

CLINICAL STUDIES OF CERTAIN AYURVEDIC FORMULATIONS IN THE MANAGEMENT OF PARAPLEGIA (PAṄGU)



Central Council for Research in Ayurveda and Siddha

Department of AYUSH, Ministry of Health and Family Welfare

Government of India

**CLINICAL STUDIES OF CERTAIN AYURVEDIC
FORMULATIONS IN THE MANAGEMENT OF
PARAPLEGIA (PAÑGU)**

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PREFACE

Paraplegia is a condition in which the lower part of a person's body is paralyzed and is usually as a result of spinal cord injury. Nerve joining the spinal cord above the level of injury or pathological lesion will be unaffected and continue to work as normal.

The disease paraplegia may be compared with *Panigu* of Ayurvedic literature. The details of the aetiology, clinical features, prognosis and management etc. have vividly been described in Ayurvedic classics. On the basis of classical literature, clinical knowledge, practice and result of preliminary research, four intensive clinical research trials were designed and conducted at two peripheral institutes of the Council.

Both *Sodhana* and *Śamana* principles were followed in four different studies. Conventional *Pañcakarma* therapies as well as selected herbo-mineral formulations were used for the study. In **Study I**, *Ekāṅgavīra Rasa* (internal) and *Mahāmāṣa Taila* (external) in Group I and *Sodhana* therapy with *Mūrchhita TilaTaila* in Group II were given. In **study II**, *Gorocanādi Guṭikā*, *Aśvagandhā Kvātha* and *Balāśvagandhālākṣādi Taila* were given in Group I and in Group II *Sodhana* therapy i.e. *Vireicana* and *Yogabasti* (*Anuvāsana* & *Nirūha*) was administered. In **Study III**, conventional *Pañcakarma* therapy and *Śamana* therapy by *Daśamūla Kvātha* and *Candraprabhā Vatī* internally followed by *Abhyanga* and *Mātrābasti* with *Daśamūlabalā Taila* were tried. In **study IV**, *Candraprabhā Vatī* along with *Daśamūla Kvātha* orally, *DaśamūlaBalā taila* for local application along with *Mātrābasti* and physiotherapy were given in Group I and in Group II, *Virecana* with *Eraṇḍa taila* and *Yogabasti* (*Anuvāsana* & *Nirūha*) were given.

Pharmacological studies of extracts have been conducted on *Sahacara* and *Nirgundi* and established their anti-inflammatory and analgesic action. Biochemical studies have been conducted on comparative analysis of *Bṛhat Māṣa Taila* and physicochemical changes in *Mūrchhita TilaTaila* and *Balāśvagandhālākṣādi Taila* on *Abhyanga*. It is found to have significant difference in specific gravity, acid value and saponification value.

All the studies showed promising and encouraging response. It is also observed that the subjective and objective improvement of disease conditions was significant when analyzed statistically. The studies also showed no adverse drug reaction as internal and external use.

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(Dr. G S. Lavekar)
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CONTENTS

	PAGE NO.
SUMMARY	
Hindi	
English	
1. BACKGROUND	
1.1. Ayurvedic Perspective	
1.1.1 General Consideration about <i>Vātavyādhī</i>	
1.1.2 <i>Nidāna</i>	
1.1.3 <i>Samprapti</i> of <i>Pāṅgu</i>	
1.1.4 Signs and symptoms of <i>Pāṅgu</i>	
1.1.5 Differential diagnosis	
1.1.6 Prognosis	
1.1.7 Principle of Treatment	
1.2 Modern Perspective	
1.2.1 Definition	
1.2.2 Special techniques for neurological diagnosis	
1.2.3 Transverse lesions of spinal cord	
1.2.4 Clinical effects	
1.2.5 Treatment	
2. DRUG PROFILE	
2.1 <i>Ekāṅgavīra Rasa</i>	
2.2 <i>Erandā Taila</i>	
2.3 <i>Mahāmāṣa Taila</i>	
2.4 <i>Mūrchhita TilaTaila</i>	
2.5 <i>Gorocanādi Gutikā</i>	
2.6 <i>Aśvagandhā Kvāṭha</i>	
2.7 <i>Balāśvagandhālākṣādi Taila</i>	
2.8 <i>Daśamūlalabala Taila</i>	
2.9 <i>Daśamūlalabala Kvāṭha</i>	
2.10 <i>Candraprabhā Vatī</i>	

3. PHARMACOLOGICAL STUDIES

- 3.1 Materials and Methods
 - 3.1.1 Acute toxicity
 - 3.1.2 Gross behaviour
 - 3.1.3 Hypnotic potentiation
 - 3.1.4 Effect on muscle tone and Balance (forced locomotor activity, FMA) in mice
 - 3.1.5 Effect on spontaneous motor activity (SMA) in mice
 - 3.1.6 Antipsychotic activity
 - 3.1.7 Anti-depressant activity
 - 3.1.8 Anti-parkinsonism activity
 - 3.1.9 Anti-convulsant activity
 - 3.1.10 Analgesic activity
 - 3.1.11 Anti-inflammatory activity
 - 3.1.12 Immunosuppressant activity
 - 3.1.13 Anti-microbial activity
 - 3.1.14 Studies on isolated tissues

4. BIOCHEMICAL STUDIES

- 4.1 Comparative analytical studies on *Bṛhat Māṣa Taila*
 - 4.1.1 Introduction
 - 4.1.2 Materials and Methods
 - 4.1.3 Results and Discussion
 - 4.1.4 Conclusion
 - 4.1.5 Physico-Chemical Changes in *Mūrchhita Tila Taila* and *Balāśvagandhālāksādi Taila* on *Abhyanga*
 - 4.1.6 Introduction
 - 4.1.7 Materials and Methods
 - 4.1.8 Results
 - 4.1.9 Discussion

5. CLINICAL STUDY

- 3.1 Materials and Methods
- 3.2 Demographic Data
- 3.3 Result

6. DISCUSSION

7. CONCLUSION

APPENDICES

- Bibliography
- References
- Case Record Form

ABBREVIATIONS

च.सू.	चरक्संहिता सूत्रस्थान
च.नि.	चरक्संहिता निदानस्थान
च.वि.	चरक्संहिता विमानस्थान
च.चि.	चरक्संहिता चिकित्सास्थान
सु.सू.	सुश्रुतसंहिता सूत्रस्थान
सु.नि.	सुश्रुतसंहिता निदानस्थान
सु.शा.	सुश्रुतसंहिता शारीरस्थान
सु.चि.	सुश्रुतसंहिता चिकित्सास्थान
अ.ह.सू.	अङ्गांहृदय सूत्रस्थान
अ.ह.नि.	अङ्गांहृदय निदानस्थान
शा.पू.	शांगधारसंहिता पूर्वज्ञान
चक्र.	चक्रपाणिदत्त

सारांश

मुख्य शब्द:- पंगु, अर्धाङ्गधात, एकाङ्गधीर रस, महामान तैल, शोधन, विरेचन, योगबस्ति, गोरोचनादि गुटिका, मात्राबस्ति, अनुवासन, निरुह!

