	*Vyosha*	*Katu*		*Ushna*	*Katu*	*Vata Kapha Shamaka*	*Deepana Shulahara Medorogaghna Pramehaghna*
28.	*Shigru*	*Katu*	*Ruksha Teekshna Sara Laghu*	*Ushna*	*Katu*	*Vata Kapha Shamaka*	*Deepana Rochana Sangrahi Vidahakara Medohara Mukha jadya hara*
29.	*Katurohini*	*Tikta*	*Ruksha Laghu*	*Ushna*	*Katu*	*Pitta Shamaka*	*Bhedana Deepana Pramehahara Daha hara Swasahara Kasahara*
30.	*Brihati*	*Katu Tikta*	*Laghu Ruksha Teekshna*	*Ushna*	*Katu*	*Kapha Vata Shamaka*	*Kasahara Pachana Ruchikara Kantya Agni deepana*
31.	*Kanthakari*	*Tikta Katu*	*Laghu Ruksha*	*Ushna*	*Katu*	*Kapha Vata Shamaka*	*Pramehahara Kasahara Swasahara*
32.	*Daruharidra*	*Tikta*	*Laghu Ruksha*	*Ushna*	*Katu*	*Pitta Kapha Shamaka*	*Netrya Pramehahara Rakta shodhaka Yakrut uttejaka Vranahara*
33.	*Patha*	*Tikta*	*Laghu Teekshna*	*Ushna*	*Katu*	*Vata Kapha Shamaka*	*Jwarahara*
34.	*Ativisha*	*Katu Tukta*	*Laghu Ruksha*	*Ushna*	*Katu*	*Kapha Pitta Shamaka*	*Deepana pachana Kasahara Krimighna Jwarahara*

35.	*Sthira,*	*Madhura Tikta*	*Guru Snigdha*	*Sheeta*	*Madura*	*Vata Pitta Shamaka*	*Brahmana Rasayana Kasahara Chardighna Mehaghna Shophahara*
36.	*Hingu*	*Katu Tikta*	*Laghu Teekshna*	*Ushna*	*Katu*	*Vata Kapha Shamaka*	*Pachana Ruchya Krimighna Swasahara Chakshushya*
37.	*Kebukamoola*	*Tikta Kashaya*	*Laghu Ruksha*	*Sheeta*	*Katu*	*Pitta Kapha Shamaka*	*Grahi Krimighna Jwarahara Kushtaghna*
38.	*Yavani*	*Katu Tikta*	*Laghu Ruksha Teekshna*	*Ushna*	*Katu*	*Vata Kapha Shamaka*	*Shulaprashamana Deepana Pachana Udararogahara Anaha Nashaka Hridya Chardi Nigrahana*
39.	*Dhanyaka*	*Madhura Kashaya Tikta*	*Snigdha Laghu*	*Ushna*	*Madhura*	*Kapha Vata Shamaka*	*Ruchya Deepana Mutrala Jwaraghna*
40.	*Chitraka*	*Katu*	*Tikshna*	*Ushna*	*Katu*	*Vata Kapha Shamaka*	*Deepana Pachana Arshoghna Kushtaghna Grahi*
41.	*Souvarchala*	*Lavana*	*Laghu Tikshna*	*Ushna*	*Madhura*	-	-
42.	*Ajaji*	*Katu*	*Laghu Ruksha*	*Ushna*	*Katu*	*Vata Kapha Shamaka*	*Depana Garbhashaya Shodhaka Jwaraghna*

43.	*Hapusha*	*Katu tikta*	*Guru*	*Ushna*	*Katu*	*Vata Kapha Shamaka*	*Deepana Shulahara Ashroghna*

Apatarpanajanyavyadhi*: Diseases and Conditions occurring because of *Apatarpana

- Decrease in the *Dehabala, Agnibala, Varna, Oja, Shukra, Mamsa*
- Conditions like *Jvara, Kasa* associated with *Parshvashula* and *Arochaka*
- *Shrotradourbalya*
- *Unmada*
- *Pralapa*
- *Hridvyatha*
- Obstruction for *Vit*and *Mutra*
- *Shula* in *Jangha, Uru, Trika*
- Pain in the smaller and bigger joints
- Diseases pertaining to *Vata* like *Urdhvavata*

Treatment Principle for *Apatarpanajanyavyadhi*:

In the above said conditions one must administer *Santarpana* line of treatment. If the person is fit for immediate *Santarpana* then in him *Sadyosantrapna* must be given otherwise, then it should be gradually brought into practice. The individual who has undergone an acute depletion of *Dhatu* (*Sadyaksheena*) he should be treated administered *Tarpana* (therapy which helps in the nourishment of *Dhatus*) which is quick in its effect (*Sadyo*) but this may not be applicable to an individual who has undergone a chronic depletion of *Dhatu* (*Chiraksheena*) and in them the process of doing *Tarpana* should be gradual (*Santarpanaabhyasa*) as in these individuals the effect of *Tarpana* depends on the factors like – *Deha, Agni, Dosha, Bhaishajya, Matra* And *Kala*. Hence one should not

try to administer *Tarpana* immediately in *Chiradurbala*. If one tries to do *Sadyatarpana* in such individual it may cause *Agnidushti*.

Method of doing *Tarpana*:

1. *Mamsa Rasa* is beneficial along with *Ksheera* and *Ghrita*.
2. Therapies like *Basti, Abhyanga*, which can have an effect of *Tarpana*.

Conditions where *Tarpana* is useful

1. *Jvara* which is chronic
2. *Kasa* which is chronic
3. *Krusha*
4. *Mutrakruchra*
5. *Trushna*
6. *Urdhvavata*

Formulations

1. Equal quantities of *Sharkara, Pippali, Taila, Ghrta, Kshoudra* and double the quantity of *Saktu* and one should prepare *Mantha* which is *Vrushya* by effect.
2. *Saktu* along with *Madira* and *Sharkara* is useful in conditions like *Vatadoshadushti* and helps in *Anulomana* of *Vin, Mutra, Kapha* and *Pitta*.
3. *Saktu* along with *Phanita, Sarpi, Dadhimanda* and *Amla Kanjika* are useful in conditions like *Mutra Kruchra* and *Udavarta*
4. Mantha prepared with *Khrjura, Mridvika, Vrukshamla, Amlika, Dadima, Parushaka* and *Amalaka* is beneficial in diseases occurring because of intake of *Madya*.
5. Use of *Rasa* like *Svadu* and *Amla* processes with *Sneha* and use of *Rookshadravya* like *Saktu* in the form of *Mantha* brings stability and increases *Varna, Bala*. Here *Amla Dravya* includes use of drugs like *Dadima* and *Amalaka*, etc[17].

Concept of *Santarapana*

Upakrama

According to Charaka *Upakrama* are of six types[24] namely –

1. *Langhana*
2. *Brumhana*
3. *Rukshana*
4. *Snehana*
5. *Svedana*
6. *Stambhana*

According to *Vagbhata Upakrama* fundamentally can be classified into two[2] as follows –

1. *Santarpana*
2. *Apatarpana*

Upakrama are considered as two because of the two kinds of patients which are clinically presented namely *Santarpanotha* and *Apatarpanotha* i.e., individuals suffering with *Santarpanothavyadhi* and *Apatarpanothavyadhi* or those with *Sama Dosha* conditions and *Niramadosha* conditions. The other four therapies namely *Snehana, Rukshana, Svedana, Stambhana* are also categorized under these two based on the *Bhoutik* composition[25].

Synonyms[2]

Santarpana is also called as *Brumhana*

Apatarpana is also called as *Langhana*

Definition

Brumhana: The one which increases the bulk of the body or increases the weight of the body is called *Brumhana.*

Langhana: The one which brings lightness in the body or decreases the weight of the body is called *Langhana*[2].

Definition of *Shadupakrama*:

1. *Langhana:* The one which brings lightness in the body or decreases the weight of the body is called *Langhana.*
2. *Brumhana:* The one which increases the bulk of the body or increases the weight of the body is called *Brumhana.*
3. *Rookshana*: The one which brings dryness, roughness and cleanses the body is called *Rookshana.*
4. *Snehana*: The one which brings the effect of unctuousness, liquification, and softness and increases the moisture in the body is called *Snehana*.
5. *Svedana:* The one which subsides stiffness, heaviness, sense of cold and induces sweating is called *Svedana*.
6. *Stambhana:* The one which helps in stopping the movement (either subtle (*Chala – KinchitGatimantam*) or gross (*gantimantam – PravyaktaGatimantam*)) of component of the body is called *Stambhana*[26].

Classification of *Langhana or Apatarpana*

- *Shodhana*
- *Shamana*

Definition

Shodhana: the one which eliminates the *Dosha* out of the body is called *Shodhana.*
Shamana: the one which does not eliminate the *Dosha* out of the body and brings the balance of *Dosha* inside the body by either increasing the *Dosha* which is reduced or decreases the *Dosha* which is increased without disturbing the balanced *Dosha* already present.

Shodhana is of five types namely,

- *Niruha*

- *Vamana*
- *Kayareka*
- *Shiroreka*
- *Asravisruti*

Shamana is of seven types as follows:

- *Pachana*
- *Deepana*
- *Kshuta*
- *Trushna*
- *Vyayama*
- *Atapa*
- *Maruta*

Bhoutik Composition

Santarpana/ Brumhana – Bhouma and *Apa*

Apatarpana / Langhana – Agni, Vayu and *Akasha*.

Indications of *Brumhana*

1. *Kshataksheena / Kshata* and *Ksheena:* Individuals in whom there is *Dhatu Kshaya* because of *Urakshata*
2. *Krusha*: Individual who is emaciated
3. *Bheshajakarshita*: Individuals emaciated because of medicine
4. *Madyakarshita*: Individuals emaciated because of alcohol
5. *Streekarshita*: Individuals emaciated because of intercourse
6. *Shokakarshita*: Individuals emaciated because of sorrow
7. *Shosha, Arsha, Grahanidoshakarshita/ Vyadhikarshita*: Individuals emaciated because of disease
8. *Durbala:* Individuals who have become weak
9. *Garbhini*: Pregnant

10. *Sutika*: Women who has recently delivered
11. *Bala*: Children
12. *Vruddha*: Elderly
13. *Ruksha*: Individuals in whom *Rukshata* has increased
14. *Nityamadhvaga/ Adhva*: Individuals who have become weak because of the habit of excessive walking
15. *Stree Nitya*: Individuals who indulge in excessive sexual intercourse
16. *Madyanitya:* Individuals who consume excessive alcohol
17. *Bhara*: Individuals who have become weak because of the habit lifting heavy weights
18. *Vatala*: Individuals of *Vatala Prakruti*
19. *Greeshme*: In general, everyone in *Greeshma Ritu*

Method of administering *Brumhana*:

1. *Mamsa: Mamsa Rasa* of *Mamsada* or *Kravyadamamsa* is said to be the best way to do *Brumhana*. The criteria for the selection of *Mamsa* the qualities of the animal have been explained as follows -
    a. *Adigdhaviddha* – the one which is not killed by a poisonous arrow
    b. *Aklishta* – the one which is not distressed
    c. *Vayastha* – the one which is young
    d. *Satmyachari* – the one which wanders around *Satmya Desha*
    e. *Mruga Matsya* and *Vihanga*
2. *Ksheera*
3. *Sita*
4. *Sarpi*
5. *Madhura* and *Snigdha Basti*
6. *Svapnasukha*
7. *Shayyasukha*
8. *Abhyanga*

9. *Snana*
10. *Utsadana*
11. *Nirvrutti*
12. *Harshana*

Indications of *Langhana*

1. *Prameedhanam / Mehadosha*
2. *Amadosha*
3. *Atisnigdha*
4. *Abhishyandi*
5. *Brumhanam*
6. *Jvara*
7. *Urustambha*
8. *Tvagdoshinam / Kushtha*
9. *Visarpa*
10. *Vidradhi*
11. *Pleeha*
12. *Shiroroga*
13. *Kantharoga*
14. *Akshiroga*
15. *Sthula*
16. In general, everyone in *Shishiraritu* and *Hemantha* even in the conditions of *Vatavikara.*

Indications for *Langhana* in the form of *Shodhana:*

1. *Brihatsharira / Sthula*
2. *Balavan*
3. *Prabhuta Shleshma Pitta Asra Mala / Pitta Kaphadhika* associated with *Maruta*
4. *Amadosha -Visoochika* and *Alasaka*

5. *Jvara*
6. *Vami / Chardi*
7. *Atisara*
8. *Hridroga*
9. Patients with the symptoms like
    a. *Vibandha*
    b. *Gourava*
    c. *Udgara*
    d. *Hrullasa*
    e. *Arochaka*

Indications for *Langhana* in the form of *Pachana Deepana*:

The same conditions with moderate severity are indicated for *Langhana* in the form of *Deepana Pachana.*

Indications for *Langhana* in the form of *Kshudha* and *Trushna Nigrahana*:

The same conditions with mild severity are indicated for *Langhana* in the form of *Kshudha* and *Trushna Nigrahana*.

Indications for *Langhana* in the form of *Vata, Atapa* and *Ayasa*:

The *Vata, Pitta and Kapha Dosha* which are moderately disturbed in the individuals of *Uttamabala* or *Alpabala* one should administer *Langhana* in the form of *Vata, Atapa* and *Ayasa*.

Samyakbrumhitalakshana

1. *Bala* – increase of strength
2. *Pushti* – well-nourished body
3. *Vyadhisankshaya* - subsiding of the disease
4. *Karshyadoshavivarjana* – the *Dosha* present in *Atikarshya* would not be seen.

Samyaklanghitalakshana

1. *Vata Mutra PureeshaVisaraga*: Evacuation of *Mala – Vata, Mutra* and *Pureesha*
2. *Svedejatou* – perspiration
3. *Vimalendriyata* – clarity of senses
4. *Malotsarga* – evacuation of *Mala*
5. *Gatralaghava/ Laghuta* – feeling of lightness
6. *Ruchi* – relishing of taste
7. *Kshuttrutsahodaya* - simultaneous appearance of appetite and thirst
8. *Hridayashuddhi* – clarity in the chest region
9. *Udgarashuddhi* – clear belching
10. *Kanthashuddhi* - clarity in the throat region
11. *Asyashuddhi* - clarity in the mouth
12. *Vyadhimardavata* – subsiding of the disease
13. *Utsaha* – enthusiasm
14. *Tandranasha* – relief from lethargy
15. *Klamanasha* – relief from fatigue
16. *Nirvyathe ca Antaratmani* – No sense of discomfort

Samyakstambhitalakshana:

1. *Balalabha* – regaining the strength
2. *Amayavarjana* – relief from the disease

Atilanghitalakshana:

1. *Parva Bheda* – pain in the joints
2. *Angamarda* – Generalised body ache
3. *Kasa* – cough
4. *Mukhashosha* – dryness of the mouth
5. *Kshutpranasha* – lack of appetite
6. *Aruchi* – loss of taste

7. *Trushna* – thirst
8. *Shrotradourbalya* – deterioration in the sense of hearing
9. *Netradourbalya* – deterioration in the sense of vision
10. *Manosambhrama* – restlessness of mind
11. *Abhikshnaurdhvavata* – Upward movement of *Vata* and appearance of diseases like *Shvasa, Kasa, Karnanada, Jrumbha,* etc.
12. *Tamohridi* – Sense of dullness
13. *Dehabalanasha* – loss of physical strength
14. *Agnibalanasha* – reduction in the ability to digest

Atibrumhitalakshana

If *Brumhana* is done excessively then it leads to *Atisthoulya* and hence all the *Lakshana* mentioned under *Atisthoulya* would be exhibited. Along with that the other diseases also will be seen which are as follows –

1. *Apachi*
2. *Meha*
3. *Jvara*
4. *Bhagandara*
5. *Kasa*
6. *Sanyasa*
7. *Kruchraama*
8. *Kushtha*

Atilanghitalakshana

If *Langhana* is done excessively then it leads to *Atikarshya* hence all the *Lakshana* mentioned under *Atikarshya* would be exhibited. Along with that the other diseases also will be seen which are as follows –

1. *Bhrama*
2. *Kasa*
3. *Trushnadhikya*
4. *Arochaka*
5. *Snehakshaya*
6. *Agnikshaya*
7. *Nidrakshaya*
8. *Drukkshaya*
9. *Shrotrakshaya*
10. *Shukrakshaya*
11. *Ojakshaya*
12. *Svarakshaya*
13. *Ruja in Basti, Hridaya, Murdha, Jangha, Uru, Trika, Parshva*
14. *Jvara*
15. *Pralapa*
16. *Urdhvaanila*
17. *Glani*
18. *Chardi*
19. *Parva Asthibhedana*
20. *Varchograha*
21. *Mutragraha*

Atirukshitalakshana*:* The same symptoms mentioned for *Atilanghitalakshana* will be seen in *Atirukshita.*

Atistambhitalakshana:

1. *Shyavagatrata:* Skin becomes dark
2. *Stabdhagatrata*: Body becomes stiff

3. *Udvega:* Anxiousness
4. *Hanusangraha* : Stiffness of jaw
5. *Hridnigraha*: Stiffness in the chest region
6. *Varchonigraha*: Constipation.

***Ayoga* of all *Upakrama*:** if there is *Ayoga* of all *Upakrama* then the diseases would not subside instead there will be increase of symptoms.