प्रस्तुत कार्य पंगु (अर्धाङ्गधात) रोग पर विभिन्न आयुर्वेदिक औन्ध योगों के चार अध्ययनों के चिकित्सकीय आंकड़ों का संकलन है। प्रत्येक अध्ययन में रोगियों को दो समूहों में विभाजित किया गया है। ये अध्ययन परिनद के दो क्षेत्रीय अनुसंधान संस्थानों में किये गये। ये अध्ययन पंगु रोग की चिकित्सा के लिए अधिक प्रभावी वानस्पतिक खनिज औन्ध योगों को जानने के लक्ष्य से किये गये। परिनद के द्वारा तैयार किये गये विशेष प्रोटोकॉल के अनुसार कुल 225 पंगु रोग से पीड़ित रुग्णों का निदान करके आतुरीय अनुसंधान के लिए चयन किया गया।

प्रथम अध्ययन के एक समूह में एकांगधीर रस (अन्तः प्रयोग) एवं महामान तैल (बाह्य प्रयोग) का प्रयोग किया गया तथा दूसरे समूह में शोधन चिकित्सा, विरेचन एवं योगबस्ति का प्रयोग किया गया। द्वितीय अध्ययन के प्रथम समूह में गोरोचनादि गुटिका, अश्वगन्धा क्वाथ एवं बलाश्वगन्धा लाक्षादि तैल का प्रयोग किया गया तथा दूसरे समूह शोधन चिकित्सा विरेचन एवं योगबस्ति (अनुवासन व निरुह) का प्रयोग किया गया। तृतीय अध्ययन के प्रथम समूह में दशमूल क्वाथ व चन्द्रप्रभावटी का अन्तःप्रयोग अभ्यंग एवं दशमूल तैल की मात्राबस्ति के साथ किया गया तथा दूसरे समूह में विरेचन एवं योगबस्ति का प्रयोग किया गया। चतुर्थ अध्ययन के प्रथम समूह दशमूल क्वाथ के साथ चन्द्रप्रभावटी का आभ्यन्तर प्रयोग, मात्राबस्ति एवं फिजियोथेरेपी के साथ दशमूलबला तैल का स्थानिक प्रयोग किया गया तथा दूसरे समूह में एरण्ड तैल के साथ विरेचन एवं योगबस्ति (अनुवासन व निरुह) का प्रयोग किया गया।

इन चारों अध्ययनों में पाया गया कि अधिकतम 25.44% रोगी 41-50 वर्ष आयु वर्ग के तथा 23.21% रोगी 51-60 आयु वर्ग के थे। अधिकतम 69.20% रोगी पुरुष वर्ग के तथा 30.80% रोगी महिला वर्ग के थे। अधिकतम 29.86% रोगी 180 दिन से कम समय से इस रोग से ग्रसित थे।

प्रथम अध्ययन से ज्ञात होता है कि दोनों समूहों में मांसपेशी शक्ति व चलने की गति में चिकित्सा का प्रभाव सांख्यिकीय दृष्टि से उल्लेखनीय ($P<0.05$) रहा। इसके अतिरिक्त प्रथम समूह की चिकित्सा का प्रभाव मूत्राशय पर नियंत्रण में सार्थक रहा तो द्वितीय समूह की चिकित्सा का शूलशमन में।

द्वितीय अध्ययन में पाया गया कि दोनों समूहों की चिकित्सा का प्रभाव मांसपेशी क्षय एवं आक्षेप (उद्वे-टन) को छोड़कर शेन सभी लक्षणों व चिन्हों में सांख्यिकीय दृष्टि से उल्लेखनीय (सार्थक) ($P<0.05$) रहा।

तृतीय अध्ययन में दोनों चिकित्सा मांसपेशी के क्षय को छोड़कर शेन सभी लक्षणों व चिन्हों में प्रभावी पाई गई। मांसपेशी के आक्षेप (उद्वे-टन) पर शमन चिकित्सा का प्रभाव पाया गया जो कि शोधन चिकित्सा का नहीं मिला।

चतुर्थ अध्ययन में दोनों समूहों की चिकित्सा का प्रभाव मांसपेशी क्षय व मांसपेशी आक्षेप (उद्वे-टन) को छोड़कर सभी लक्षणों व चिन्हों पर सार्थक पाया गया। यद्यपि द्वितीय समूह में चिकित्सा का प्रभाव मूत्राशय व मलाशय पर नियंत्रण में सांख्यिकीय दृष्टि से उल्लेखीय रहा, जो कि प्रथम समूह में नहीं रहा।

परिणामों के विश्लेषण से ज्ञात होता है कि चारों अध्ययनों में प्रयुक्त औन्धियाँ आतुरीय लक्षणों को कम करने में प्रभावी हैं तथा आतुरीय अध्ययन काल में इनका कोई दु-प्रभाव भी नहीं देखा गया।

SUMMARY

Key words: *Paṅgu, Paraplegia, Ekāṅgavīra Rasa, Mahāmāṣa Taila, Śodhana, Virecana, Yogabasti, Gorocanādi Gutikā, Mātrābasti, Anuvāsana, Nirūha*

The present work consisting of the data of clinical studies conducted on *Paṅgu* (Paraplegia) with four studies of different Ayurvedic formulations. Each study comprises of two groups. These studies were conducted at two peripheral research institutes of CCRAS. These studies were carried out with the objectives to find out more effective herbomineral formulations for the management of *Paṅgu*. A total number of 235 cases of *Paṅgu* were diagnosed and recruited for clinical research as per the protocol specially prepared by the Council.