Management of *Atibrumhitalakshana*

Management of the above said conditions should be done by using diet, medicine and activities which subside *Meda, Anila* and *Shleshma*.

Diets

1. *Kulattha*
2. *Joorna*
3. *Shyamaka*
4. *Yava*
5. *Mudga*
6. *Madhoodaka*
7. *Dandahata / Takra*
8. *Arishta*

Activities

1. *Chinta*
2. *Jagarana*

Therapies and medicines

1. *Shodhana*
2. *Triphala* with *Madhu*
3. *Guduchi* with *Madhu*
4. *Ghana* with *Madhu*
5. *Abhaya* with *Madhu*
6. *Rasanjanaprayoga* with *Agnimantharasa*
7. *Brihatpanchamulaprayoga* with *Agnimantharasa*

8. *Guggulu Prayoga* with *Agnimantharasa*
9. *Shilajatu Prayoga* with *Agnimantharasa*
10. Equal quantities of *Vidanga, Nagara, Kshara (Yava Kshara), Kalaloha Raja* alongwith *Madhu*
11. *Yava* and *Amalaka Churna*
12. *Vyoshadi Yoga*
13. Management of *Atilanghitalakshana*
14. Management of the above said conditions should be done by using diet, medicine and activities which have *Brumhana* effect. *Achinta, Harshana* and *Svapna* along with *Santarpana* will make the *Krusha* person stout just like a *Varaha* because of the nourishment of the *Dhatu*. *Mamsa* is said to be the best *Brumhanadravya* but nothing is better than *Mamsadamamsa* for the purpose of *Brumhana*[27].

Prameha

Nidana

The *Nidana* for *Prameha* which are formed due to *Kapha Dushti* are mentioned as follows.

1. Excessive and continuous usage of freshly harvested grains such as *Hayanaka* (a kind of red rice), *Yavaka* (wild barley), *Chinaka* (proso millet), *Uddalaka* (kodo millet), *naishadha* (variety of *shali* rice), *itkata*, *mukundaka*, *mahavrihi*, *Pramodaka*, *Sugandhaka*, *Harenuka*,
2. Excessive and continuous usage *Masha* processed with ghee.
3. Excessive and continuous usage Meat of *Gramya* and *Anupa* animals processed in ghee
4. Excessive and continuous usage *Shaka, Tila, Palala, Pishtaanna, Payasa, Krshara, Vilepi, Ikshu Vikara, Kshira, Navamadya, Mandaka Dadhi*,
5. Excessive and continuous usage foods which are *Nava, Madhura* and *Taruna*.

6. Lack of hygiene and physical exercise, excessive rest and sleep, and other practises of food and activity which essentially vitiate *Shleshma* and *Mutra*[28].

PRAMEHA SAMPRAPTI

The primary *Dosha* involved in the pathogenesis of *Prameha* is *Bahudravashleshma,* i.e, the *Sthira*, *Sheeta* and *Snigdhaguna* of *Shleshma* are compromised, leading to an increase in *Drava Guna*. A quantity-wise extensive increase in the *Drava Shleshma* occurs, which can lead to *Prameha*. A comparatively less increase in *Drava Shleshma* is not capable of generating *Prameha*. *Dushyas* in *Prameha* are *Medas, Mamsa, Shariraja Kleda, Shukram, Shonitam, Vasa, Majja, Lasika, Rasa, Ojas,* which have lost their structural and functional integrity. *Abaddham* indicates loss of functional and structural integrity. Loss of integrity and imbalance in inherent *Ushnaguna* leading to looseness and flaccidity is indicated by *Bahutvam*, and loss of compactness and *Saara* is indicated by *Ghanatvam*, and each of these two have to be understood contextually. Such as: *Medas*, *Mamsa*, *Vasa* and *Majja* get deranged in both the ways, while the others – *Shariraja Kleda*, *Shukram*, *Shonitam*, *Lasika*, *Rasa* and *Ojas* get deranged and become *Aghana*.The *Nidana*, *Dosha* and *Dushya*, during favourable conditions of pathogenesis, cause an immediate *Prakopa* in *Shleshma*, as a result of gradual accumulation. Since there is already a compromise in the *Snigdha*, *Sheeta* and *Sthiraguna* in the body, *Shaithilyata* gets imparted in the body and owing to this, the *Prakupitashleshma* circulates throughout the body very quickly. These disturbed set of *Gunas* affect the *Medas* first, due to predominant similarity in the *guna* and also because the structure of *Medas* is such that it is less compact as compared to other *Dhatus*. Then *Medas* undergoes *Dushti* due to the imbalance in the *Gunas*. This further causes *Dushti* of *Sharirakleda* and *Mamsa* due to the quantity-wise increase in the *Guna* of *Mamsa* and *Kleda*. When there is continued *Dushti* of *Mamsa,* there arise *Pidakas* as a result of excessively deranged *Mamsa*, termed as *Sharavika*, *Kacchapika,* etc. Further continuation of the *Nidana* causes further vitiation of the *Sharira Kleda*, which transforms into *Mutram*. When all these systems are deranged, the *Dosha Dushya Sammurchhana* reaches the *Mutravahasrotas* comprising of

Vankshana and *Vasti*, and leads to the obstruction from normal functioning due to the deranged and *Gurumedas* and *Kleda*. Therefore, *Pramehasamprapti* is established, which can either get further deept rooted into deranging all the systems, or can turn incurable[29].

PRAMEHA CHIKITSITAM

There are two kinds of patients suffering from *Prameha*. The *Sthula* patient who has more *Bala*, and the *Krsha* patient who has less *Bala*. Here, *Bala* signifies both *Dosha Bala* and *Deha Bala*. The *Deha Bala* is compromised in the *Krsha* patient due to the loss of *Dhatu* and in the *Sthula* patient, the *Deha Bala* is comparatively more due to the *Santarpananidana* which caused the *Prameha*. In the formed condition – *Krsha* patient with less *Bala*, the treatment to be given in *Brimhanam* and in the latter condition, the treatment to be given is S*amshodhanam*.

After undergoing *Snehana*, the patient has to be administered with *Shodhanayoga* with the formulations mentioned in *Kalpasthana*. *Shodhana* has to be done in such a way that the *Dosha* in the *Urdhwa* and *Adhobhaga* of the body are eliminated. After *Shodhana*, the line of treatment to be followed is *Santarpana*, to replenish the *Dhatus*.

If *Santarpana* is not carried out properly in a *Pramehi*, then it causes *Gulma*, *Kshaya*, *Mehanashula*, *Vastishula*, *Mutragraha*. According to the *Agnibala* of the patient, the supplements which lead to channelised nourishment of the *Dhatu* called *Paritarpana* are to be administered[30].

YAVA[31]

Morphology of Hordeum vulgare

Hordeum vulgare is an erect, annual grass growing up to 1 meter tall. Leaves are linear-lanceolate in shape, 15-25cm long. Stem cylindrical, and smooth.

Vernacular names:

Botanical name – Hordeum vulgare

Sanskrit Name – *Yava*

English Name – Barley

Hindi Name – Java, Jo, Jau

Marathi Name – Java, Cevad, Jav, Satu

Kannada Name – Javegodhi

Tamil Name – Baali arisi, Barliyarishi

Telugu Name – Yava Dhanya, Barlibiyam, Dhanyabhedam, Pachchayava, Yava

Bengali Name – Jaba, Jau, Jav

Gujarati Name – Cheno, Jau

Malayalam Name – Javegambu

Punjabi Name – Javo, Jawa, Nai

Table No. 7 Classical categorization of *Triphala*:

Bhavaprakasha Nighantu	*Dhanya Varga*
Raja Nighantu	*Shalyadi Varga*
Dhanvantari Nighantu	*Suvarnaadi Varga*
Shodala Nighantu	*Shukadhanya Varga*
Kaiyadeva Nighantu	*Dhanya Varga*

Table No. 8 Medicinal properties of *Triphala*[32]:

Rasa	Astringent (*Kashaya*), Sweet (*Madhura*)
Guna	*Ruksha, Aguru, Mridu, Sara, Pichhila*
Veerya	*Sheeta* (Cold)
Vipaka	*Katu* (Pungent)
Karma	*Pramehahara, Kaphahara, Balaprada, Vrishya*, gives *Bahu Veera* and *Pusti* to the person.

Synonyms:

Sitashuka, Divyaakshata, Kanchukidhanaya Raajau, Teekshana Shuka, Tura Priya, Shaktu, Hayaista, Pavitra Dhanyam, Shuchi, Nishooka, Atiyava, Akshata, Yava

Karma:

Kaphapittahara increases *Vata*, stabilizes, *Balya*, *Srotoshuddikara*, useful in *Kaphaja* disorder, *Pathya* in *Vrana*, if it is taken daily it will create *Baddamutra*, *Bahuvata* and *Varcha*, *Vrishya*, useful in *Mootra*, *Medajaroga*. Useful in *Peenasa*, *Shwasa*, *Kasa*, *Urusthamba* and diseases of throat and skin, *Trishnaurusthambha*, disorders of *Rakta*. It is not *Abhishyandi*, it acts as *Lekhana*, it increases *Bala* and *Swara*, increases complexion, *Lekhana*, *Medha* and *Agnivardhana*. Helps in easy evacuation of bowel. It is *Swarya*, given in *Meha*, alleviates *Trushna*, *Vatarakthahara*, *Mootra*, *Kapha* and *Medahara* property. If it is taken daily it will create *Badda Mutra, Bahu Vata* and Varcha, Agni, *Medha*, *Bala*, and *Varna*, *Sthulahara* property. Given in *prameha*, does *Shamana* of *Thrut*, does the *Prasadana* of *Shonita* and *Pitta*.

Table No. 9 Nutritional Value of Barley[33]

Energy	1,474 kJ (352 kcal)
Carbohydrates	77.7 g
Sugars	0.8 g
Dietary fibre	15.6 g
Fat	1.2 g
Protein	9.9 g
Thiamine (vit. B1)	0.2 mg (17%)
Riboflavin (vit. B2)	0.1 mg (8%)
Niacin (vit. B3)	4.6 mg (31%)
Vitamin B6	0.3 mg (23%)
Calcium	29.0 mg (3%)
Iron	2.5 mg (19%)
Phosphorus	221 mg (32%)
Zinc	2.1 mg

REVIEW ON METABOLISM

The body's metabolism encompasses all the chemical processes involved in energy production, energy release and growth. These processes can be anabolic (formation of substances) or catabolic (breakdown). Ultimately, all energy contained in ingested nutrients manifests as heat, workdone on the environment or growth. A healthy young man requires ~ 30kcal/kg body weight to sustain resting metabolism for 1 day. Thus, a 70–kg human requires 2100 kcal/day, an amount known as the Resting metabolic rate (RMR). The number of calories rises with increased activity, illness, or other stress. For example, the metabolic rate can increase 2 to 3 fold with exposure to a cold environment or during performance of heavy exercise. The Basal Metabolic rate (BMR) is a clinical definition for metabolism that is measured under standardized conditions in which the subject

1. Has had a full night of restful sleep
2. Has been fasting for 12 hours
3. Is in neutral thermal environment (see chapter 59)
4. Has been resting physically for 1 hour and
5. Is free of psychic and physical stimuli

The BMR (units: kcal per hour and per square meter of body surface area) in adults is ~5% higher for male than for female subjects and falls with age. The BMR is less than RMR.

Regulation of energy metabolism in human involves a complex interplay among ingested nutrients, hormones and inter–organ exchange of substrates to maintain a constant and adequate supply of fuel for all organs of the body. Because energy acquisition by the body is intermittent, whereas energy expenditure is continuous, the body needs to store and then parcel out energy in a carefully coordinated fashion. Insulin is the key hormone that orchestrates this exchange and distribution of substrates between tissues under fed and fasting conditions (see chapter 51). Glucagon and catechol amines, cortisol and growth hormone play major roles in energy regulation at times of acute energy needs, which occur during exercise, in conditions of stress, or in response to hypoglycemia. The major organs involved in fuel homeostasis are as follows –

1. The liver which is normally the only major producer of Glucose
2. The brain which in the fed state or early in the fasted state is a near obligate glucose consumer and,
3. The muscle and adipose tissue, which respond to insulin and store energy in the form of glycogen and fat respectively.

Forms of energy

Virtually all energy that sustains human life is derived directly or indirectly from breaking carbon–carbon bonds, which are created in plants during photosynthesis. Cellulose, the principal form of this stored energy in the biosphere, consists of polymers of glucose joined by beta – 1, 4 linkages that we cannot digest (see chapter 45). However, ruminants (are the mammals that are able to acquire nutrients from plant based food by fermenting it in a specialized stomach prior to digestion, principally through microbial actions) can degrade cellulose to glucose because they have cellulose producing bacteria in their digestive tracts. Humans obtain their energy from food in three forms –

1. Carbohydrates
2. Proteins
3. Lipids

Moreover, each form of consists of building blocks: monosaccharides (glucose, fructose and galactose) for carbohydrates, amino acids for proteins and fatty acids for Lipids.

Carbohydrates, which exist in the whole body mainly in the form of glucose, contain 4.1kcal/g of energy. The major storage form is glycogen, a polymer of glucose (10^6 to 10^8 Da) that consists of glucose molecules linked together by alpha – 1, 4 linkages in the straight portions of the polymer and alpha – 1, 6 linkages at the frequent branch points.

Virtually all cells of the body store glycogen; the highest concentrations occur in liver and muscle. Cells store glycogen in cytoplasmic granules that also contain the enzymes needed for glycogen synthesis and degradation, glycogen is highly hydrophilic; 1 to 2 gram of water is stored with each gram of glycogen, thus providing a handy storage depot for glucose without affecting the osmotic pressure of the intracellular space. However, this packaging of glycogen with water makes glycogen a relatively inefficient means of storing energy because it yields only 1 – 2 k/cal for each gram of hydrated glycogen instead of the theoretical 4.1 kcal/g pf dry carbohydrate. In contrast to the other potential stored forms of energy (Lipid and protein, the Liver can quickly breakdown glycogen by glycogenolysis to provide glucose for brain during hypoglycemia. Muscle can quickly break down glycogen into glucose–6–phosphate (G6P) to provide energy necessary to run an intensity anaerobic sprint.

The liver normally contains 75 to 100 g of glycogen but can store upto 120g (8% of its weight) as glycogen. Muscle stores glycogen at much lower concentrations (1% to 2% of its weight). However because of its larger mass, skeletal muscle has the largest store of glycogen in the body (300 to 400g). A typical 70–kg human has upto ~700g of glycogen (~1% of body weight). Thus the total energy stored in the body in the form of glycogen can be nearly 3000 kcal, which is still a tiny fraction of that stored in the form of lipid, enough to supply resting metabolism for less than a day and a half, assuming 100% efficiency. Nonetheless, Carbohydrate stores are essential because certain tissues, particularly brain, rely heavily on carbohydrates for their fuel.

Whereas muscle contains the largest store of glycogen in the body, this pool of glycogen cannot directly contribute directly to blood glucose in response to hypoglycemia because muscle lack G6pase, which is necessary to convert G6P derived from glycogenolysis to glucose. Instead the primary role of muscle glycogen is to supply energy locally for muscle contraction during intense exercise.

Proteins are linear polymers of L–amino acids (L means left) which have the general molecular structure $+H_3N - HC(R) - COO^-$. Different functional R groups (functional groups are specific substituents or moieties within molecules that are responsible for the characteristic chemical reactions of those molecules. The same functional group will undergo the same or similar chemical reaction regardless of the size of the molecule it is a part of. This allows a systematic prediction of chemical reactions and behavior of chemical compounds and design of chemical syntheses.

Furthermore the reactivity of a functional group can be modified by other functional groups nearby. Functional groups are groups of one or more atoms of distinctive chemical properties no matter what they are attached to. The atoms of functional groups are linked to each other and to the rest of the molecule by covalent bonds. R group usually means an alkyl group. Amino acids are organic compounds containing amine ($-NH_2$) and carboxyl (-COOH) functional groups along with a side chain (R group) specific to each amino acid) distinguish the 20 amino acids incorporated into nascent proteins (a protein as it is being formed by a

ribosome before it folds into its active shape) during mRNA translation. (in molecular biology and genetics, translation is the process in which ribosomes in the cytoplasm or ER synthesize proteins after the process of transcription of DNA to RNA in the cell's nucleus. The entire process is called gene expression. In translation, messenger RNA (mRNA) is decoded in a ribosome to produce a specific amino acid chain, or poly peptide) In addition, four other amino acids are present in mature proteins: gamma – carboxyglutamic acid, hydroxylysine, 4 hydroxyproline, and 3 hydroxyproline. However, these amino acids result from post translational modification of amino acids that are already in the polypeptide chain. In alpha amino acids, the amino group (- NH_3^+), the carboxyl group (- COO^-), and R all attach to the central or alpha – carbon atom. In proteins, the amino acids are linked together by peptide bonds that join the alpha amino group of one amino acid with the alpha carboxyl group of another. Nine of the amino acids are termed as essential amino acids because the body cannot synthesize them the rates sufficient to sustain growth and normal functions. Thus we must obtain these amino acids in the diet. Proteins contain 4.3 kcal/g, which approximately the same as carbohydrates. A typical 70kg human with 14% protein (9.8 kg) only about half of which is available as a fuel source can thus store~ 21000 kcal in the form of available protein which could potentially provide ~10 days worth of energy. Unlike carbohydrate, protein is not a primary energy reserve in human. Instead proteins serve other important structural and functional roles.

Structural proteins make up skin, collagen, ligaments, and tendons. Functional proteins include enzymes that catalyze reactions, muscle filaments such as myosin and actin and various hormones. The body constantly breaks down proteins to amino acids, and vice versa therevy allowing cells to change their protein make up as demands change. Thus it is not surprising that protein catabolism makes only a small contribution much less than 5% to normal resting energy requirements. In contrast during starvation when carbohydrate reserves are exhausted, protein catabolism can contribute as much as 15% of the energy necessary to sustain the resting metabolic requirements by acting as a major substrates for gluconeogenisis.

In the healthy human adult who is eating a weight maintaining diet, amino acids derived from ingested protein replenish those proteins that have been oxidized in normal daily protein turnover. Once these protein requirements have been met, the body first oxidizes excess protein to CO_2 and then converts the reminder to glycogen or triacylglycerols (TAGs).

Lipids are the most concentrated form of energy storage because they represent, on average, 9.4 kcal/g. Lipids are dietary substances that are soluble in organic solvents but not in water and typically occur in the form of TAGs. The gastrointestinal tract breaks down ingested TAGs into Fatty acids and 2 – monoacylglycerols. FAs are composed of long carbon chains (14 to 24) with carboxyl terminus, and they can be either saturated with hydrogen atoms or unsaturated (i.e. double bonds may connect one or more pairs of carbon atoms).

When fully saturated, FAs have the general form of $CH_3 – (CH_2)$ n – COOH. In contrast to glycogen and protein, fat is stored in a non aqueous environment and therefore yields energy very close to its theoretical 9.4 kcal/g of TAGs. This greater efficiency of energy storage provided by fat is crucial for human existence in that it allows for greater mobility and promotes survival during famine. Therefore, although humans have two large storage depots of potential energy (protein and fat), fat serves as the major expendable fuel source. Most of the body's fat depots exist in the subcutaneous adipose tissue layers, although fat also exists to a small extent in muscle and in visceral (deeper) depots in obese individuals. A typical 70–kg human with 20% fat (14 kg) thus carries 131,600 kcal of energy stored in adipose tissue. Assuming an RMR of 2100 kcal/day and 100% efficiency of converting the fat to energy, mobilization and subsequent oxidation of this entire depot could theoretically sustain the body's entire resting metabolic requirement for nearly 9 weeks.

ENERGY BALANCE

Energy Input to the Body is the Sum of Energy Output and Storage.

The first law of thermodynamics states that energy can neither be created nor destroyed; in a closed system, total energy is constant. This concept is illustrated as follows –

Energy input is from

1. Carbohydrate
2. Fat
3. Protein

Energy output is in the form of

1. Mechanical work
    a. Muscle contraction
    b. Movement of cells, organelles and appendages
2. Synthetic reactions
    a. Creation of essential functional molecules
3. Membrane transport
    a. Minerals and organic anions/ cations Amino acids
4. Signal generation and conduction
    a. Electrical
    b. Chemical
    c. Mechanical
5. Heat production
    a. Temperature regulation

b. Inefficient chemical reactions

6. Detoxification and degradation

   a. Urea formation

   b. Conjugation

   c. Oxidation

   d. Reduction

Fuel storage and Growth

So Energy input is equal to Energy output + Fuel storage and Growth

Humans acquire all their energy from ingested food, store it in different forms and expend it in different ways. In the steady state, energy intake must equal energy output.

The GI tract breaks down ingested carbohydrates, proteins, and fats into smaller components and then absorbs them into the blood stream for transport to sites of metabolism (see chapter 45). For example the GI tract reduces ingested carbohydrates to simple sugars (e.g. glucose), which are then transported to muscle cells and are either oxidized to release energy or converted to glycogen for storage. Oxidation of fuels generates not only free energy but also waste products and heat (thermal energy)

REVIEW ON DIABETES MELLITUS [34]

Definition: As per the WHO, diabetes mellitus (DM) is defined as a heterogeneous metabolic disorder characterised by common feature of chronic hyperglycaemia with disturbance of carbohydrate, fat and protein metabolism.

Classification: The older classification systems dividing DM into primary (idiopathic) and secondary types, juvenile onset and maturity onset, and insulin dependent and non-insulin dependent.

I. TYPE 1 DIABETES MELLITUS (10%) (earlier called Insulin-dependent, or juvenile-onset diabetes)

- DM: Immune-mediated
- DM: Idiopathic

II. TYPE 2 DIABETES MELLITUS (80%) (Earlier called non-insulin-dependent, or maturity-onset diabetes)

III. OTHER SPECIFIC TYPES OF DIABETES (10%)

- Genetic defect of β-cell function due to mutations in various enzymes (earlier called maturity-onset diabetes of the younger MODY)
- Genetic defect in insulin action (e.g. type A insulin resistance)
- Diseases of exocrine pancreas (e.g. chronic pancreatitis, pancreatic tumours, post-pancreatectomy)
- Endocrinopathies (e.g. acromegaly, Cushing's syndrome, pheochromocytoma)
- Drug- or chemical-induced (e.g. steroids, thyroid hormone,thiazides, β-blockers etc)
- Infections (e.g. congenital rubella, cytomegalovirus)
- Uncommon forms of immune-mediated DM (stiff mansyndrome, anti-insulin receptor antibodies)

- Other genetic syndromes (e.g. Down's syndrome, Klinefelter's syndrome, Turner's syndrome)

TYPE 1 DM. It constitutes about 10% cases of DM. It was previously termed as juvenile-onset diabetes (JOD) due toits occurrence in younger age, and was called insulin-dependent DM (IDDM) because it was known that these patients have absolute requirement for insulin replacement as treatment. However, in the new classification, neither age nor insulin-dependence are considered as absolute criteria. Instead, based on underlying etiologic, type 1 DM is further divided into 2 subtypes they are

- DM (immune-mediated) characterised by autoimmune destruction of β-cells which usually leads to insulin deficiency.
- DM (idiopathic) characterised by insulin deficiency with tendency to develop ketosis but these patients are negative for autoimmune markers.

Clinical features:

- Patients of type 1 DM usually manifest at early age, generally below the age of 35.
- The onset of symptoms is often abrupt. At presentation, these patients have polyuria, polydipsia polyphagia.
- The patients are not obese but have generally progressive loss of weight.
- These patients are prone to develop metabolic complications such as ketoacidosis and hypoglycaemic episodes.

TYPE 2 DM. This type comprises about 80% cases of DM. It was previously called maturity-onset diabetes, or non-insulin dependent diabetes mellitus (NIDDM) of obese and non-obese type. Although type 2 DM predominantly affects older individuals, it is now known that it also occurs in obese adolescent children; hence the term MOD for it is inappropriate. Moreover, many type 2 DM patients also require insulin therapy to control hyperglycaemia or to prevent ketosis and thus are not truly non-insulin dependent contrary to its former nomenclature.

Clinical features:

- This form of diabetes generally manifests in middle life or beyond, usually above the age of 40.
- The onset of symptoms in type 2 DM is slow andinsidious.
- Generally, the patient is asymptomatic when the diagnosis is made on the basis of glucosuria or hyper-glycaemia during physical examination, or may present with polyuria and polydipsia.
- The patients are frequently obese and have unexplained weakness and loss of weight.
- Metabolic complications such as ketoacidosis

Other specific etiologic types of DM: Besides the two main types, about 10% cases of DM have a known specific etiologic defect. One important subtype in this group is maturity-onset diabetes of the young (MODY) which has autosomal

dominant inheritance, early onset of hyperglycaemia and impaired insulin secretion.

GESTATIONAL DM: About 4% pregnant women develop DM due to metabolic changes during pregnancy. Although they revert back to normal glycaemia after delivery, these women are prone to develop DM later in their life.

PATHOGENESIS

Depending upon etiology of DM, hyperglycaemia may result from the following:

- Reduced insulin secretion
- Decreased glucose use by the body
- Increased glucose production.

PATHOGENESIS OF TYPE 1 DM. The basic phenomenon in type 1 DM is destruction of β-cell mass, usually leading to absolute insulin deficiency. While type 1B DM remains idiopathic, pathogenesis of immune mediated DM is immune-mediated and has been extensively studied. Currently, pathogenesis of immune mediated DM is explained on the basis of 3 mutually-interlinked mechanisms: genetic susceptibility, autoimmune factors, and certain environmental factors.

Genetic susceptibility: Immune mediated DM involves inheritance of multiple genes to confer susceptibility to the disorder:

- It has been observed in identical twins that if one twin has immune mediated DM, there is about 50% chance of the second twin developing it, but not all. This means that some additional modifying factors are involved in development of DM in these cases.
- About half the cases with genetic predisposition to immune mediated DM have the susceptibility gene located in the HLA region of chromosome 6 (MHC class II region), particularly HLADR3, HLA DR4 and HLA DQ locus.

Autoimmune factors: Studies on humans and animal models on immune mediated DM have shown several immunologic abnormalities:

- Presence of islet cell antibodies against GAD (glutamic acid decarboxylase), insulin etc, though their assay largely remains a research tool due to tedious method.
- Occurrence of lymphocytic infiltrate in and around the pancreatic islets termed insulitis. It chiefly consists of CD8+ T lymphocytes with variable number of CD4+ T lymphocytes and macrophages.
- Selective destruction of β-cells while other islet cell types (glucagon-producing alpha cells, somatostatin-producing delta cells, or polypeptide-forming PP cells) remain unaffected. This is mediated by T-cell mediated cytotoxicity or by apoptosis.

- Role of T cell-mediated autoimmunity is further supported by transfer of immune mediated DM from diseased animal by infusing T lymphocytes to a healthy animal.
- Association of immune mediated DM with other autoimmune diseases in about 10-20% cases such as Graves' disease, Addison's disease, Hashimoto's thyroiditis, pernicious anaemia.
- Remission of immune mediated DM in response to immunosuppressive therapy such as administration of cyclosporin A.

Environmental factors: Epidemiologic studies in immune mediated DM suggest the involvement of certain environmental factors in its pathogenesis, though role of none of them has been conclusively proved. In fact, the trigger may precede the occurrence of the disease by several years. It appears that certain viral and dietary proteins share antigenic properties with human cell surface proteins and trigger the immune attack on β-cells by a process of molecular mimicry. These factors include the following:

- Certain viral infections preceding the onset of disease e.g. mumps, measles, coxsackie B virus, cytomegalovirus and infectious mononucleosis.

- Experimental induction of immune mediated DM with certain chemicals has been possible e.g. alloxan, streptozotocin and pentamidine.
- Geographic and seasonal variations in its incidence suggest some common environmental factors.
- Possible relationship of early exposure to bovine milk proteins and occurrence of autoimmune process in immune mediated DM is being studied.

PATHOGENESIS OF TYPE 2 DM. The basic metabolic defect in type 2 DM is either a delayed insulin secretion relative to glucose load (impaired insulin secretion), or the peripheral tissues are unable to respond to insulin (insulin resistance).

Type 2 DM is a heterogeneous disorder with a more complex etiology and is far more common than type 1, but much less is known about its pathogenesis. A number of factors have been implicated though, but HLA association and autoimmune phenomena are not implicated. These factors are as under genetic factors, constitutional, insulin resistance, impaired insulin secretion, increased hepatic glucose synthesis.

Genetic factors: Genetic component has a stronger basis for type 2 DM than immune mediated DM. Although no definite and consistent genes have been

identified, multifactorial inheritance is the most important factor in development of type 2 DM:

- There is approximately 80% chance of developing diabetes in the other identical twin if one twin has the disease.
- A person with one parent having type 2 DM is at an increased risk of getting diabetes, but if both parents have type 2 DM the risk in the offspring rises to 40%.

Constitutional factor: Certain environmental factors such as obesity, hypertension, and level of physical activity play contributory role and modulate the phenotyping of the disease.

Insulin resistance: One of the most prominent metabolic features of type 2 DM is the lack of responsiveness of peripheral tissues to insulin, especially of the skeletal muscle and liver. Obesity, in particular, is strongly associated with insulin resistance and hence type 2 DM. Mechanism of hyperglycaemia in these cases is explained as under:

- Resistance to action of insulin impairs glucose utilisation and hence hyperglycaemia.
- There is increased hepatic synthesis of glucose.

- Hyperglycaemia in obesity is related to high levels of free fatty acids and cytokines (e.g. TNF-α and adiponectin) affect peripheral tissue sensitivity to respond to insulin.

The precise underlying molecular defect responsible for insulin resistance in type 2 DM has yet not been full identified. Currently, it is proposed that insulin resistance may be possibly due to one of the following defects:

- Polymorphism in various post-receptor intracellular signal pathway molecules.
- Elevated free fatty acids seen in obesity may contribute e.g. by impaired glucose utilisation in the skeletal muscle, by increased hepatic synthesis of glucose, and by impaired β-cell function.

Insulin resistance syndrome is a complex of clinical features occurring from insulin resistance and its resultant metabolic derangements that includes hyperglycaemia and compensatory hyperinsulinemia.

Clinical features

- Accelerated cardiovascular disease
- Mild hypertension
- Dyslipidaemia

Impaired insulin secretion: In type 2 DM, insulin resistance and insulin secretion are interlinked:

- Early in the course of disease, in response to insulin resistance there is compensatory increased secretion of insulin (hyperinsulinemia) in an attempt to maintain normal blood glucose level.
- Eventually, however, there is failure of beta-cell function to secrete adequate insulin, although there is some secretion of insulin i.e. cases of type 2 DM have mild to moderate deficiency of insulin (which is much less severe than that in type 1 DM) but not its total absence.

Increased hepatic glucose synthesis: One of the normal roles played by insulin is to promote hepatic storage of glucose as glycogen and suppress gluconeogenesis. In type 2 DM, as a part of insulin resistance by peripheral tissues, the liver also shows insulin resistance i.e. in spite of hyper insulinaemia in the early stage of disease, gluconeogenesis in the liver is not suppressed. This results in increased hepatic synthesis of glucose which contributes to hyperglycaemia in these cases.

Complications of DM: Both types of DM may develop complications which are broadly divided into 2 major groups

Acute metabolic complication:

- Diabetic ketoacidosis
- Hyperosmolar non-ketotic coma
- Hypoglycaemia

Late systemic complication:

- Atherosclerosis
- Diabetic microangiopathy
- Diabetic nephropathy
- Diabetic neuropathy
- Diabetic retinopathy

Chapter III

METHODOLOGY

METHODOLOGY

Objectives of the study:

1. To critically analyse the concept of *Apatarpana* from *brihattrayee* and *laghutrayee*.
2. To evaluate the concept of *apatarpana* in *Prameha* with *Triphala Sadhita Yava* in Streptozotocin (STZ) induced diabetes in Wistar albino rats.

The study was planned in two phases – conceptual study and applied study.

MATERIALS AND METHODS:

Conceptual study

Source of Data: The literature survey on the conceptual study was compiled from *Brihatrayee* and all published literature source.

Plan of the study: The conceptual study was carried out under these headings

1. Review of the fundamental concepts which will act as the frame work for the concept under study that is *Apatarpana*.
2. Review of the concept of *Apatarpana* as a *Upakrama* along with *Santarpana* as a *Hetu* and also as a *Upakrama*.
3. Review on the *Prameha Santarpana Janya Vyadhi* and *Triphala Sadhitayava* as *Apatarpana Upakrama*.
4. Drug Review on *Yava* and *Triphala*

- The interpretation of concept was done by emphasising on the contextual meaning and its significance is studied in *Chikitsa*.

Applied study

PHARMACEUTICAL STUDY: Pharmaceutical study deals with the process of preparation of medicine starting from collection of drugs till obtaining the final product. It is divided into following sections:

1. Collection of *Yava*: It was collected from a local vendor
2. Collection of *Triphala Churna*: It was collected from Sri Dharmasthala Manjunatheshwara Ayurveda Hospital Dispensary.
3. Authentication of Drugs: The authentication of the all four drugs was done at Pharmaceutical and Pharmacognosy lab at SDM Centre for Research in Ayurveda and Allied Sciences, Udupi.
4. Preparation of *Triphala Sadhita Yava*: It was done in Teaching Pharmacy of Sri Dharmasthala Manjunatheshwara College of Ayurveda and Hospital, Hassan.

Preparation of *Triphala Sadhita Yava*:

- Preparation of *Kashaya*: The four kilogram of *Kwatha Churna* of *Triphala* [*Amalaki* (*Emblica officinalis* Gaertn), *Haritaki* (*Terminalia chebula* Retz) and *Vibhitaki* (*Terminalia bellerica* Roxb)] was taken in a clean sterile stainless steel vessel and added with thirty two liters of water and kept for boiling on medium flame till the *Kashaya* was reduced to eight liters. The *Kashaya* was filtered through a clean cloth and was allowed to cool[35].

- Preparation of *Triphala Sadhita Yava*: The cleaned *Yava* were soaked in the *Triphala Kashaya* for 12 hours and then allowed to dry under shade for two days. The dried *Yava* was made into *Yavakuta Churna*[4].

EXPERIMENTAL STUDY:

Place of study: The applied study was planned and carried out at SDM Centre for Research in Ayurveda and Allied Sciences, Udupi.