For Study I, *Ekāṅgavīra Rasa* (internal) and *Mahāmāṣa Taila* (external) were used in Group I and *Śodhana* therapy i.e. *Virecana* and *Yogabasti* were used in Group II. For Study II, *Gorocanādi Gutikā*, *Aśvagandhā Kvāṭha* and *Balaśvagandhālākṣādi Taila* were used in Group I. In Group II *Śodhana* therapy i.e. *Virecana* and *Yogabasti* (*Anuvāsana* & *Nirūha*) were administered. For Study III, *Daśamūla Kvāṭha* and *Candraprabhā Vatī* internally followed by *Abhyāṅga* and *Mātrābasti* with *Daśamūlabalā Taila* has been studied in Group I and in Group II *Virecana* and *Yogabasti* were administered. For study IV, *Candraprabhā Vatī* along with *Daśamūla Kvāṭha* orally, *DaśamūlaBalā taila* for local application along with *Mātrābasti* and physiotherapy were given in Group I. In Group II, *Virecana* with *Eranḍa taila* and *Yogabasti* (*Anuvāsana* & *Nirūha*) has been given.

In these four studies, it was observed that more number of patients (25.44%) were belongs to the age groups of 41-50 years followed by 23.21% patients in 51-60 years of age group. As per sex wise distribution, more number of patients (69.20%) was male and 30.80% were female. As per the chronicity of the disease, maximum patients (29.86%) were reported with the duration of less than 180 days.

In study I, it was observed that the effect of the therapies in both the groups was significant ($P<0.05$) in muscle power, pressing power and walking speed. In addition to this, the effect of therapy in Group-I was significant ($P<0.05$) in control over bladder, tone and abdominal reflex where as the therapy was effective in pain in Group-II. On over all assessment, both the therapies were significant to manage the Pa'gu, but there is no major difference in the effect between two therapies.

In Study II, it was observed that the effect of therapies on both the groups was significant ($P<0.05$) in all signs and symptoms except clonus and wasting of muscle.

In Study III, both the therapies were found significant in all the signs and symptoms except wasting of muscle. In case of clonus, the effect of *Samana* therapy was significant which was not significant on *Sodhana* therapy.

In Study IV, it was found that both the therapies were significant in all signs and symptoms except clonus and muscle wasting. However in Group II the effect of therapy was significant in control over bladder and rectum and abdominal reflex which was not significant in group I.

On analysing the results it was found that, the drugs used in these four studies shown better efficacy in bringing down the clinical symptoms without any side effect during the course of clinical trial.

1. BACKGROUND

BACKGROUND

1.1 Ayurvedic Perspective

Ability of locomotion is a natural gift to human beings. *Paingu* (Paraplegia) is a clinical condition where this natural gift loses. Paralysis or weakness of both lower limbs occurs in *Paingu*. It is a *Vāta* predominant disease and limping movement (*vikalagati*) is the literal meaning of *Paingu*, where both the lower limbs are involved (*Dvipāda vikalatvam Painguḥ*)¹

The word *Paingu* is found in *Rgveda* (2:15:17). It enlightens the fact that this disease was persisting since the Vedic period (500 BC to 2500 BC). *Pāṅgulya* is coming under the 80 types of disorders of *Vāta* (*Nānātmaja roga*)² as described by *Caraka* and *Vāgbhāṭa*.

Khañja is also described along with *Paingu*, because it is having the similar aetiology and the same line of treatment.

¹ तत् पर्यायः खोडः इत्यमरः खोल इति शब्दरत्नावलि।
खोरः खञ्जकः इति हेमचन्द्र ।
खोटः इति खोडाद्यात्वर्थं दर्शनात् ।
शब्दकल्पद्रुमः द्वितीयो पृ-ठ 271

खञ्ज-खजि गतिवैकल्ये अच् (खोडा) एक पाद् विकले।
तस्य लक्षणादिकं भावमिश्र उत्कं यथा वायुः.....
खञ्जः द्विपादविकलत्वे पांगुरिति भेदः।
वाचस्पत्यम् तृतीयो भागः पृ-ठः 2455

² तत्रादौ वातविकाराननुव्याख्यास्यामः, तद्यथा..... पांगुल्यं च... खञ्जत्वं च... । च. सू. 20-11
तत्र वाविकाराः तद्यथा.....पांगुत्वं..... खञ्जत्वं..... । अ.सं.सू. 20-13

संकोचः पर्वणां स्तंभो भेदोऽस्थनां पर्वणामपि।
लोमहर्षः प्रलापश्च पाणिपृ-ठशिरोग्रहः ।
खञ्जया पांगुल्यं कुब्जत्वं..... । वराह संहिता 28-21

Khañja and *Paṅgu* both are included in the diseases caused by vitiated *Vāta*³.

Suśruta has given some more advanced information on symptomatology and pathogenesis of *Paṅgu*.⁴ Suśruta has included *Paṅgu* in the *Janmabala Pravṛtta* diseases, which afflicts due to *Apacāra* (indulgence in faulty regimen) by the mother during pregnancy⁵. The blood-letting is not advised generally in *Vātaroga*⁶ as it causes *Dhātukṣaya*, one of the major factors of *Vātaprakopa* and it may lead to severe complications. But Suśruta has prescribed blood letting in *Pāṅgulya*⁷.

Vāgbhaṭa has clearly mentioned this disease and his description on the symptomatology and pathogenesis of the said disease is similar to that of Suśruta⁸.

As described earlier, the word *Paṅgu* is used to express two clinical conditions viz.

(i) to express walking disability, which manifest as a disease with specific

³ वायु कट्यां स्थितः सकथनः कण्डरामाक्षिपेद्यदा ।
खञ्जस्तदा भवेष्जन्तुः, पंगुः सकथनोद्धयोर्वधात् ॥ सु.नि.1-77

⁴ तत्रादिरन्तबलप्रवृत्ता.....पंगु..... ॥ सु.सू.24-6

⁵ न तून-गोडशातीत-सप्तत्यध्य स्नुतासृजाम् ।
अस्निग्धा स्वेदितात्यर्थस्वेदितानिलरोगिणाम् ॥ अ.ह. सू. 27-5

⁶ गृध्रसी विश्वाची क्रो-टुकशिरः खञ्जपंगुलवातकण्टक.....
....यथाद्वेषां च सिराव्यधं कुर्यात्..... ॥ सु. चि.5-23

⁷ वायुः कट्यां स्थितः सकथनः कण्डरामाक्षिपेद्यदा ।
तदा खञ्जो भवेत् जन्तुः पंगु सकथेनोद्धयोरपि ॥ अ.ह.नि. 15-45

⁸ वायुः कट्याश्रितः सकथनः कण्डरामाक्षिपेद्यदा ।
खञ्जस्तदा भवेष्जन्तुः पंगु सकथनोद्धयोर्वधात् ॥ मा. नि. 22-51

वायुः कट्याश्रितः सकथनः कण्डरामाश्रयेद्यदा ।
खञ्जस्तदा भवेष्जन्तुः पंगु सकथनोद्धयोर्वधात् ॥ शा. सं. 7

वायुः कट्याश्रितः सकथनः कण्डरामाश्रयेद्यदा ।
खञ्जस्तदा भवेष्जन्तुः पंगु सकथनोद्धयोर्वधात् ॥ भा. प्र. 24-151

करोति खञ्जं पंगुं वा शरीरे सर्वतश्चरन् । अ.ह.नि. 16-11

aetiopathogenesis and its associated complaints and (ii) it may be a symptom of some other disease (*Vāgbhāta*).