Test Drug: *Triphala Sadhita Yava*

Standard Drug: The standard reference drug Glibenclamide 10mg/ kg

Experimental Animal: Wistar albino rats were selected from animal house of SDM Centre for Research in Ayurveda and Allied Sciences, Udupi. Rats were fed with normal rat diet and water *ad libitum* throughout the study. They were acclimatized in the laboratory condition for two weeks prior to experimentation. The experimental rats were maintained at 12:12h light and dark cycle, at temperature of 25^0C and relative humidity of approximately 50%. Prior to the experimentation permission was obtained from Institutional Animal Ethical Committee.

Animal grouping for the study

Group I – Diabetic group	Diabetic control- treated with 0.5% CMC
Group II Diabetic group	Reference standard–treated with Glibenclamide 10mg/ kg
Group III Diabetic group	Extract of ***Triphala Sadhita Yava*** 200 mg/kg once daily for 28 days
Group IV Diabetic group	Extract of ***Triphala Sadhita Yava*** 400 mg/ kg once daily for 28 days
Group V Non Diabetic group	Extract of ***Triphala Sadhita Yava*** 400 mg/ kg once Daily for 28 days

Instruments: Gluconometer and strips (Routine lab items)

Induction of diabetes:

STZ solution of 10 mg/ ml was prepared in ice-cold citrate buffer 0.1 M, pH 4.5 was kept in ice and was administered within 5 minutes at a dose of 35-40mg/ kg-body weight intra-peritoneally. After 48-72 hours of STZ administration, rats with moderate diabetes having glycosuria and hyperglycaemia (i.e., with a blood glucose of 200 - 300 mg/dl) were taken for the experiment[36].

TOTAL NUMBER OF ANIMALS REQUIRED- 24 (6 in each group)

PARAMETERS TO BE ESTIMATED:

Rats were fasted overnight and blood was withdrawn from the retro orbital plexus or blood from tail tip by making small incision on the 0, 3, 5, 10, 14, 21 & 28^{th} day of induction of diabetes for all groups and fasting glucose level was determined.

On 28^{th} day the following parameters will be determined:

1. Serum lipid profile on 28^{th} day
2. Histopathological changes in the pancreases and liver tissues.
3. Antioxidant status (liver and pancreases)

Statistical analysis

The obtained data was expressed as Mean ± SEM and analysed by one-way ANOVA, followed by Dunnett's multiple comparison 't' test using Graph Pad Prism 3. A $p<0.05$ will be considered as statistically significant.

Chapter IV

Observations

Observations

Table No. 10 Fasting Blood glucose levels in mg/dl

Rt no:	Normal control	Diabetic control	Standard	Test 1	Test 2	Non diabetic
1	149	595	150	539	183	99
2	148	787	131	549	174	130
3	159	641	214	569	495	106
4	149	558	152	650	213	112
5	148	620	214	218	576	124
6	159	652	172	634	586	112

Table No. 11 HbA1c in percentage

Rt no:	Normal control	Diabetic control	Standard	Test 1	Test 2	Non diabetic
1	3.4	7.7	3.2	8.1	8.9	3.0
2	3	9.8	3.7	7.7	6.7	3.0
3	3.9	8.6	4.3	7.8	9.6	3.0
4	3.4	8.4	4.3	8.9	6.7	3.7
5	3	7.2	4.0	4.8	9.3	3.0
6	3.9	9.8	3.9	9.2	7.6	4.1

Table No. 12 Mean Fasting Blood Glucose in mg/dl

Rt no:	Normal control	Diabetic control	Standard	Test 1	Test 2	Non diabetic
1	27	237	21	174	210	14
2	14	274	37	170	137	14
3	44	167	57	174	234	14
4	27	227	47	210	137	37
5	14	225	57	74	224	14
6	44	240	43	220	167	51

Table No. 13 Level of SGOT in IU/I

Rt no:	Normal control	Diabetic control	Standard	Test 1	Test 2	Non diabetic
1	186	122	126	101	145	94
2	187	137	139	140	126	99
3	165	194	192	112	200	106
4	186	108	136	124	125	112
5	187	160	192	101	197	89
6	165	149	157	163	85	104

Table No. 14 Level of SGPT in IU/I

Rt no:	Normal control	Diabetic control	Standard	Test 1	Test 2	Non diabetic
1	86	102	66	112	44	38
2	81	97	68	163	82	43
3	74	115	60	151	124	45
4	86	102	68	142	80	57
5	81	99	71	60	127	42
6	74	92	67	146	69	40

Table No. 15 Level of ALP KAU/dL

Rt no:	Normal control	Diabetic control	Standard	Test 1	Test 2	Non diabetic
1	472	1533	200	1230	616	484
2	499	807	330	1632	1392	380
3	380	969	332	1107	954	384
4	472	1580	428	1620	958	325
5	499	964	428	1313	1253	240
6	380	862	343	1638	914	219

Table No. 16 Level of TP in mg/ml

Rt no:	Normal control	Diabetic control	Standard	Test 1	Test 2	Non diabetic
1	6.2	6.8	7.4	6.8	5.1	6.3
2	5.2	5..3	7.2	5.4	6.0	5.9
3	6.2	5.9	5	6.8	6.2	6.0
4	6.2	6.4	7	6.6	5.2	6.3
5	5.2	6	7.2	7.4	6.1	7.3
6	6.2	5.4	6.7	6.9	5.4	6.6

Table No. 17 Level of ALB in g/dL

Rt no:	Normal control	Diabetic control	Standard	Test 1	Test 2	Non diabetic
1	3.4	3.4	3.9	2.9	2.5	3.0
2	4	2.6	4	3.1	2.9	2.9
3	3.8	3	3.4	3.5	2.5	2.9
4	3.4	3.4	3.7	3.6	3.1	3.1
5	4	3.5	4	3.0	2.6	3.2
6	3.8	2.6	3.8	2.9	2.6	3.0

Table No. 18 Level of GLO in g/dL

Rt no:	Normal control	Diabetic control	Standard	Test 1	Test 2	Non diabetic
1	2.8	3.4	3.5	3.2	3.0	2.0
2	1.2	2.7	3.2	3.5	3.2	1.5
3	2.4	2.9	1.6	1.5	1.0	2.2
4	2.8	3	3.3	3.2	3.0	2.5
5	1.2	2.5	3.2	3.5	3.2	1.2
6	2.4	2.8	3.2	3.5	3.2	2.5

Table No. 19 Level of T. Bil in mg/dL

Rt no:	Normal control	Diabetic control	Standard	Test 1	Test 2	Non diabetic
1	0.14	0.2	0.12	0.5	0.8	0.7
2	0.18	0.15	0.11	0.7	0.5	0.8
3	0.17	0.39	0.13	0.3	0.7	0.7
4	1.16	0.15	0.11	0.6	0.6	0.7
5	0.15	0.17	0.13	0.5	0.7	0.7
6	0.14	0.20	0.12	0.6	0.7	0.8

Table No. 20 Level of D.Bil in mg/dL

Rt no:	Normal control	Diabetic control	Standard	Test 1	Test 2	Non diabetic
1	0.14	0.1	0.1	0.08	0.1	0.1
2	0.18	0.06	0.02	0.01	0.02	0.1
3	0.17	0.10	0.1	0.4	0.1	0.2
4	0.16	0.07	0.01	0.03	0.05	0.1
5	0.17	0.05	0.1	0.3	0.09	0.2
6	0.16	0.80	0.06	0.1	0.04	0.4

Table No. 21 Level of Urea in mg/dL

Rt no:	Normal control	Diabetic control	Standard	Test 1	Test 2	Non diabetic
1	37	73	61	40	104	41
2	33	76	53	42	65	37
3	29	82	57	40	91	35
4	34	91	61	58	56	41
5	29	89	61	29	70	46
6	32	72	58.6	54	74	47

Table No. 22 Level of Creatinine in mg/dL

Rt no:	Normal control	Diabetic control	Standard	Test 1	Test 2	Non diabetic
1	0.4	0.7	1.0	0.6	0.4	0.6
2	0.3	0.7	0.9	0.6	0.4	0.5
3	0.2	0.7	1	0.5	0.5	0.5
4	0.3	0.6	0.8	0.6	0.6	0.5
5	0.5	0.6	1.0	0.5	0.6	0.5
6	0.3	0.6	0.9	0.5	0.5	0.5

Table No. 23 Level of Cholesterol in mg/dL

Rt no:	Normal control	Diabetic control	Standard	Test 1	Test 2	Non diabetic
1	33	52	46	109	40	77
2	34	25	65	91	90	76
3	32	31	57	99	62	81
4	33	39	48	91	118	70
5	32	28	57	54	88	87
6	33	22	55	105	99	108

Table No. 24 Level of TG in mg/dL

Rt no:	Normal control	Diabetic control	Standard	Test 1	Test 2	Non diabetic
1	46	129	70	130	25	38
2	76	68	135	171	47	48
3	79	58	108	212	45	43
4	67	37	280	199	51	55
5	67	64	108	62	56	126
6	66	88	140	139	54	107

Table No. 25 Protein estimation mg/ml

Rt no:	Normal control	Diabetic control	Standard	Test 1	Test 2	Non diabetic
1.	0.82	0.030	0.72	0.078	0.065	0.060
2.	0.84	0.020	0.58	0.029	0.064	0.052
3.	0.79	0.010	0.58	0.046	0.069	0.069
4.	0.79	0.021	0.68	0.027	0.022	0.033

Table No. 26 Catalase activity mmoles/min/mg protein

Rt no:	Normal control	Diabetic control	Standard	Test 1	Test 2	Non diabetic
1.	0.992	3.75	0.989	1.528	1.841	1.24
2.	0.765	3.67	2.079	4.339	1.940	1.34
3.	0.992	5.98	2.170	2.662	1.843	1.67
4.	0.307	4.89	1.851	4.104	5.698	2.56

Table No. 27 Glutathione peroxidase mmoles of glutathione/mg protein/min

Rt no:	Normal control	Diabetic control	Standard	Test 1	Test 2	Non diabetic
1.	22.82	45.67	10.519	9.677	31.113	14.28
2.	13.025	54.34	13.804	50.525	15.376	13.21
3.	10.278	51.98	9.409	18.225	12.897	25.43
4.	12.406	49.81	14.740	30.607	37.636	21.22

Table No. 28 Lipid peroxidation mmoles of MDA formed/g wet tissue

Rt no:	Normal control	Diabetic control	Standard	Test 1	Test 2	Non diabetic
1.	1.542	2.41	1.422	0.894	1.067	1.04
2.	1.790	2.01	1.079	0.918	0.771	0.98
3.	1.324	1.98	0.795	0.927	1.115	1.11
4.	1.578	1.87	0.867	1.199	0.870	0.91

Observation and result:

Table No. 29: Fasting Blood glucose levels in mg/dL

Groups	**Sugar(unit)**	**% change**
Normal control	152.00±2.22	--
Diabetic control	642.16±32.08**	322.47↑@
Standard	172.2±17.45**	73.18↓#
Test 1	526.16±64.28	18.06↓#
Test 2	369.33±81.26**	42.48↓#
Non diabetic	115.37±3.68	24.09↓@

Data: MEAN ± SEM, **P<0.01. @-Compared with normal control. #-compared with diabetic control

Data shows that Fasting blood glucose sugar level is reduced in test drug group as compared to Diabetic control group and it is not statistically significant

Data shows that Fasting blood glucose sugar level is reduced in test drug double dose group and the change is statistically significant.

Table No. 30 HbA1c in percentage

Groups		**% change**
Normal control	3.43±0.16	
Diabetic control	8.58±0.43**	150.14↑ @
Standard	3.90±0.20**	54.54 #↓
Test 1	7.18±0.60	16.31#↓
Test 2	7.66±0.44	10.72#↓
Non diabetic	3.58±0.25	4.18 @↑

Data: MEAN ± SEM, **P<0.01. @-Compared with normal control. #-compared with diabetic control

Data shows that Hb1Ac level is reduced in test drug group and test drug double dose group as compared to Diabetic control group and it is not statistically significant

Table No. 31 Mean Fasting Blood Glucose in mg/dL

Groups		**% change**
Normal control	28.33±5.49	
Diabetic control	228.33±14.22**↑	87.59 @
Standard	43.8±6.80**↓	80.81 #↓
Test 1	151.2±19.38**↓	33.78 #↓
Test 2	169.00±14.76**↓	25.98 #↓

Non diabetic	36.28±9.10↑	28.06 @↑

Data: MEAN ± SEM, **P<0.01, @-Compared with normal control, #-compared with diabetic control

Data shows that mean fasting blood glucose level is reduced in test drug group and test drug double dose group as compared to Diabetic control group and it is statistically significant.

Table No. 32 Level of SGOT in IU/I

Groups :		**% change**
Normal control	118.66±3.90	
Diabetic control	145.00±12.38	22.19 @↑
Standard	157.0±14.45	7.64 #↑
Test 1	124.14±8.44	14.38 #↓
Test 2	141.0±14.77	2.75 #↓
Non diabetic	98.25±2.98	17.20 @↓

Data: MEAN ± SEM, @-Compared with normal control, #-compared with diabetic control

Data shows that SGOT level is reduced in both test drug group and test drug double dose group as compared to Diabetic control group and it is not statistically significant.

Table No. 33 Level of SGPT in IU/I

Groups		**% change**
Normal control	80.33±2.20	
Diabetic control	101.16±3.15*	25.93 @↑
Standard	66.6±1.83	51.89 #↓
Test 1	129.57±13.05	28.08 #↑
Test 2	89.62±10.54	11.40 #↓

Non diabetic	45.75±2.57*	43.04 @↓

Data: MEAN ± SEM, *P<0.05, @-Compared with normal control, #-compared with diabetic control

Data shows that SGPT level is has increased in test drug group as compared to Diabetic control group and it is not statistically significant. Data shows that SGPT is reduced in test drug double dose group and the change is not statistically significant.

Table No. 34 Level of ALP KAU/dL

Groups		**% change**
Normal control	450.33±22.78	
Diabetic control	1119.16±140.69**	147.85@↑
Standard	343.6±41.94**	69.29#↓
Test 1	1398.57±85.03	24.96#↑
Test 2	996.25±83.81	10.98#↓
Non diabetic	365.87±46.23	18.75@↓

Data: MEAN ± SEM, **P<0.01, @-Compared with normal control, #-compared with diabetic control

Data shows that ALP level is increased in test drug group as compared to Diabetic control group and it is not statistically significant. Data shows that ALP level is reduced in test drug double dose group and the change is not statistically significant.

Table No. 35 Level of TP in mg/ml

Groups :		**% change**
Normal control	5.86±0.21	
Diabetic control	5.96±0.23	1.70@↑
Standard	6.76±0.44	13.42#↑

Test 1	6.45±0.23	8.22#↑
Test 2	5.46±0.13	8.38#↓
Non diabetic	6.35±0.16	8.36@↑

Data: MEAN ± SEM, @-Compared with normal control, #-compared with diabetic control

Data shows that TP level is increased in test drug group as compared to Diabetic control group and it is not statistically significant. Data shows that ALP level is reduced in test drug double dose group and the change is not statistically significant.

Table No. 36 Level of ALB in g/dL

Groups		**% change**
Normal control	3.73±0.11	
Diabetic control	3.08±0.16**	17.42@↓
Standard	3.80±0.11	23.37#↓
Test 1	2.95±0.15	4.22#↓
Test 2	2.65±0.06	13.96#↓
Non diabetic	2.95±0.04**	20.91@↓

Data: MEAN ± SEM, **P<0.01, @-Compared with normal control, #-compared with diabetic control

Data shows that ALB level is decreased in test drug group and test drug double dose group as compared to Diabetic control group and it is not statistically significant.

Table No. 37 Level of GLO in g/dL

Groups		**% change**
Normal control	2.13±0.30	
Diabetic control	2.88±0.12	35.21@
Standard	2.96±0.34	2.77#↑
Test 1	3.48±0.31	20.83#↑
Test 2	2.96±0.24	2.77#↑

Non diabetic	3.38±0.16**	58.68@↑

Data: MEAN ± SEM, **P<0.01, @-Compared with normal control, #-compared with diabetic control

Data shows that GLO level is increased in test drug group and test drug double dose group as compared to Diabetic control group and it is not statistically significant.