Vātavyādhi is a group of the diseases, which can occur only by the vitiation of *Vāta Dosa*. *Paigu* has not been described as a separate disease of *Vāta*, but it is described as one of the symptoms of vitiated *Vāta*.

1.1.1 General Consideration about *Vātavyādhi*:

The word *Vātavyādhi* has been composed of the words viz. *Vāta* and *Vyādhi*. *Vāta* is considered to be the most powerful and active amongst the three *Dosas*. Although the entire body is the dwelling of three *Dosa*, *Vāta*, *Pitta* and *Kapha*, but the prime importance has been given to the *Vāta* due to its capacity to move in entire body without help of other *Dosa*. To explore the supremacy of *Vāta*, Caraka has mentioned that “*Vāyu* is life and vitality; *Vāyu* is the supporter of all embodied beings; *Vāyu* is verily the whole universe and *Vāyu* is the lord of all⁹. By this reference it is clear that *Vāyu* is the main factor, which is responsible for the healthy and diseased status of the individual. *Pitta* and *Kapha* also have a capacity to disturb the normal state of the health, but they are unable to do so without the support of *Vāta*. Due to the higher efficacy, *Vāta* can produce eighty types of defects and derangements in the body.

The word “*Vyādhi*” is suggestive of circumstances in which body and mind both are in distress. In this way the collective meaning of *Vātavyādhi* indicates the specific disorders occurred due to the *Vāta Dosa*. While commenting on the word “*Vātavyādhi*” *Cakrapāni* has mentioned two definitions of it viz. a)“*Vāta Eva Vyādhi Vātavyādhīh*”

⁹ वायुरायुर्बलं वायुर्वायुर्धाता शरीरिणाम् ।
वायुर्विश्वमिदं सर्वं प्रभुर्वायुश्च कीर्तितः ॥ च. चि. 28/3

Means *Vāta* itself disordered, which combined with particular *Dusya* and attains the form of generalized or localized disease and because of producing pain it is called as *Vātavyādhi*. b) “*Vātato Vyādhi Vātavyādhi*”. It means that *Vāta Dosa* causes the disease by particular pathogenesis in which particular type of *Dosadūṣyasammūrcchanā* leads to the particular disease (*Cakrapāni* on Ca. Ci. 28/1). To distinguish the *Vātavyādhi* from *Sāmanyā Vyādhi*, *Cakrapāni* has given the explanation that though *Jvara* etc. diseases are also can be caused by *Vāta*, they are produced even by other *Dosas* without involving *Vāta* and as such they are not called as *Vātavyādhi*. Where as *Vātavyādhi* cannot be manifested until *Vāta* is not involved and such type of diseases of *Vāta* are known as *Nānātmaja* disorders of *Vāta*. In *Madhikoṣa* commentary of *Mādhava Nidāna*, *Vijayarakṣita* has tried to find out the exact meaning of the word “*Vātavyādhi*”, because he found the lacuna in the definitions given above. According to him ““*Vāta Eva Vyādhi*” indicates that *Vāta* itself is a disease. Hence no one can be considered as healthy because *Vāyu* has been called life and vitality⁹(Ca. Ci. 28/3). The other definition “*Vātato Vyādhi Vātavyādhi*” is also not suitable because according to this definition all the disease in which *Vāta* plays a major role as one of the causative factors (e.g. *Sāmanyaja Vātavyādhi*) may be included under the *Vātavyādhi*. After discussing on *Cakrapāni*,s definitions of *Vātavyādhi*, *Vijayarakṣita* has explained his own thought about the meaning of *Vātavyahdhi*. He explained that *Vātajanito Asādhāraṇavyādhirvātavyādhi*”, means specific diseases caused by vitiated *Vāta* are known as *Vātavyādhi*. This definition seems to be correct because of its specificity and differentiability from other *Sāmanyaja* diseases of *Vāta* (*Vijayarakṣita* on *Ma. Ni., Vātavyādhi*). In this way it is clear that *Vātavyādhi* is a group of specific disorders, which caused only by vitiated *Vāta*.

As per fundamental principles of *Ayurveda*, *Dhātukṣaya* and *Mārgāvaraṇa* are the two basic factors for *Vātaprakopa*¹⁰. But the actual manifestation including the extent of the body affected depends upon the place of *Doṣadūṣyasammūrcchanā*. Since the disease originates at *Kati*, the *Doṣadūṣyasammūrcchanā* can be envisaged at spinal cord and hence a lesion in the spinal cord due to any reason impairs the normal functioning of lower limbs.

1.1.2 *Nidāna*:

Specific *Nidāna* of *Pāṅgu* has not been mentioned, because it is mentioned as one of the disorder of vitiated *Vāyu*. Hence the factors, which are responsible for the vitiation of *Vāyu* can be considered as the *Nidāna* of *Pāṅgu* also. *Vāta Prakopaka Nidāna* described by various authors has been presented in Table, 1.

¹⁰ धातुक्षयकरैर्वायुः कुप्यत्यति निर्वितैः ।
चरन् स्त्रोतःसु रिक्तेनु भृशं तान्येव पूरयन्॥
तेभ्योऽन्यदो-पूर्णभ्यः प्राप्य वाऽवरणं बली ॥ अ.ह.नि15-5

वायोधार्थतुक्षयात् कोपो मार्गस्यावरणेन च ।
वातपित्त कफादेहे सर्वस्त्रोतोऽनुसारिणः ॥
वायुरेव हि सूक्ष्मत्वाद्वायोस्तत्राप्युदीरणः ।
कुपितस्तौ समुद्धूय तत्र तत्र क्षिपन् गदान् ॥
करोत्यावृत मार्गत्वाद् रसादीश्वेषो-नयेत । च. चि. 28/59-61