Table No. 38 Level of T.Bil in mg/dL

Groups		**% change**
Normal control	0.16±0.00	
Diabetic control	0.21±0.03	31.25@↑
Standard	0.12±0.00	42.85#↓
Test 1	0.52±0.04**	147.61#↑
Test 2	0.66±0.03**	214.28#↑
Non diabetic	0.72±0.01**	350@↑

Data: MEAN ± SEM, **P<0.01, @-Compared with normal control, #-compared with diabetic control

Data shows that T. Bil level is increased in test drug group and test drug double dose group as compared to Diabetic control group and it is statistically significant

Table No. 39 Level of D.Bil in mg/dL

Groups		**% change**
Normal control	0.16±0.00	
Diabetic control	0.19±0.12	18.75@↑
Standard	0.06±0.02	68.42#↓
Test 1	0.06±0.01	68.42#↓

Test 2	0.06±0.01	68.42#↓
Non diabetic	0.13±0.02	18.75@↓

Data: MEAN ± SEM, @-Compared with normal control, #-compared with diabetic control

Data shows that D. Bil level is decreased in test drug group and test drug double dose group as compared to Diabetic control group and it is not statistically significant

Table No. 40 Level of Urea in mg/dL

Groups		**% change**
Normal control	33.0±1.46	
Diabetic control	80.5±3.33**	143.93 @↑
Standard	58.6±1.60**	27.20#↓
Test 1	41.71±4.21**	48.1#↓
Test 2	65.83±2.48**	18.2#↓
Non diabetic	43.5±2.13*	31.8@↓

Data: MEAN ± SEM,*P<0.05, **P<0.01, @-Compared with normal control, #-compared with diabetic control

Data shows that Urea level is decreased in test drug group and test drug double dose group as compared to Diabetic control group and it is statistically significant

Table No. 41 Level of Creatinine in mg/dL

Groups		**% change**
Normal control	0.3±0.03	
Diabetic control	0.65±0.02**	116.6@↑
Standard	0.94±0.04**	44.61#↑
Test 1	0.54±0.02	16.9#↓

Test 2	0.5±0.03**	23.07#↓
Non diabetic	0.53±0.01**	76.6@↓

Data: MEAN ± SEM, **P<0.01, @-Compared with normal control, #-compared with diabetic control

Data shows that Creatinine level is decreased in test drug group as compared to Diabetic control group and it is not statistically significant. Data shows that Creatinine level is decreased in test drug double dose group as compared to Diabetic control group and it is statistically significant

Table No. 42 Level of Cholesterol in mg/dL

Groups		**% change**
Normal control	33±0.36	
Diabetic control	32.83±4.51	0.51@
Standard	54.6±3.44	66.31#↑
Test 1	87±8.46**	167.4#↑
Test 2	79±9.43**	140.6#↑
Non diabetic	78.2±2.81**	136.96@↑

Data: MEAN ± SEM, **P<0.01, @-Compared with normal control, #-compared with diabetic control

Data shows that Cholesterol level is increased in test drug group and test drug double dose group as compared to Diabetic control group and it is statistically significant

Table No. 43 Level of TG in mg/dL

Groups		**% change**
Normal control	67±6.63	
Diabetic control	74±12.88	10.4@↑
Standard	140.2±36.45	89.45#↑

Test 1	146.14±19.78*	97.4#↑
Test 2	53.87±7.79	27.2#↓
Non diabetic	80.5±13.45	20.14 @↑

Data: MEAN ± SEM,*P<0.05, @-Compared with normal control, #-compared with diabetic control

Data shows that triglyceride level is increased in test drug group as compared to Diabetic control group and it is statistically significant. Data shows that triglyceride level is decreased in test drug double dose group as compared to Diabetic control group and it is not statistically significant

Table No. 44 Wt. of liver:

Groups		**% change**
Normal control	8.43±0.48	
Diabetic control	6.48±0.43*	23.1@↑
Standard	8.85±0.25**	36.5#↑
Test 1	6.48±0.30	0#
Test 2	6.55±0.40	1.08#↑
Non diabetic	7.10±0.55	15.7 @↓

Data: MEAN ± SEM,*P<0.05, **P<0.01, @-Compared with normal control, #-compared with diabetic control

Data shows that weight of liver is unchanged in test drug group as compared to Diabetic control group. Data shows that weight of liver has increased in test drug double dose group as compared to Diabetic control group and it is not statistically significant

Table No. 45 Wt. of kidney:

Groups		**% change**
Normal control	1.57±0.03	
Diabetic control	1.80±0.11	14.6@↑

Standard	2.07±0.14	15#↑
Test 1	1.56±0.16	13.3#↓
Test 2	1.23±0.01**	31.6#↓
Non diabetic	1.45±0.06	7.6@↓

Data: MEAN ± SEM, **P<0.01, @-Compared with normal control, #-compared with diabetic control

Data shows that weight of kidney has reduced in test drug group as compared to Diabetic control group and it is not statistically significant. Data shows that weight of kidney has decreased in test drug double dose group as compared to Diabetic control group and it is statistically significant

Table No. 46 Wt. of heart:

Groups		**% change**
Normal control	0.85±0.02	
Diabetic control	0.89±0.07	4.7@↑
Standard	1.02±0.06	14.6#↓
Test 1	0.71±0.08	22.22#↓
Test 2	0.64±0.01**	28.0#↓
Non diabetic	0.83±0.02	2.35@↓

Data: MEAN ± SEM, **P<0.01, @-Compared with normal control, #-compared with diabetic control

Data shows that weight of heart has reduced in test drug group as compared to Diabetic control group and it is not statistically significant. Data shows that weight of heart has decreased in test drug double dose group as compared to Diabetic control group and it is statistically significant

Table No. 47 Wt. of pancreas:

Groups		**% change**

Normal control	0.28±0.01	
Diabetic control	0.75±0.06**	167.8@↑
Standard	0.44±0.04**	41.3#↓
Test 1	0.64±0.07	14.6#↓
Test 2	0.58±0.04	22.6#↓
Non diabetic	0.39±0.01	39.2@↑

Data: MEAN ± SEM, **P<0.01, @-Compared with normal control, #-compared with diabetic control

Data shows that weight of pancreas has reduced in test drug group and test drug double dose group as compared to Diabetic control group and it is not statistically significant.

Anti-oxidant study:

Table No. 48 Protein estimation mg/ml

Groups		**% change**
Normal control	0.05±0.00	
Diabetic control	0.02±0.00*	60@↓
Standard	0.08±0.00**	300#↑
Test 1	0.06±0.00**	200#↑
Test 2	0.04±0.01	100#↑
Non diabetic	0.05±0.01	0@

Data: MEAN ± SEM,*P<0.05, **P<0.01, @-Compared with normal control, #-compared with diabetic control

Data shows that Protein estimation has increased in test drug group as compared to Diabetic control group and it is statistically significant.Data shows that Protein estimation

has increased in test drug double dose group as compared to Diabetic control group and it is not statistically significant.

Table No. 49 Catalase activity mmoles/min/mg protein

Groups		**% change**
Normal control	1.70±0.30	
Diabetic control	4.57±0.54**	168.8@↑
Standard	0.76±0.16**	83.3#↓
Test 1	1.72±0.21**	62.3#↓
Test 2	3.15±0.65	31.07#↓
Non diabetic	2.83±0.95	66.4@↑

Data: MEAN ± SEM, **P<0.01, @-Compared with normal control, #-compared with diabetic control

Data shows that Catalase activity has decreased in test drug group as compared to Diabetic control group and it is statistically significant.Data shows that Catalase activity has decreased in test drug double dose group as compared to Diabetic control group and it is not statistically significant.

Table No. 50 Glutathione peroxidase mmoles of glutathione/mg protein/min

Groups		**% change**
Normal control	18.53±2.90	
Diabetic control	50.45±1.84**	172.2@↑
Standard	14.63±2.79**	71#↓
Test 1	14.26±2.36**	71.7#↓
Test 2	27.25±8.86*	45.9#↓

Non diabetic	24.25±6.01	30.8@↑

Data: MEAN ± SEM,*P<0.05, **P<0.01, @-Compared with normal control, #-compared with diabetic control

Data shows that Glutathione peroxidase has decreased in test drug group and test drug double dose group as compared to Diabetic control group and it is statistically significant

Table No. 51 Lipid peroxidation mmoles of MDA formed/g wet tissue

Groups		**% change**
Normal control	1.01±0.04	
Diabetic control	2.06±0.11**	103.9@↑
Standard	1.55±0.09**	24.7#↓
Test 1	1.00±0.11**	51.4#↓
Test 2	0.98±0.07**	52.4#↓
Non diabetic	0.95±0.08	5.9@↓

Data: MEAN ± SEM, **P<0.01, @-Compared with normal control, #-compared with diabetic control

Data shows that Lipid peroxidation has decreased in test drug group and test drug double dose group as compared to Diabetic control group and it is statistically significant.

Chapter V

DISCUSSION

Discussion on concept:

Concept of *Upakrama* through *Samanya Vishesha Siddhanta*

Samanya Vishesha Siddhanta is a fundamental concept for the application of treatment. *Upakrama* (Treatment principles) in Ayurveda are based on the fundamental doctrine of *Samanya* and *Vishesha*. *Dosha* and *Dhatu Vaishamya* are brought back to *Samya* by using the effects exerted by *Dravya / Aushadha* by virtue of *Samanya* and *Vishesha*. The effect of *Samanya* on a *Dosha* can only be seen when there are no counteracting elements within the *Dravya*. Along with this the *Dravya* which may not be *Samanya* can still cause *Vrddhi* because of the *Prabhava*.

Vishesha can be of two kinds – *Viruddhavishesha* (stark opposite) and *Aviruddhavishesha* (merely dissimilar). *Viruddhavishesha* is a sure factor for decrease due to the presence of opposite qualities. Meanwhile, *Aviruddhavishesha* can be attributed to decrease only due to the observation that it does not contribute to increase. Any *Dravya* used in a *Upakrama* can subside *Dosha* when they are exactly opposite to *Dosha (Viruddha Vishesha)* and also any *Dravya* which is dissimilar in *Guna* (*Aviruddhavishesha*) can still cause *Shamana* because it stops the chain of *Samanya* which would have led to *Vrddhi* with respect to the bodily *Dhatu*, as they are being deteriorated in every smallest frame of time, they require constant supplementation, which is nothing but increase, attributed to *Samanaguna*. When the supplementation does not take place due to unmatched qualities (dissimilar/ *Aviruddhavishesha*), there is no net

increase and as a result it is still considered as decrease. Therefore, having dissimilar qualities is also considered as a cause for decrease.

Upakrama and *Pravuttir Ubhayasya Tu*

Upakrama (Treatment modalities) in Ayurveda are mediated through the *Dosha* hence the concept of *Dosha* becomes the most fundamental concept that needs to be understood.

The introduction to *Dosha* primarily starts with the description of *Guna* because they are the most important factor not only to understand the underlying pathology and design the treatment but also to assess the effectiveness of the treatment applied. There are *Dosha* which are both similar and dissimilar to each other in terms of *Guna*. Even though the *Guna* are dissimilar to one another but still the very nature of *Dosha* is such that they do not obstruct the existence of the other which help them to co–exist in a state of health. But when there is excessive use of a particular *Dravya* with certain attributes then either one or more *Dosha* which are similar undergo *Vrddhi* and eventually leading to the pathology. So when the disease is manifested there is not only one *Dosha* involved instead there could be combination which is because of the similarity that exists between the two *Dosha* in terms of their *Guna*. This kind of presentation is called as *Sansarga* and *Sannipata* of *Dosha*. Because of the contrary *Guna* of *Dosha* a *Dravya* not only increases the *Dosha* which is similar but also counteracts the *Dosha* whose *Guna* is opposite. Also except for the *Nanatmaja Vyadhi* all the diseases have the involvement of *Tridosha* and hence the application of treatment has to be done in such a way that only the *Dosha*

which is impaired needs to be tackled without disturbing the *Dosha* which is in *Sama Avastha*. Therefore while treating the disease where there is combination of two or three *Dosha* one has to give priority to the *Dosha* which has undergone derangement but keeping in mind that the other *Dosha* which is associated should not undergo derangement. Because of this, the application of a *Upakrama* (Treatment modalities) should be done in caution keeping the above factor in mind.

Upakrama* aiming at *Shuddha Chikitsa

Because the disease is an outcome of more than one *Dosha* it should always be understood that using any *Upakrama* can lead to pacification of one/ more *Dosha* on one hand, but on the other, gives a possibility of vitiating the other. This implies that for each *Upakrama,* an end point must be determined so that the other *Dosha* do not get affected. The physician is therefore required to apply moderation in the treatment protocol. This can be understood better through the concept of *Chikitsa* for *Sansarga* and *Sannipata Rogas*. The *Upakrama* specific to one *Dosha* must in no way interfere that of the other *Dosha*. To manage such diseases in such a way that pacification of two contradictory *Dosha* without disturbing one another a general principle has been mentioned below –

- *Vata Pitta – Greeshma Ritu Carya*
- *Kapha Vata - Vasanta Ritu Carya*
- *Kapha Pitta – Sharad Ritu Carya*

Status of *Dosha* in Planning the *Upakrama*

Cayaavastha:

The *Dosha* in *Cayaavastha* must be recognized and pacified right away. This emphasizes the time of *Upakrama*, meaning that the *Dosha* has to be recognized and treated during the earliest stage of *Dushti* as possible. The *Dosha* are to be pacified during the stage of *Caya*. If it is left untreated during this stage, it progresses to *Kopa*. In such a case it must be pacified using such a treatment that does not lead to the aggravation of the other *Dosha* which have not yet attained *Prakopa*.

Kupitaavastha:

The *Kupitadosha* can be of two kinds – *Prakrtisamasamaveta* and *Vikrtivishamasamaveta.* The former is called so when the *Swarupa* of both the *Dosha* and the *Roga* are similar. The latter is called so when the *Swarupa* of the *Dosha* and the *Vyadhi* are mutually contradictory. Such as *Kaphajwara*, where the aggravated *Guna* is *Sheeta*, which is also the *Guna* of *Kapha*. In such a case, the treatment should not be given as expected, using *Ushnaguna*. This is because *Ushanguna* is the *Swarupa* of *Jwara*. Therefore, the treatment of *Kaphajwara* should be such that the *Ushnaguna* is not strong which strikes a balance between the *Dosha* and the *Vyadhi.*

Tiryakgatadoshas are called so when they are situated in sites from where their elimination is not possible. The understanding of *Koshta* to *Shakha Gati* of *Dosha* becomes important to understand the *Samprapti* of a *Vyadhi*. In such a case *Tiryakgatadoshas* cannot be removed through external or internal *Shodhana* therapies

and have the capacity to cause diseases even after an exceptionally long time. The *Gati* of *Dosha* from *Shakha* to *Koshta* helps in understanding the underlying mechanism of the *Chikitsa Sutra* mentioned for the diseases and thereby helping to choose the right *Upakrama*. Therefore, they must not be given immediate *Shodhana* therapy and thereby aid in accurate understanding of the *Avastha* of the *Vyadhi*. For this the physician assess the status of the *Deha* i.e., the physical strength and the status of the *Agni,* i.e., the strength of the *Agni*. The *Tiryakgatadosha* can be managed with either of the two methods, depending upon their intensity (*Taratama bhava* of *Dosha*). If the degree of *Dushti* is less then they can be pacified through appropriate *Shamanaprayoga* as mentioned in the *Shastra*. If *Dosha* which has undergone maximum *Dushti* then *Shodhana* has to be selected and by bringing the *Dosha* to *Koshta* through appropriate methods such as *Sneha* and *Sweda*, after which they become eligible to be eliminated through *Shodhana* such as *Vamana, Virechana*. If *Dosha* are *Sama* then appropriate *Upakrama* like *Deepana Pachana* has to be selected and then one can proceed with previously mentioned *Upakrama*.

Based on the *Taratama Bhava* of *Dosha*

The *Upakrama* has to be planned based on the degree of disturbance of *Dosha* based on which priority has to be given to the *Dosha* which is disturbed to greater degree followed by the next *Dosha*. If the degree of disturbance that the different *Dosha* have undergone is at the same level then one should give importance to the *Sthana* and classify the *Dosha* as *Sthanika* and *Agantu* and *Sthanika Dosha* should be treated first. But if the

Bala of *Agantu Dosha* is more the treatment has to be given to *Agantu Dosha*. Therefore, the concept that lies here is that, the untreated *Agantudosha* can gain strength and can even overpower the *Sthanadosha* to cause grave diseases. Here, the *Agantudosha* can be one or two, never three.

Selection of *Rasa as Upakrama*

For *Vata* the order is *Lavana, Amla* and *Madhura*. For *Pitta* it is *Tikta, Madhura* and *Kashya*. For *Kapha* the order is *Katu, Tikta* and *Kashaya*.

Dvividhopakrama / Shadupakrama

The classification of *Upakrama* into two and six even though appear to be distinctive but on a closer observation one can identify that they are not different. Instead *Vagbhata's Dvividhapakramaneeyam* has its base from *Charaka*'s *Santarpaneeyam Adhyayam* and further elaborated in *Langhaneeya Brimahaneeyam Adhayayam*. So *Upakrama* are basically of two types *Santarpana (Brimahana)* and *Apatarpana (langhana)* which are further classified into six which have to be adopted based on the *Dosha Bala* and *Deha bala*.

Apatarpana or *Langhana* is classified into two types – *Shodhana* and *Shamana* and this classification is done exclusively on the basis of the status of *Dosha*, degree of vitiation and *Bala* of the patient.

Shodhana is more effective line of treatment but also needs a lot of caution while selecting the patient. *Dosha* always need to be eliminated through the nearest orifice and hence based the on this principle *Shodhana* is again classified into five.

Shamana on the other hand is less effective as compared to *Shodhana* but still it requires minimal after care as compared to *Shodhana*. The further classification of *Shamana* is done based on extent of the effectiveness on the *Dosha*. So in order the effectiveness is less as follows – *Pachana, Deepana, Kshut, Trushna, Vyayama, Atapa and Maruta*

Exception of *Shodhana and Shamana*:

Even though *Shodhana* and *Shamana* are the types of *Langhana* but in some cases *Brumhana* also can act as *Shodhana* and *Shamana.* *Dugdha* which is a *Brumhana Dravya* can be used for *Shodhana* in case of *Vata* or *Pitta* associated with *Vata* which in such cases it acts as a *Shamana Dravya*.

Bhoutik Composition

Generally all *Brimhana Dravya* have *Pruthvi* and *Jala* and all *Langhana Dravya* have *Vayu, Akasha* and *Agni*.

Exceptions to the rule of *Bhoutik* composition

The *Dravya* which are predominant of *Bhouma* and *Apa* which show *Apatarpana* effect

- *Yavaka*
- *Masura*
- *Makushtha*
- *Tanduliya*

The *Dravya* which are predominant of *Agni*, *Vayu* and *Akasha* which show *Santarpana* effect like *Vrushyatva.*

- *Shunthi*
- *Pippali*

Brimhana is generally indicated in all the conditions where *Vata* is dominant, *Dhatu Kshaya* is seen and in the population who are vulnerable. *Langhana* is generally indicated in conditions where *Kapha* is dominant, where there is *Atipurana* of *Dhatu*. *Shodhana Rupi Langhana* can be advised in two ways –

1. Without doing the *Purvakarma* like *Senhana* and *Swedana* which is considered as *Sadya Shodhana*. While doing *Sadya Shodhana* one should carefully analyze the status of the *Dosha* which should be in *Pradhana Avastha*. *Pradhana Avastha* of *Dosha* refers to the *Dosha* which should be in *Utklishta* and *Nirama* along with the presence of *Dosha* on the place where *Dosha* can be easily removed.
2. The second method where the *Pradhana Avastha* of the *Dosha* can be brought in by doing the *Purva Karma* line – *Deepana, Pachana, Snehana* and *Swedana*.

Shamana Rupi Langhana

The classification of *Shamana Rupi Langhana* is done on the basis of the degree of effectiveness with respect to degree of *Dosha Dushti* and *Bala* of the patient. *Deepana Pachana* involves the administration of medicine hence the effect is more as compared to the others hence it is indicated in the conditions where the *Dosha Dushti* is comparatively more and the *Bala* of the patient is good. *Kshudha* and *Pipasa* even though do not involve the administration of medicine but still requires the energy to follow hence it is advised in moderately disturbed *Dosha* and where the bala of the patient is moderate.

Atapa and *Maruta* are very mild in the effect hence is advised in the condition where *Dosha* are mildly disturbed.

Interjection of *Langhana* and *Brumhana*

Those conditions which are indicated for *Langhana* like – *Mehadosha, Amadosha,* etc. should never be selected for *Brumhana.*

The conditions which are indicated for *Brumhana* if come under the category of *Langhanasadhya* should be subjected for *Mridulanghana* in a balanced way with a due consideration to *Desha, Kala,* etc.

Concept of *Snigdhasantarpana* and *Rukshasantarpana*

Santarpana is of two kinds – *Snigdhasantarpana* and *Rookshasantarpana* which is evident with the word "सक्तूनांषोडषगुणोभागःसन्तर्पणंपिबेत्". Here the diseases are arising because of *Snigdhasantarpana*. *Snigdhasantarpanajavyadhi* occur because of intake of *Snigdha Dravya* like *Navanna* and other *Dravyas* and doing those activities which bring the effect of *Santarpana* like not involving any kind of physical exercise and no immediate measure is taken to nullify the effect.The word *Snigdhadi* have been used by *Acharya Charaka* with intention of clarifying the fact that there are substances which are not *Snigdha* and yet can be *Santarpana* like *Shaktu*. Hence the *Santarpana* should not just restrict to the derivation as *Truptikaraka* like for e.g. here in the same context there is mention of the use of *Shaktu* along with 16 parts of water for the purpose of *Santarpana*.

The *Hetu* mentioned under *Santarpana Janya Vyadhi* mostly have *Kapha Vardhaka, Vatahara* and *Medo Dushtikara* property. They are also *Guru* in nature and affect the

Agni. Hence most of diseases occurring have to be treated by *Vamana* as first line of treatment followed by other *Shodhana* like – *Virechana*, *Raktamokshana* and other external line of Treatment like – *Dhoomapana Svedana* and *Churna Pradeha.* Along with these one should also advise – *Upavasa* and *Vyayama.*

In case of *Apatarpana Janya Vyadhi* one needs to carefully analyse the *Agni* because the prolonged use of *Apatarapna Janya Hetu* can lead to *Agnimandya*. Therefore direct administration of *Santarpana* may not be beneficial. So there are two ways of administering *Santarpana* – one in case of *Sadya Ksheena* in whom *Agni* is not affected much one should administer *Sadya Santarpana*, in case of *Chiraksheena* in whom *Agni* is disturbed one should first improve *Agni* and then gradually administer *Santarpana*.

Santarpana Janya Vyadhi:

Pandu: Even though the end result of the disease is *Kshaya* in *Sneha guna* of *Ojas* leading to *Bala Hani, Varna Hani* and appears to be treated by *Snatarpana* but on a close observation it becomes clear the origin of *samprapti* starts with *Pitta Vrddhi* with a diminished *Dhatvagni* which is not capable of converting the *Ahara Rasa* into *Rakta Dhatu.* So unless this root cause is not corrected it is not possible to reverse the disease. This gets even justified with the *Chikitsa Sutra* which is mentioned as *Teekshna Urdhva Adho Dosha Nirharana* as main treatment principle.

Jwara: The *samprapti* of *Jwara* is very clear as the factors involved *Samprapti Ghtaka* are *Amashaya* as a *Sthana, Agni* is *Dushta, Ahara Rasa* is *Apakva*, involved *Srotas* is

Rasavaha Srotas and *Srotorodha* is the *Sroto Dushti Prakara*. All these directly indicate that the primary line of treatment should be *Apatarpana.*

Kushtha: The *Nidana* of *Kushtha* are majorly *Nindita Ashana* which are primarily *Agnidushtikara Nidana*. Hence the *Tridosha* are equally responsible for the manifestation of the disease. Hence *Apatarpana* is the primary line of treatment while attention needs to be paid for the *Gati* of *Dosha.*

Tandra Buddhi Moha: The impact of *Tamoguna* and *Dushana* of *Rakta* by the *Tridosha* are the primary factors for the manifestation of these conditions hence *Apatarpana* becomes the primary line of treatment.

Drugs used in the treatment of *Santarpana Janya Vikara*

The close observation on the *Yoga* mentioned for the management of *Santarpana Janya Vikara* are mostly predominant of

1. *Rasa – Tikta Katu*
2. *Guna – Laghu Ruksha Teekshna*
3. *Veerya – Ushna*
4. *Vipaka – Katu*
5. *Doshaghnata – Kapha Pitta Shamaka* or *Tridosha Shamaka*
6. *Karma – Deepana, Pachana, Grahi, Lekhana*

The disease is rectified by correcting the *Agni*, converting the *Sama Dosha* into *Nirama Dosha*, by doing *Sroto Shodhana*.

Apatarpana as *Upakrama in Prameha*

Prameha Samprapti

Due to *Nidana*, all of which aggravate *Kapha* the first *Dushya* contributing to the *Samprapti of Prameha* is *Medas*. The loss of compactness and integrity of *Medas* makes way for progress in the *Samprapti*, involving *Sharira Kleda* and *Mansa*. The increase in quantity of *Kleda* and *Mansa*, along with the excessively vitiated *Medas* and *Kapha* disrupt the entire physiology of *Dhatu Parinama* in the body, leading to *Vikrti*. At a later stage, owing to irreversibility of the *Samprapti*, the deranged *Medas* and *Sharira Kleda* involves *Mutravaha Srotas* and *Vasti* leading to *Vikrti* in the quality and quantity of *Mutra*. According to the variations in the *Nidana* pertaining to *Kapha, Pitta* and *Vata*, there is involvement of the corresponding dosha, which thereby leads to the 20 types of *Prameha*.

***Prameha Chikitsa Sutra* based on the type of *Prameha Rogi*.**

It has been observed that a *Pramehi* can be either *Sthula* and *Balavan* or *Krsha* and *Durbala*. *Sthula Pramehi*, in possession of good strength is given *Shodhana* therapies in case of excessive *Dosha* involvement. *Krsha Pramehi* who does not have good strength cannot be given *Shodhana* therapies, and is therefore treated with *Shamana Chikitsa*. In both the kinds, the first step is to bring balance about the *Doshas* by means of Apatarpana (*Shodhana* or *Shamana*, using *Apatarpana* drugs). Once the *Dosha Samya* is attained, *Prakrtisthapana* is achieved by incorporating *Brmhanam.*

Prolonged usage of *Apatarpana* can have deleterious effects on the patient leading to diseases such as *Gulma, Kshaya, Mehana Shula, Vasti Shula,* and *Mutra Graha*.

Shamana Chikitsa is given using formulations such as *Mantha, Kashaya, Churna, Leha, Laghu Bhakshya, Odana, Saktu, Supa, Mansa Rasa, Yusha* and *Anna. Yava, Pratuda Mansa, Prasaha Mansa, Vatya. Mudga, Tikta Shaka, Purana Shali* and *Trna Dhanya* are the ingredients used to prepare these formulations.These are all *Apatarpana* in nature, so that both *Dosha Kshaya* and *Kshina Dhatu Vrddhi* are achieved. This kind of *Apatarpana* can be understood as restrained *Antarpana*, which is exactly what is required in a disease like *Prameha.*

Mode of treatment indicated in *Medoroga* can be adopted in *Prameha* – which is a carefully structured combination of *Apatarpana* initially followed by restrained *Santarpana (Rukshana).*

The role of *Apatarpana* in the initial stages of *Kaphaja* and *Pittaja Prameha* is well-appreciated throughout the *Chikitsa of Prameha*. But the most important determining factor is the *Nidana* and *Kala*. Both are to be thoroughly assessed before administering the intensity and method of *Apatarpana Chikitsa* in *Prameha*.

Discussion on Results

Effect on Serum Glucose, HbA1c and Mean Sugar levels

Effect on Serum Glucose level: The hypoglycemic effects of *Apatarpana* by *Triphala Sadhita Yava* serum glucose level markedly reduced in in Test 2 group as opposed to Test group 1.

Effect on Mean Glucose level: Mean sugar levels significantly reduced in both Test 1 and Test 2 groups which again is because of beta glucan which helps in significant reduction.

Yava being a dietary item needs to be introduced as diet to reduce blood sugar level. The soluble dietary fiber beta glucan is rich in barley which helps in reducing the acute glucose and serum cholesterol levels due its slow movement in the intestine and also delay the digestion and absorption of nutrients due its high viscosity.[37]The diet advised in *Medo Vikara* like *Sthoulya* is *Guru* and *Atarpana*.

Effect on Glycated hemoglobin (HbA1c) level: HbA1c levels reduced in both Test 1 and Test 2 groups but both are not statistically significant. This could be possible because of the unusual presence of various hemoglobin components in the rat and determination of glycated hemoglobin is considered as difficult hence it is considered as an unreliable in rats. [38]

Effect on Liver Function Tests

Effect on Serum Glutamic Oxaloacetic Transaminase (SGOT) level – There was a marked reduction in elevated levels of SGOT levels in both Test 1 and Test 2 groups and both were statistically significant.

Effect on Serum Glutamic Pyruvic Transaminase (SGPT) level – There is marked reduction in the elevated levels of SGPT in Test 2 group as opposed to Test 1 but the change is not statistically significant.

Effect on Alkaline Phosphatase (ALP) level – There was reduction in the ALP levels in Test 2 group whereas there was increase in the Test 1 group. However these changes were not statistically significant.

SGOT and SGPT are considered as cytosolic marker enzymes which indicate hepato- cellular necrosis which get released into the blood stream hence they act as an indicator of hepatic damage. This liver damage is caused because of concentration of free radicals in plasma and which leads to oxidative stress.[39]The test drug when administered in the double dose had a significant change in reducing the SGOT levels as compared to standard drug which indicates that it has hepato - protective effect. Barley is rich in phenolic compounds like ferulic acid, p–coumaric acid, caffeic acid protocatechuic acid, flavan–3–ols and flavanols. Higher the phenolic compound higher is the anti-oxidant effect which leads to the hepato protective activity. [40]

Effect on Total Protein (TP) levels: Total protein value reduced Test 2 group even though there was an increase in Test group1 and range was similar to standard. However the increase in both groups was not statistically significant.

Effect on Albumin (Alb) levels – The albumin levels did not vary and were within the normal range.

Effect on globulin (GLO) levels – globulins are the group of proteins which include enzymes, antibodies (immunoglobulins) and other proteins and these are produced in the liver. These help in transport of nutrients and fight infections. The Globulin levels did not vary and were within normal range. There was a marked reduction in the globulin levels in both Test 2 and standard group but this was not statistically significant.

Effect on Total Bilurubin (T Bil) and Direct bilirubin (D Bil) Levels – There was marked increase in the Total Bilurubin (T Bil) levels in the Test 1 and Test 2 group. However there was reduction in the Direct bilirubin levels in both the groups which was not statistically significant. This is generally seen in short term streptozotocin – diabetic rats because there is increased production of bilirubin and also hepatic conjugation which further leads to excretion of the pigment. This the consequence seen as an effect of diabetes induced by streptozotocin. [41]

Effect on Renal Function Tests

Effect on Urea level: The blood Urea levels markedly reduced in both the Test 1 and Test 2 groups and both were statistical significant.

Effect on Creatinine level: The Serum creatinine level markedly reduced in both Test 1 and Test 2 group and the statistical significance was seen in Test 2 group.

Diabetic Kidney disease (DKD) eventually progresses with glomerular hyperfiltration and low glomerular filtration rate. Hyperglycemia induces migration of peritubular pericytes from the peritubular capillaries to the interstitial space leading arteriosclerosis and also accelerates tubular interstitial changes by enhancing the transition of pericytes into myofibroblasts.[42] Intake of barley reduces post prandial hyperglycaemia and the prebiotic effect of beta glucan alters the intestinal bacterial flora improves the DKD. Trimethylamine – N – Oxide is a small amine plasma compound has a close association with GFR. Beta glucan a major constituent in barley is considered is effective in reducing serum TMAO levels which indicates that Barley has the potential to increase GFR.[43]

Effect on Lipid Metabolism

Effect on Cholesterol: There was increase in Blood cholesterol level in all the groups and all the changes were statistically significant.

Effect of Triglycerides: The triglycerides increased in all the groups except for Test 2 group but this change is not statistically significant.

This is considered to be normal as the base level of Cholesterol and triglycerides would increase after 180 minutes of ingestion.

Weight of Liver

There is decrease in the weight of the liver in both the groups but this decrease is not statistically significant. The increase in the size of the liver is possibly because of increased triglyceride accumulation in the liver because of the STZ treatment. But the use of Barley would probably reverse the change.

Weight of Kidney

There is decrease in the weight of the Kidney in both the groups and the decrease in Test group 2 is statistically significant. The increase in the size of the Kidney is possibly because of the STZ treatment. But the use of Barley would probably reverse the change.

Weight of Heart

There is reduction in the weight of heart in all the groups and the decrease in Test group 2 is statistically significant. The increase in the size of the heart is possibly because of the STZ treatment. But the use of Barley would probably reverse the change.

Weight of Pancreas

There is decrease in the weight of the pancreas in both the groups and this decrease is not statistically significant.

The reduction the weight could be because of disruption of pancreatic islets and selective destruction of insulin producing cells induced because of STZ treatment.

Effect on Antioxidant property:

Effect on Protein estimation: Protein has increased in both the groups and it is statistically significant in Test Group 1. Peptides have a broad scope of bioactivities out of which antioxidation is one. During the metabolism the reactive oxygen species or free radicals produced by oxidation reactions causes oxidative stress. The peptides derived from natural proteins which have characteristic peptide chemical structures influence the peptide antioxidant activities thereby prevent the non-communicable diseases like DM.[44]

Effect on Catalase activity: There is decrease in catalase activity of both groups but the decrease in Test Group 1 is statistically significant. This reduction could be because of the STZ treatment. However there increase in the Test group two as compared to diabetic induced group which is also statistically significant. Catalase (hydrogen peroxidase / hydrogen peroxide oxidoreductase) is an antioxidant enzyme which protects cells from toxic effects of high concentrations of hydrogen peroxide $H_2O_2$by catalysing the decomposition into molecular oxygen and water without producing the free radicals. Oxidative stress is an integral part of Diabetes mellitus and the raise in catalase activity indicates the increase in the antioxidant property. [45]

Effect on Glutathione peroxidase: There is significant reduction glutathione peroxidase both the groups however the reduction in Test Group 2 is comparatively less which is statistically significant also. Glutathione peroxidase is an enzyme which aids in reduction of lipid hydroperoxides to their corresponding alcohols and to reduce free hydrogen peroxide to water thus aiding in antioxidant property. [46]

Effect on Lipid peroxidation: There is marked reduction on Lipid Peroxidation in both the groups which is statistically significant. Lipid peroxidation is process under which the oxidants such as free radicals attack lipids containing carbon – carbon double bonds mainly polyunsaturated fatty acids. This process in which free radicals steal electrons from the lipids in the cell membrane resulting in cell damage.[47] So reduction in the lipid peroxidation prevents the cell damage which can be seen in both the groups.

Histopathology

Liver- Injection of STZ resulted in mild fatty changes, sinusoidal dilatation and diffused degenerative changes in hepatocytes.