Table, 1

Āhārataḥ	Ca.	Su.S.	A.S.	B.P.	S.D.	Ma.Ni	Y.R.
<i>Ati Tikta Rasa Sevana</i>	-	+	+	+	-	-	-
<i>Ati Katu Rasa Sevana</i>	-	+	+	+	-	-	-
<i>Ati Kaśāyas Rasa Sevana</i>	-	+	+	+	-	-	-
<i>Rūksānna Sevana</i>	+	+	+	+	+	+	+
<i>Sitānna Sevana</i>	+	+	+	-	+	+	+
<i>Laghvanna Sevana</i>	+	+	+	+	+	+	+
<i>Śuṣka Bhojana</i>	-	+	+	-	-	-	-
<i>Pramita Bhojana</i>	-	-	+	+	-	-	-
<i>Hīna Bhojana</i>	-	-	+	-	-	-	-
<i>Alpa Bhojana</i>	+	-	+	-	-	-	-
<i>Abhojana</i>	+	-	-	-	-	+	+
<i>Trsītāśana</i>	-	-	+	-	-	-	-
<i>Kṣudhitāśana</i>	-	-	+	-	-	-	-
<i>Vistambhi Bhojana</i>	-	-	+	-	-	-	-
<i>Viṣamāśana</i>	-	+	-	-	-	-	-
<i>Adhyaśana</i>	-	+	-	-	-	-	-
<i>Śuṣka Māṃsa</i>	-	+	-	-	-	-	-
<i>Varaka</i>	-	+	-	-	-	-	-
<i>Uddālaka</i>	-	+	-	-	-	-	-
<i>Koradūṣa</i>	-	+	-	-	-	-	-
<i>Śyāmāka</i>	-	+	-	-	-	-	-
<i>Nīvāra</i>	-	+	-	-	-	-	-
<i>Mudga</i>	-	+	-	-	-	-	-
<i>Masura</i>	-	+	-	-	-	-	-
<i>Ādhakī</i>	-	+	-	-	-	-	-
<i>Harenu</i>	-	+	-	-	-	-	-
<i>Kalāya</i>	-	+	+	-	-	-	-
<i>Nispāva</i>	-	+	-	-	-	-	-
<i>Kalinga</i>	-	-	+	-	-	-	-
<i>Virūḍhaka</i>	-	-	+	-	-	-	-
<i>Truṇa Dhanya</i>	-	-	+	-	-	-	-
<i>Karīra</i>	-	-	+	-	-	-	-
<i>Tumbī</i>	-	-	+	-	-	-	-
<i>Bis Śālūka</i>	-	-	+	-	-	-	-
<i>Jāmbava</i>	-	-	+	-	-	-	-
<i>Tinduka</i>	-	-	+	-	-	-	-

Vihārataḥ	Ca.	Su.S.	A.H.	B.P.	S.D.	Ma.Ni	Y.R.
<i>Ati Vyāyāma</i>	+	+	+	+	+	+	+
<i>Ati Prajāgarana</i>	+	+	+	+	+	+	+
<i>Ati Laṅghana</i>	+	+	+	-	-	+	+
<i>Ati Plavana</i>	+	+	+	-	-	+	+
<i>Atyadhva</i>	+	+	-	-	-	+	+
<i>Ati Vyavāya</i>	+	+	+	-	+	+	+
<i>DuhKhaśaiyyā</i>	+	-	-	-	-	-	-
<i>Duhkhaśana</i>	+	-	-	-	-	-	-
<i>Divāsvāpa</i>	+	-	-	-	-	-	-
<i>Vegadhāraṇa</i>	+	+	+	+	-	+	+
<i>Ati Adhyayana</i>	-	+	+	-	-	-	-
<i>Pratarana</i>	-	+	+	+	-	-	-
<i>Balāvadvigraha</i>	-	+	-	-	-	-	-
<i>Prapīḍana</i>	-	+	-	-	-	-	-
<i>Gaja, Turaṅga,Ratha</i>	-	+	+	-	-	+	+
<i>Padāticaryā</i>							
Mānasika	Ca.	Su.S.	A.S.	B.P.	S.D.	Ma.Ni	Y.R.
<i>Cintā</i>	+	-	-	+	+	+	+
<i>Śoka</i>	+	-	+	+	+	+	+
<i>Krodha</i>	+	-	-	-	-	-	-
<i>Bhaya</i>	+	-	-	+	+	-	-
<i>Kāma</i>	-	-	-	-	+	-	-
Anya	Ca.	Su.S.	A.S.	B.P.	S.D.	Ma.Ni	Y.R.
<i>ViśĀma Upcāra</i>	+	-	-	-	-	+	+
<i>Atidosāsrāvana</i>	+	-	+	-	-	+	+
<i>Atyasrgsrāvana</i>	+	-	+	-	-	+	+
<i>Pañcakarma Atiyoga</i>	-	-	+	+	-	-	-
<i>Rogātikraśana</i>	+	-	-	+	-	+	+

<i>Dhātukṣaya</i>	+	-	-	+	-	+	+
<i>Āmadoṣa</i>	+	-	-	-	+	+	+
<i>Abhighāṭa</i>	+	+	+	+	+	+	+
<i>Marmāghāṭa</i>	+	-	-	-	-	+	+
<i>Gaja, Uṣṭra, Aśva, Śīghrayāna-aptansanat</i>	+	+	+	-	-	+	+

1.1.3 Samprāpti of Paṅgu:

The term *Samprāpti* is applied to express the course of the appearance of disease right from *Nidānasevana* to *Vyādhi Utpatti*. The knowledge of *Samprāpti* helps in the comprehension of the specific features of a disease like *Dosā*, *Dūṣya*, *Srotodushti*, *Āma* and *Agni* etc. The study of *Samprāptivighāṭana* is said to be done by treatment. *Charkachārya* has described six types of *Samprāpti* namely *Saṅkhyā*, *Vidhi*, *Vikalpa*, *Prādhānya*, *Balā* and *Kāla*¹¹. *Suśruta* has described *Samprāpti* process in six stages *Saṅcaya*, *Prakopa*, *Prasara*, *Sthānasamśraya*, *Vyakti* and *Bheda*, which are known as *Ṣatkriyākāla*. During *Sthānasamśrayāvasthā* the vitiated *Dosā* are said to have reached to particular *Sthāna* and get obstructed here and intimately mix with and vitiate one, two or more *Dūṣyas* in that particular portion of body. This is the reason that though *Nidāna* of all the *Vātavyādhi* are same but only due to the *Samprāpti Viśeṣa* of disease *Vāta* can produce so many *Vāta* disorders. If vitiated *Vāta* is accumulated in *Nitamba* and *Kukundara Marma* by *Srotosāṅga*, it produces *Paṅgu*. It is enlisted under the 80 varieties of *Nānātmaja Vātavyādhi*; thereafter its *Samprāptivyāpāra* is on the similar lines of *Vātavyādhis*, hence predominance of *Vāta Dosā* in its *Samprāpti* is clear. *Paṅgu* is manifested with *Karmahāni* (loss of function) of both the lower extremities. According to *Āyurveda*, *Mārgāvaraṇa* and

¹¹ संप्राप्तिर्जातिरागतिरित्यनर्थान्तरं व्याधेः ॥ च.नि.1/11

Dhātukṣaya are the two basic factors for manifestation of *Vātaja* disorders, which ultimately can be the cause for *Pāṅgulya*. *Mārgāvaraṇa* may occur due to compression, tumour, viral or bacterial infection or vascular lesion of spinal cord. The *Dhātukṣayaja* *Pāṅgu* can be considered as degenerative and congenital type of *Pāṅgulya* (paraplegia). As per Suśruta, affliction of *Kukundara* and *Nitamba Marma* produce loss of sensation, loss of function, weakness and wasting of muscle¹².

In normal state *Vāta* governs *Utsāha* (enthusiasm), *Swāsa-Nihśvāsa* (respiration), *Ceṣṭā* (all motor activities), *Vegapravartana* (regulation of natural urges), *Dhātūnām Samyakgati* (the regulation of circulation and functioning of seven fold *dhātu*, *Ākṣepatava* (proper functioning of sensory organs). Thus the function ascribed to *Śarīra Vāyu* in the ancient medical classics are exactly those which modern physiology ascribed to the nervous system. It has been observed that all the *Vātavyādhis* are manifested with some varieties of nervous disorder. On the basis of symptomatology given in classics, the probable *Samprāpti Ghaṭaka* of *Pāṅgu* can be traced out as below:

Samprāpti Ghaṭaka:

<i>Dosa</i>	:	<i>Vāta, especially Apāna</i>
<i>Dūṣya</i>	:	<i>Rakta, Kandarā, Snāyu, Māmsa</i>
<i>Srotas</i>	:	<i>Raktavaha, Māmsavaha,</i>
<i>Srotoduṣti Prakāra</i>	:	<i>Sanga</i>
<i>Agni</i>	:	<i>Jātharāgni and Dhātvāgni māndya</i>
<i>Āma</i>	:	<i>Jātharāgni māndyajanya and Dhātvāgni māndyajanya</i>
<i>Udbhava Sthāna</i>	:	<i>Pakvāśaya</i>
<i>Sañcāra Sthāna</i>	:	<i>Adhah Kāya (Lower extremities)</i>
<i>Adhiṣṭhāna</i>	:	<i>Kandarā and Snāyu</i>

¹² कुकुन्दरे, तत्र स्पर्शज्ञानमधःकाये चे-टोपधातश्च,...
नितम्बौ, तत्राधःकाय शो-गो, दोर्बल्याच्च मरणं.... || सु.शा.6/26

1) *Dosā* :

Vāta is the essential *Dosā* for the manifestation of disease *Paṅgu*. It is known that the *Prakopa* of *Vāta* may occur in two ways i.e. due to *Dhātuksaya* and *Āvaraṇa*¹³. In case of *Dhātuksaya*, continuous ingestion of food materials which are *Rūkṣa*, *Laghu*, *Śīta*, *Śuṣka* in nature *Rātrijāgarāṇa*, *Vegavidhāraṇa*, *Pramitāsana* and all such causes lead to *Dhātuksaya* and subsequently manifestation of *Paṅgu*. *Āvaraṇa* is another way for the vitiation of *Vāta*. *Āvaraṇa* is a distinctive pathological condition of *Vāta* leading to its *prakopa* resulting into various disorders of *Vāta*. The *Āvaraṇa* of *Vāta* can be caused by the increased and vitiated *Dosā*, *dhātu*, *mala*, *anna* and *Āma*, which are treated as *Āvaraka*. The *Vāta* whose *āvaraṇa* occurs is termed as *āvariya of āvrita*.

2) *Dūṣya*

Acharya Madhavakara says that, in *Paṅgu* the vitiated Doshas affects the *Kandarā* (tendon) present in the *Katī pradeśa* and thus manifestation of the disease occurs. According to Caraka, *Kandarā* is the *Upadhātu* of *Rakta dhātu*¹⁴. Chakrapani has mentioned that *Sthūla Snāyu* may be taken as *Kandarā* (tendon)¹⁵. Therefore in the pathogenesis of *Paṅgu*, *Rakta*, *Māṃsa*, *Kandarā* and *Snāyu* are considered as *Dūṣya*.

3) *Srotas*

As mentioned above, here *Rakta* and *Māṃsa dhātu* are vitiated and their respective *Srotas* also are involved in this disease process. *Māṃsa śōṣa* (emaciation of muscles) are

¹³ वायोर्धातुक्षयात् कोपो मार्गस्यावरणेन च (वा)।
वातपित्तकफा देहे सर्वस्त्रोतोऽनुसारिणः ॥ च.चि.28/59

¹⁴ रसात् स्तन्यं ततो रक्तमसृजः कण्डराः सिराः ।
मांसाद्वसा त्वचः -नट्च मेदसः स्नायुसंभवः ॥ च.चि.15/17

¹⁵ कण्डरा इहतन्त्रे स्थूलस्नायुः । च.सू. 11/48 (चक्रपाणि)

due to the involvement of *Māṃsavaha Srotoduṣṭi*. *Māṃsa śoṣa* may not be present in the first stage, which may develop later if proper management is not given in time, or mainly due to the disuse of the legs. *Samjñāvaha Srotoduṣṭi* can be envisaged from the loss or impaired sensation on the legs. *Asthivaha Srotoduṣṭi* can be evidenced by the vertebral lesions in certain *Pāṅgu* cases. *Asthi* and *Snāyuvaḥa Srotoduṣṭi* is a clear evidence of *Abhighātaja* (traumatic) *Pāṅgu* cases.

4) *Srotoduṣṭi Prakara*:

Saṅga type of *Srotoduṣṭi* is found in *Pāṅgu*. As *vāta* is the main dosa in *Pāṅgu* and *gati* (movement) is the main function of *Vāta*, *saṅga* type of *Srotoduṣṭi* causes obstruction of the movement of *Vāta*, which produces symptoms viz. loss of function of the lower extremities.