Liver sections from standard group exhibited normal cytoarchitecture in majority of the rats, however, fatty degenerative changes along with sinusoidal dilatation, bile stasis was observed in few sections.

Liver sections from Test 1 administered groups exhibited mild to moderate fatty changes and sinusoidal dilation.

In sections from Test 2 group Mild cell infiltration in sections from three rats and bile stasis with bile duct dilation, mild fatty changes in section from one rat was observed.

Liver sections from non diabetic group exhibited normal cytoarchitecture in majority of the rats, however, mild fatty degenerative changes along with sinusoidal dilatation, bile stasis was observed in sections.

Pancreas: Scanning of sections from STZ control group exhibited few islets of small size with much reduced granulation, cellularity was less, vacuolization was observed.

In sections from standard group- medium to large sized islets with good cellularity and granulation, mild vacuolization was observed.

In sections from Test 1 administered group- medium to large sized islets with high cellularity and much reduced vacuolization was observed.

In sections from Test 2 administered groups- higher number of medium to large sized islets with good cellularity and mild vacuolization was observed.

In sections from non diabetic group- mild to medium to sized islets with mild cellularity and very less reduced vacuolization was observed.

Chapter VI

CONCLUSION

CONCLUSION

1. Concept of *Samanya Vishesha* forms the fundamental concept of *Apatarpana Upakrama* with key concepts like - *Viruddha Vishesha*, *Aviruddha Vishesha* and *Pravruttir Ubhaysaya Tu* which form the base for moderate use of *Upakrama*, and in deciding the end point of *Upakrama*.
2. *Shadupakrama* is an extension of *Dvividhopakrama* which has its base from *Santarpaneeyam Adhyayam*.
3. *Prameha* as *Santarpana Janya Vyadhi* have the dominance of *Kapha* with all its *Ashraya Bhava* which needs to be tackled by *Apatarpana* in moderation with due consideration to *Pravruttir Ubhayasya Tu*.
4. *Triphala Sadhita Yava* being *Apatarpana yoga* has the ability to reduce *Kapha, Meda* while taking care of the *Vata.*
5. *Triphala Sadhita Yava* as a *Apatarpana Upakrama* is effective in *Prameha* by reducing the blood glucose along with hepatoprotecitve, nephroprotective and anti- oxidative effects.

SUMMARY

SUMMARY

This Thesis work entitled with **"An in – vivo study on the concept of Apatarpana in Santarpanajanya Vikara with special reference to Prameha "**comprise of different parts namely Introduction, Objective, Review of literature, Methodology, Sample Size Estimation, Observation, Results, Discussion and Conclusion.

INTRODUCTION

This part includes brief introduction about the concept of Apatarpana Upakrama

REVIEW OF LITERATURE

In this the fundamental concepts that form the foundation for Apatarpana like Samanya Vishesha, Treatment of Dosha based on various criteria, exploration of Santarpaneeyam Adhyayam, Dvividhopakramaniya – Shadupakrama, Prameha and Diabetes Mellitus was carried out.

METHODOLOGY

It was planned under two phases

Conceptual study:

1. Review of the fundamental concepts which will act as the frame work for the concept under study that is Apatarpana.
2. Review of the concept of Apatarpana as a Upakrama along with Santarpana as a Hetu and also as a Upakrama.
3. Review on the Prameha Santarpana Janya Vyadhi and Triphala Sadhita yava as Apatarpana Upakrama.
4. Drug Review on Yava and Triphala

Applied study

Pharmaceutical study: This included drug procurement, authentication and preparation of Triphala Sadhita Yava.

Experimental study: It was carried to study the effect of Apatarpana (Triphala Sadhita Yava) on Prameha by using Triphala Sadhita Yava on 24 parameters.

OBSERVATION AND RESULTS

The observations were tabulated and subjected for statistical test and results were computed by Dunnett's multiple comparison't' test using Graph Pad Prism 3. A $p<0.05$ was considered as statistically significant.

DISCUSSION

It was carried out two sections as follows-

Concept: The links between the fundamentals were established to understand Apatrapana as Upakrama and its role in the management of Santarpana Janya Vikara wrt to Prameha.

Applied: The role of Apatarpana Upakrama in the management of Prameha (Diabetes mellitus) with various parameters and its impact in modifying the samprapti were discussed.

CONCLUSION

Conclusions drawn from the several sections of the study are given.

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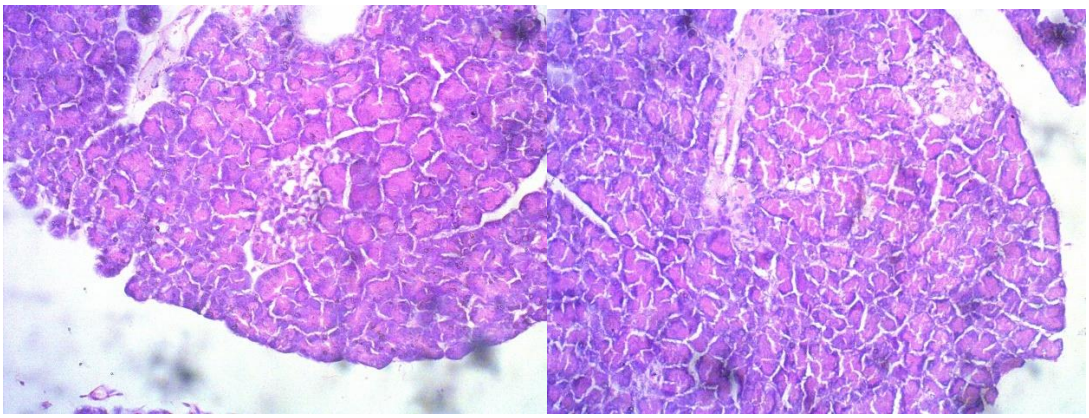
ANNEXURE