5) *Agni-Āma*

Agni always play primary role in the vitiation of *Dosā*. Derangement of *Agni* affects the *Dosā* prior to the *Dosadūṣya sammūrcchanā*, which is the initial stage of manifestation of disease. *Jāṭharāgnī* and *Dhātvāgnī* of *Rakta*, *Māṃsa* and *Asthi Dhātu* are vitiated, which further lead to *Kandarākshepa* and *Māṃsasaithilya* manifesting *Pāṅgu*. Vitiated *Agni* produces respective *Āma*. In this disease *Jāṭharāgnimandyā janya* and *Dhātvāgnimandyā janya* *Āma* of *Rakta* and *Asthi Dhātu* is produced.

6) *Udbhava Sthāna*:

UdbhavaSthāna of this disease is *Pakvāśaya* as it is a *Nānātmaja Vātavyādhī*.

7) *Saṅcāra Sthāna*

Here, *Saṅcāra Sthāna* of the vitiated *Dosā* is the *Kandarā* (tendon) present in the *Katī pradeśa* (waste region).

8) *Adhisṭhāna*:

Both the lower limbs are the adhishthana (site of manifestation) of *Pāṅgu*.

1.1.4 Signs and symptoms of Paigu

Walking disability is the main feature of Paigu. The muscles may be rigid or flaccid, which depends on the type/side/nature and severity of affliction and chronicity of the disease. Suśruta has mentioned that *Paigu Śakthnordwayorvadhāt Vadha Kriyānāsa*.

Changes in deep tendon reflexes, electric chorea and *Māmsaśoṣa* (emaciation of muscles) are due to the involvement of *Māmsavaha Srotoduṣṭi*. *Māmsaśoṣa* may not be present in the first stage, which may develop later if proper management is not given in time, or mainly due to the disuse of the legs. *Samjñāvaha Srotoduṣṭi* can be envisaged from the loss or impaired sensation on the legs. *Asthivaha Srotoduṣṭi* can be evidenced by the vertebral lesions in certain *Paigu* cases. *Asthi* and *Snāyuvaha Srotoduṣṭi* is a clear evidence of *Abhighātaja* (traumatic) *Paigu* cases.

Suśruta as *Kandarākshepa* explains rigidity and flaccidity (*Māmsasithilata*) is explained by *Paigu Sakthnordvayorvadhāt Vadha Kriyanāśa*, *Mutravaha Srotoduṣṭi* and *Puriśavaha Srotoduṣṭi* (impairment of micturition and defecation) is the result of an *Apānavaiguna*.

1.1.5 Differential diagnosis

<i>Grdhrasī</i>	<i>Paksavadha</i>	<i>Sarvāṅgavāta</i>	<i>Kalāyakhañja</i>
Clinical features			
<ul style="list-style-type: none"> ➤ It starts from sacrum & subsequently goes to <i>Kati</i>, <i>Prsthā</i>, <i>Uru</i>, <i>Jānu</i> and <i>Jaighā</i> (Calf muscles) with the features of Stiffness, 	<ul style="list-style-type: none"> ➤ Loss of function of Half part of the body. ➤ <i>Sirā Snāyuśoṣa</i> ➤ The affected part becomes stiff, contracted and weak. ➤ Burning sensation, and 	<ul style="list-style-type: none"> ➤ Pricking and tearing type of pain. ➤ Tremor. ➤ Stiffness. ➤ Convulsions ➤ Contractions. ➤ Shivering. 	Sri.Taranath Chakravarti describes a disease <i>Kalāyakhañja</i> , a variety of <i>Paigu</i> (<i>Tasya vaibheda Kalāyakhañja</i> ,

Pain, Pricking sensation and <i>spandana</i> .	loss of function of that part.		Tārānātha Tarka Vācaspati).
<p>It is of two types :</p> <p>1. <i>Vātaja</i> - Stiffness, radiating pain starting from the buttock to the toes. Pricking sensation and <i>spandana</i>.</p> <p>2. <i>Vātakaphaja</i> - Stiffness, Pain, Pricking sensation, <i>spandana</i>, <i>Tandrā</i>, <i>Gaurava</i> and loss of Appetite (<i>Aruci</i>).</p>			<ul style="list-style-type: none"> ➤ Typical kind of tremors to the affected leg just before beginnisng to walk is the symptom of <i>Kalāyakhañja</i>., ➤ Limping gait. ➤ Sub-luxation of joints of lower limb.
Management			
1. Hot fomentation (<i>Sevadana</i>). 2.Venapuncture (<i>Sirāvedha</i>)& Cautary (<i>Agnikarma</i>) at the point in between <i>Kandarā</i> and <i>Gulpha</i> . 3. <i>Rūksa</i> <i>Bālukāsveda</i> (dry fomentation).	1. <i>Abhyanga</i> with <i>Balā Taila</i> 2 <i>Pinda sveda</i> . 3. <i>Virecana</i> (Purgative) drugs.	1. Use of <i>Salbi</i> , <i>Vasā</i> , <i>taila</i> , <i>majjā</i> . 2. <i>Abhyanga</i> <i>Snehabasti</i> . 3. Fomentation Patient should be kept in the space where there will be no air. 4. <i>Pathya</i> : - Use of <i>madhura</i> , <i>Amla</i> and <i>Lavana Rasa</i> and <i>Br̥mhanīya Āhāra</i> .	1. <i>Snehana</i> . 2. <i>Snigdha Svedana</i> 3. <i>Abhyanga</i> 5. <i>Basti</i> 6. <i>Śirosneha</i> 7. <i>Dhūmapāna</i> , 8. <i>Nasya</i> . 8. <i>Pathya</i> :- Meat soup (<i>Māmsarasa</i>), milk (Dugdha), oil (<i>Taila</i>), <i>Amla,Lavana</i> , food.

1.1.6 Prognosis

Paigutva and *Angamāmsaśosa* (emaciation) of limbs are described as *Krcchrasādhyā* (difficult to cure). Even after all efforts, complete relief may not be possible. When emaciation starts there will be minimum improvement. Improvement will be better in strong persons and in fresh and non-complicated cases.

1.1.7 Principle of Treatment

As *Pangū* is a *Vātaroga*, the treatment prescribed for *Vātaroga* is recommended in general. As per the treatment of *Vāta Vyādhī*, if the disease is caused by *vāta* exclusively, and if no occlusion is involved, then in the beginning, the patient should be treated by oleation therapy for which ghee (*Ghṛta*), muscle fat (*Vasā*), oil (*Taila*) and bone-marrow (*Majjā*) should be administered. Thereafter, when the patient gets disgusted with the intake of oleation therapy, he should be consoled (rested for some time), and again oleation therapy should be administered with the help of milk, vegetable soup and soup of the meat of domesticated, aquatic and marshy-land-inhabiting animals after adding fat. He may be given *pāyasa* (preparation of rice and milk) and *Kṛśarā* (a preparation of rice, legumes, etc.) added with sour ingredients as well as salt. He may also be given *Anuvāsana* type of medicated enema, inhalation therapy and refreshing food.