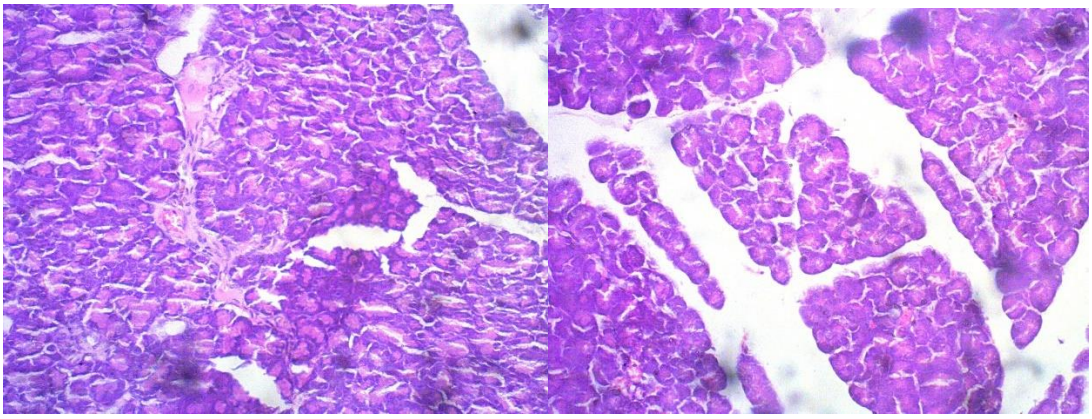
Annexure 1. Histopathology

Pancreas: Group I Diabetic Control Group



R - 1

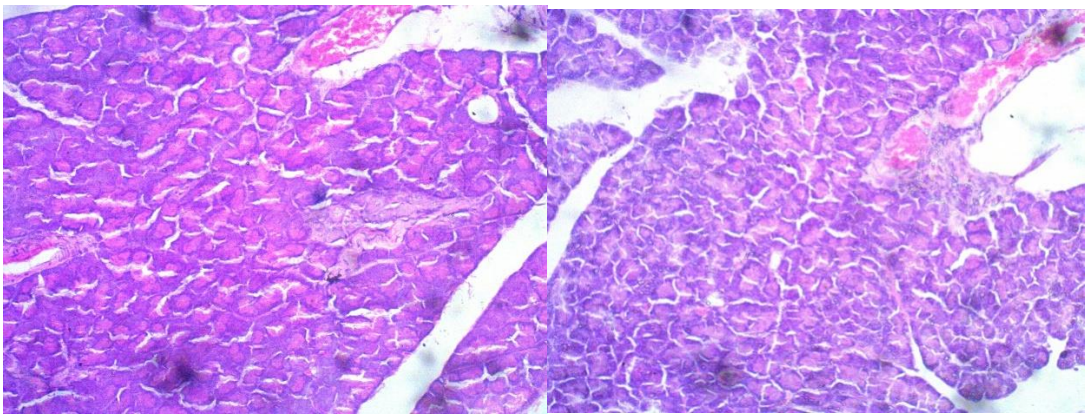
R - 2



R - 3

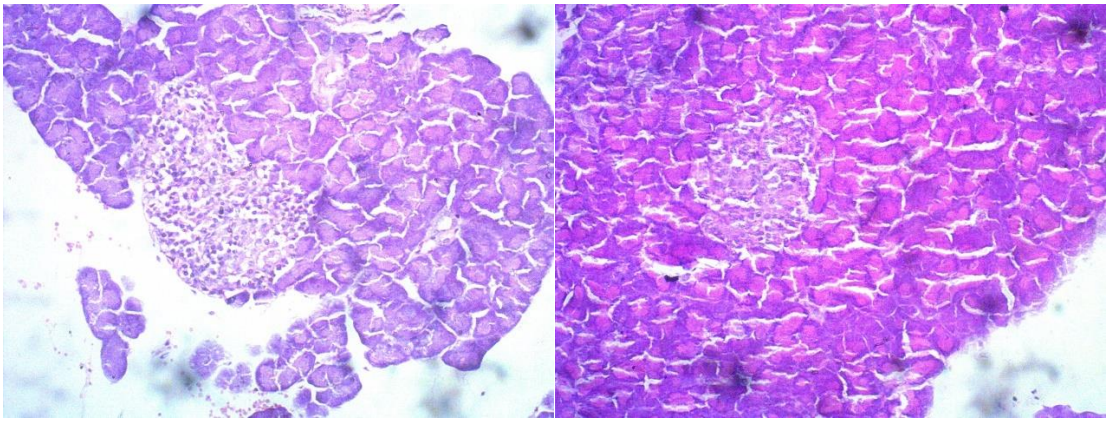
R - 4

Pancreas: Group II Reference Standard



R - 1

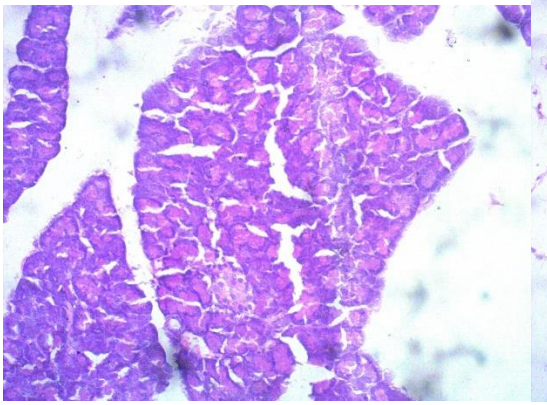
R - 2



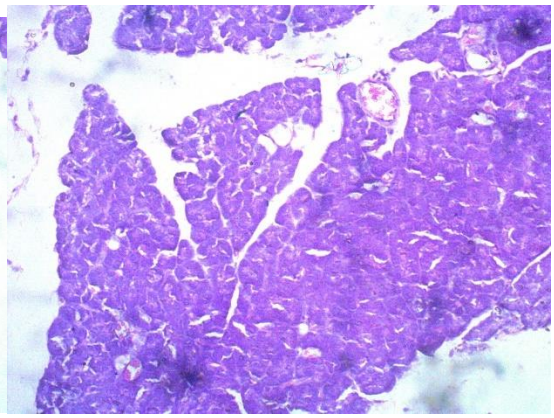
R - 3

R - 4

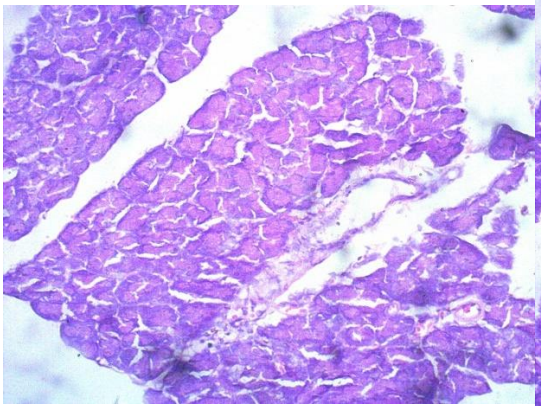
Pancreas: Group III Test -1 group



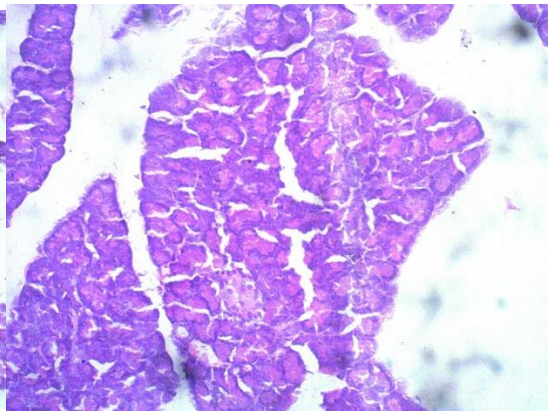
R - 1



R - 2

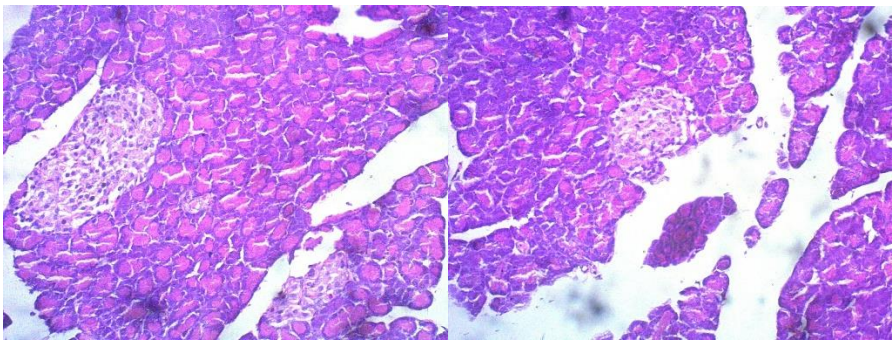


R - 3



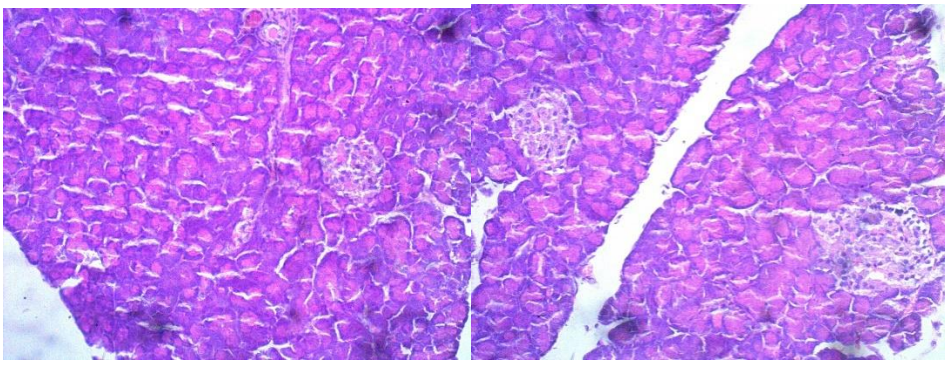
R - 4

Pancreas: Group IV Test -2 group



R - 1

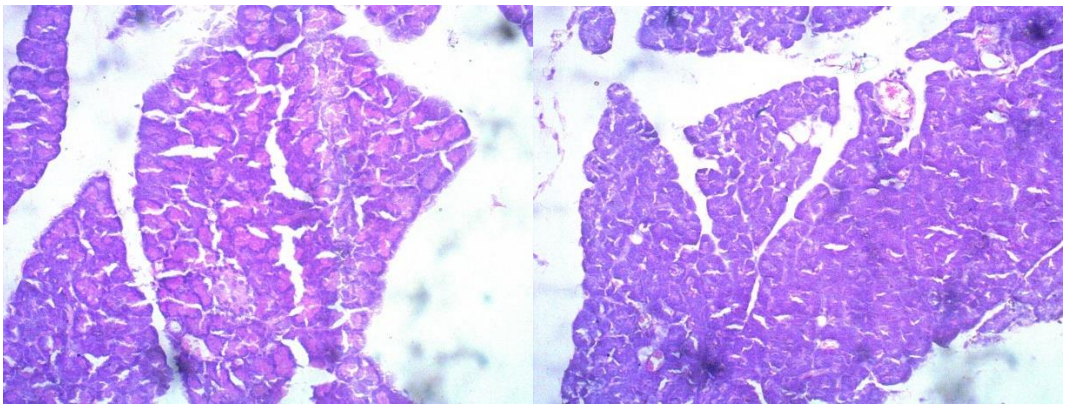
R - 2



R - 3

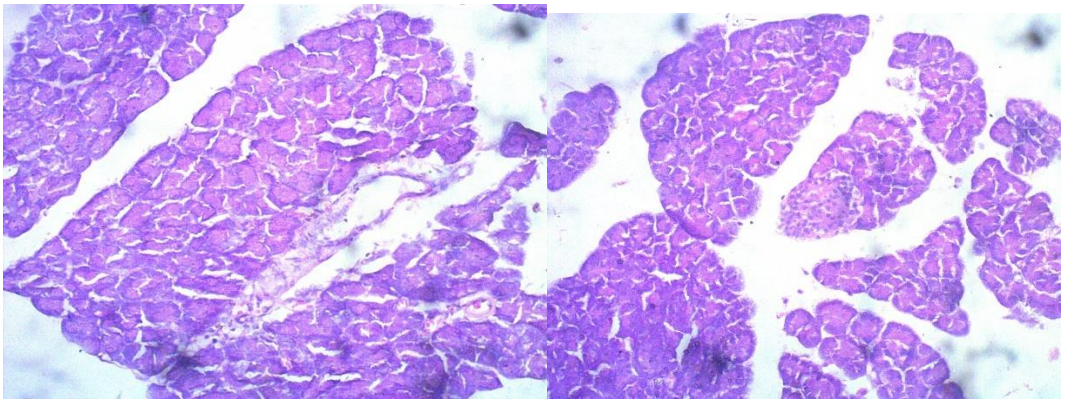
R - 4

Pancreas: Group V Non Diabetic



R - 1

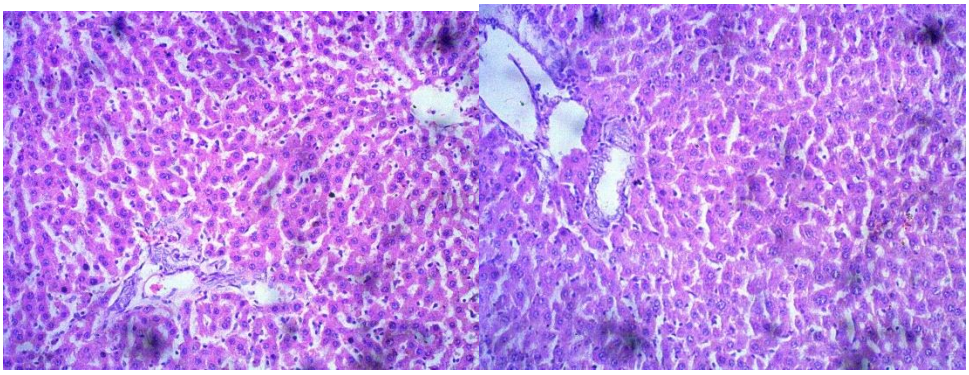
R - 2



R - 3

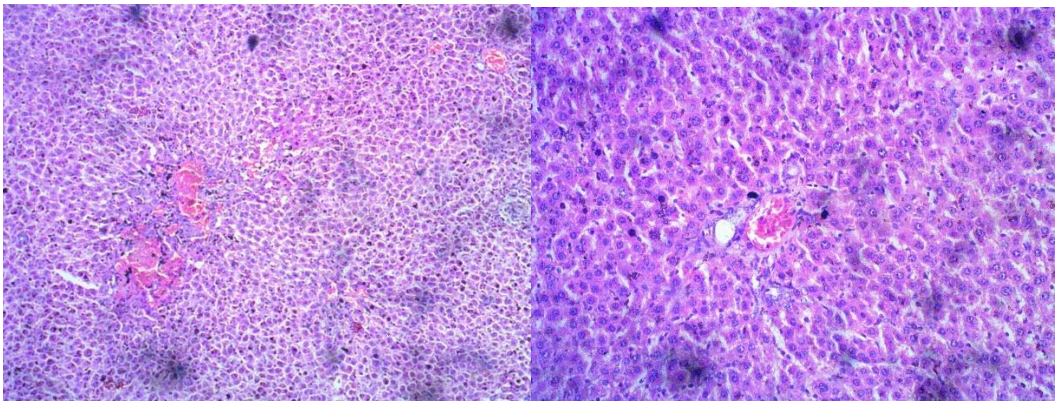
R - 4

Liver: Group I Diabetic Control Group



R - 1

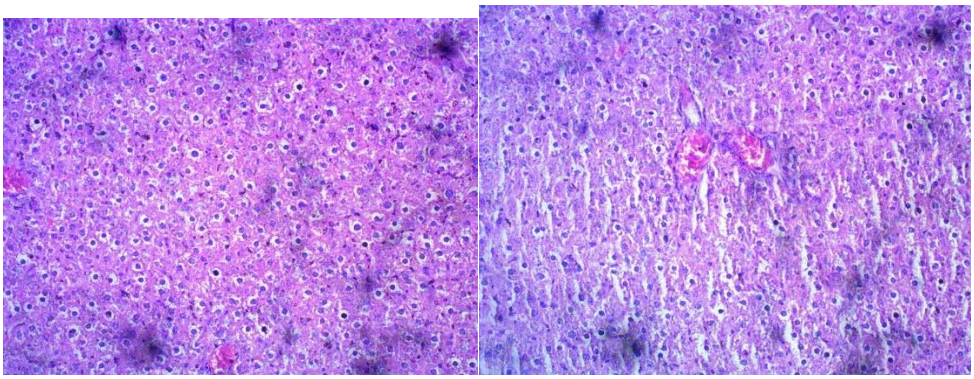
R - 2



R - 3

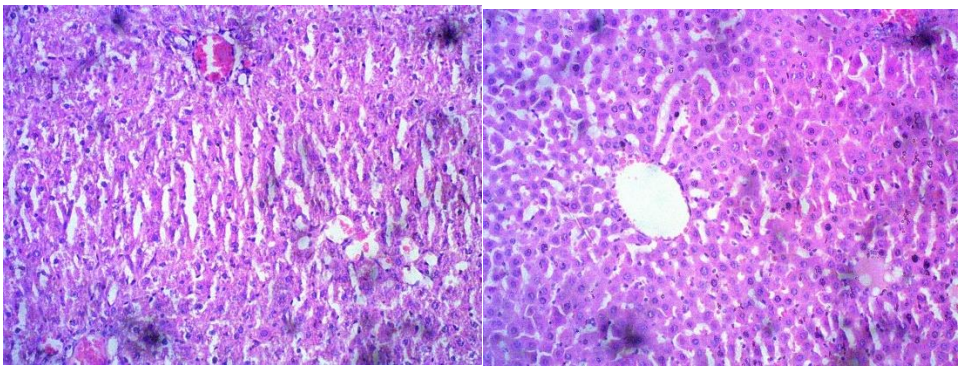
R - 4

Liver: Group II Reference Standard



R - 1

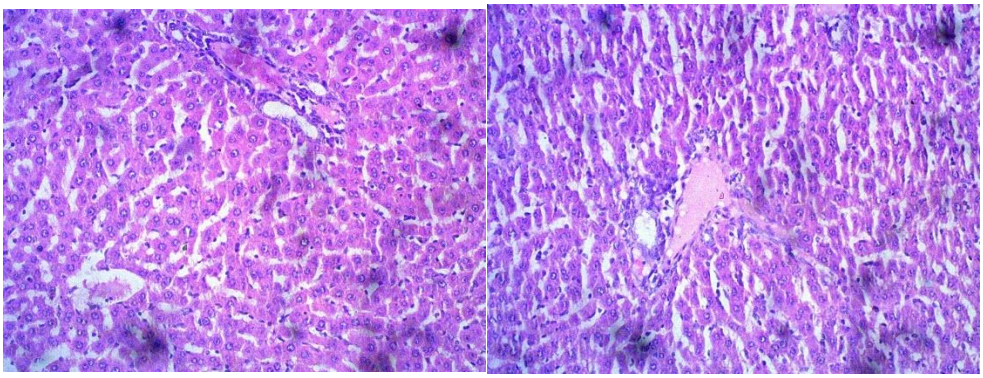
R - 2



R - 3

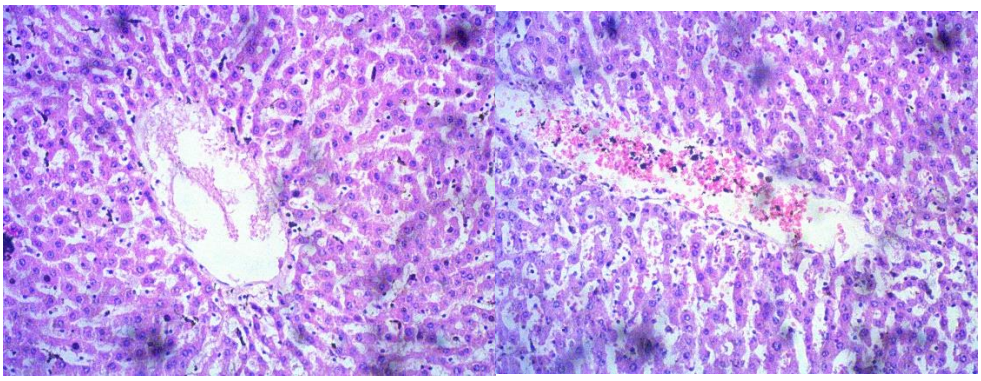
R - 4

Liver : Group III Test -1 group



R - 1

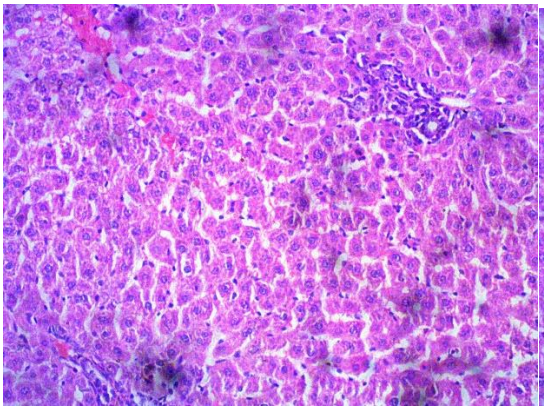
R - 2



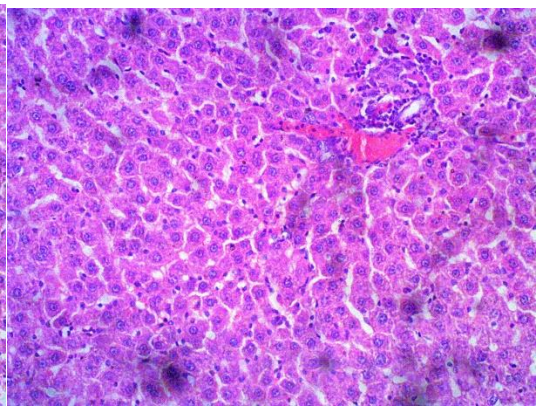
R - 3

R - 4

Liver: Group IV Test -2 group



R - 1



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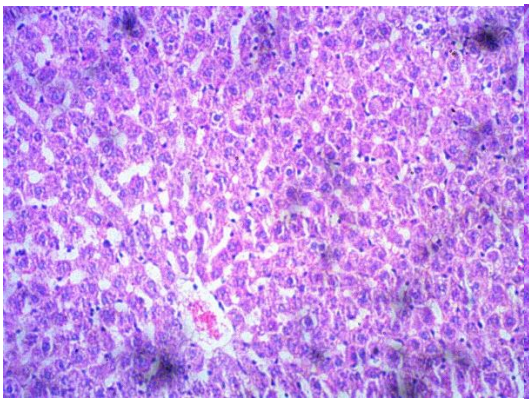
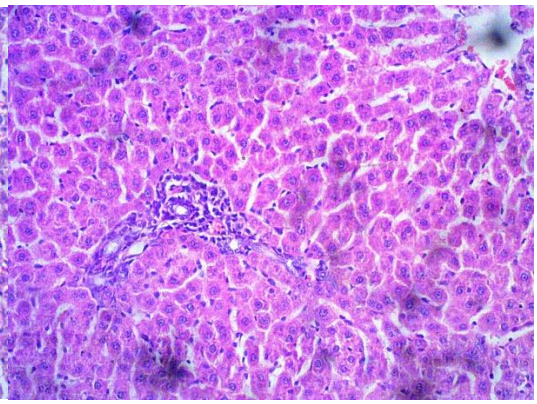
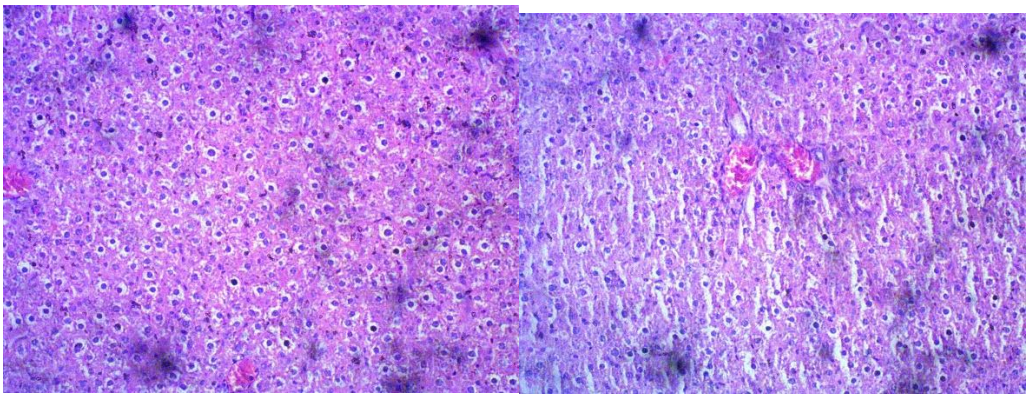


R - 3



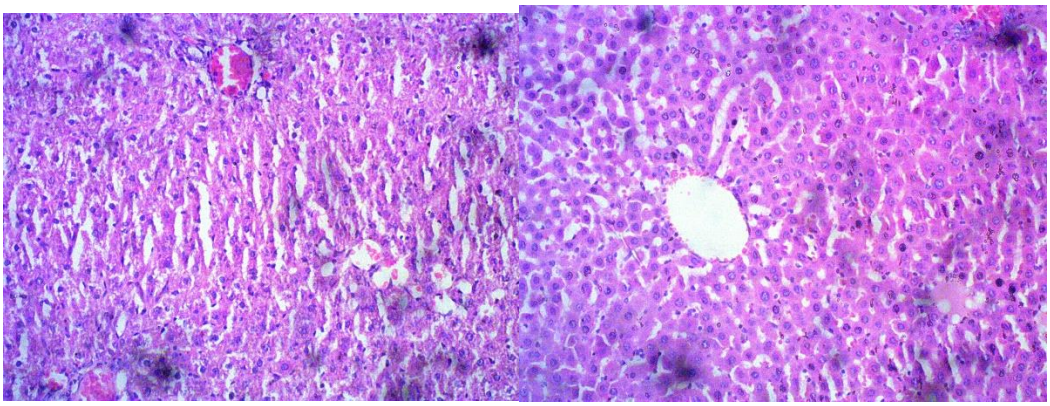
R - 4

Liver: Group VNon-Diabetic



R - 1

R - 2



R - 3

R - 4

Annexure 2 Drug Authentication report

||Om Shri Manjunathaya Namaha||
SRI DHARMASTHALA MANJUNATHESHWARA CENTRE FOR RESEARCH IN AYURVEDAAND ALLIED SCIENCES
(AYUSH Centre for Excellence and Recognized SIROs by DSIR)
Laxminarayana Nagar, P.O. Kuthpady – 574 118
UDUPI [Karnataka]
Ph. 0820 – 2533971 E-mail: sdmcriau@gmail.com

ANALYSIS REPORT FOR 1241/20121201-04

Part A: Particulars of sample submitted

Test requested by: Dr.Raja Rajeshwari N M, SDMCA and Hopital, Hassan
Requested on: 12-12-2020
Investigation to be performed: Macroscopy
Sample coded as: 20121201-04
Sample details: Haritaki, Vibhitaki, Amalaki, Yava

Part B: Methodology

Macroscopy

The external features of the test samples were documented using Canon IXUS digital camera. The macroscopic features were compared to local flora for authentication.

Part C: Results

Figure 1: Macroscopy of Haritaki, Vibhitaki, Amalaki, Yava

Fig 1a. Haritaki

Fig 1b. Vibhitaki

Fig 1c. Amalaki

Fig 1d. Yava

Part D: Remarks

The sample of Haritaki, Vibhitaki, Amalaki, Yava macro-microscopically authentified comparing with Voucher specimen results are depicted in respective figures. Authenification No Haritaki 20121201, Vibhitaki 20121202, Amalaki 20121203, Yava 20121204

Testing Personnel

Suchitra N. Prabhu M.Pharm.
Research Officer –
Pharmaceutical chemistry and
Pharmacognosy

Authorized Signatory

Principal

Annexure 3 IAEC

SDM Centre for Research in Ayurveda & Allied Sciences
SDM college of Ayurveda Campus,
Kuthpady, Lakshminarayana Nagar
Udupi- 574 118

APPROVAL CERTIFICATE

This is to certify that the project title "Study On the Concept of Apatarpana in Santarpanajanya Vikara with Special Reference to Prameha" submitted by

Dr. Rajarajeshwari NM has been approved

(Approval No: SDMCRA /IAEC/ 55 02 by the IAEC in its meeting held on 07/01/2019

(Chairman IAEC)

(CPCSEA Nominee)

* * * * *