Oleation therapy, when administered, instantaneously provides nourishment to the emaciated tissue elements. It promotes strength, *Agni* (enzymes responsible for digestion and metabolism), reduces plumpness of the body and *vyāna Vāta*. The patient should be given oleation and fomentation therapies repeatedly as a result of which the *Kostha* (viscera,s in the abdomen and thorax) becomes soft and the diseases of *Vāyu* do not get an opportunity to get lodged there permanently.

After the patient is properly oleated, he should be given fomentation therapy. Before the administration of fomentation therapy, the body of the patient should be properly oleated and thereafter, fomentation therapies, viz., *Nādi-sveda*, *prastara-sveda*,

Saṅkara-sveda as well as other types of appropriate fomentation therapies should be administered. [Vide Sutra 14: 39-69 for details of these fomentation therapies.]¹⁶

If because of inappropriate administration of [the above mentioned] therapies (oleation and fomentation) the ailments [caused by *Vāyu*] do not subside, then the patient should be given elimination therapy with the help of mild drugs added with unctuous ingredients.

On account of the intake of food which is unctuous, sour, saline, hot, etc., the morbid material gets accumulated and it obstructs the channels of circulation leading to the occlusion of the [movement of] *Vāyu*. Therefore, the patient should be given elimination (purgation) therapy.

If the patient is weak, and is therefore, unsuitable for the administration of purgation therapy, then he should be given *Nirūha* type of medicated enema prepared with ingredients which are *pācana* (carminative) and *Dīpana* (stimulant of digestion). He should also be given food added with ingredients which are *pācana* (carminative) and *Dīpana* (digestive stimulants). (Ca.Ci. 28/83-87)

¹⁶ केवलं निरूपस्तम्भमादौ स्नेहैरुपाचरेत् ।
वायुं सर्पिवसातैलमञ्जपानैर्नरं ततः ॥
स्नेहकृत्तं समाश्वास्य पयोभिः स्नेहयेत् पुनः।
यू-ग्रीष्माम्बुजानूपरसैवा स्नेहसंयुतै ॥
पायसैः कृशरैः साम्ललवणैरनुवासनैः ।
स्वभ्यक्तं स्नेहसंयुक्तैर्नाडी प्रस्तर संकरैः ॥
तथाऽन्यैर्विधैः स्वेदैर्यथायोगमुपायरेत् ।
स्नेहाक्तं खिन्नमंगं तु वक्रं स्तब्धमथापि वा ॥
शनैर्नामयितुं शक्यं यथे-टं शु-कदारुवत् । च.चि.28/78-79

1.2 Modern Perspective

1.2.1 Introduction

Paraplegia means paralysis of both lower extremities. This may occur in diseases of spinal cord, spinal roots peripheral nerves or may be hysterical. If the onset is acute, it may be difficult to distinguish spinal from neuropathic paralysis because the element of spinal shock may result in abolition of reflexes and flaccidity.

The most common cause of acute paraplegia is spinal cord trauma usually combined with fracture or dislocation of spine. Spontaneous hematomyelia due to vascular malformation, thrombosis of the anterior spinal artery or occlusion of a spinal branch of the aorta are less common causes. Paraplegia due to post infectious or post vaccinal myelitis, acute demyelinative or necrotising myelopathy and epidural abscess or tumour with spinal cord compression tend to develop somewhat more slowly, over a period of hours or days or longer. Paralytic poliomyelitis and acute idiopathic polyneuritis, the former a purely motor disorder with mild meningitis, the latter predominantly motor but often with minimal sensory disturbances are other conditions. Congenital cerebral diseases account for a majority of cases of infantile diplegic (weakness), or on legs with minimal affection of the arms. Though present at birth, it becomes manifest in the first month of life and may appear to progress, but actually the disease is stationary and the slow improvement as a result of normal maturation processes of childhood. Friedreichs ataxia, familial paraplegia, progressive muscular dystrophy and the chronic varieties of polyneuropathy tend to appear later during childhood and adolescence and are slowly progressive.

In adult life multiple sclerosis, sub acute combined degeneration (vit.B12 deficiency) tumour, protruded cervical disc and cervical spondylosis, syphilitic meningomyelitis, epidural abscess and other infections (tuberculous, fungal and other granulomatous diseases) motor system diseases, syringomyelia and degenerative diseases of lateral and posterior column of unknown cause represent the most frequently encountered forms of spinal paraplegia. Several varieties of polyneuropathy and polymyositis must also be considered in the differential diagnosis of paraplegia. In hysterical paralysis one arm or leg or both legs or all or one side of the body may be affected.

1.2.2 Special techniques for neurological diagnosis

The following techniques are adopted for the diagnosis of paraplegia:

Lumbar puncture: This is performed for the following reasons (i) to obtain pressure measurements and to secure a sample of cerebrospinal fluid for cellular, chemical and bacteriological examinations, (ii) to aid in therapy by the administration of spinal anaesthetics and occasionally antibiotics and anti-tumour agents and (iii) to inject air as in pneumo-moencephalography or contrast media as in myelography.

Once the lumbar puncture is done following aspects of CSF should be studied, pressure dynamics, gross appearance, number and types of cells, presence of microorganisms, protein and sugar, exfoliate cytology, Wasserman reaction, immunoelectrophoresis and bacteriologic cultures. The implications of each disease are discussed along with.

Radiological examination of skull and spine (X-Ray of skull or spinal column): According to the nature of symptoms constitute an indispensable part of the thorough study of traumatic, spondylitic and neoplastic disease but are of little value in others.

CT Scan: This permits visualization of ventricles, subarachnoid space the major cisternal fissures and sulci in several horizontal planes. One can see haemorrhage, softened and edematous brain, abscess and tumour tissue and also the precise size and position of ventricles. It also provides excellent images of spinal canal and intervertebral foramen in three planes.

Myelography: By injecting 5-25 ml. of opaque dye (contrast medium) through a lumbar puncture needle and then keeping the patient on tilted table the entire spinal subarachnoid space may be seen. Ruptured lumbar and cervical discs, cervical spondylitic and bony spurs encroaching on the spinal cord tumours can be diagnosed accurately.

Electromyogram (EMG): EMG is useful in studying the muscle unit and its potential and nerve conduction studies. The conduction studies are useful in assessing status of surviving muscle fibres in paraplegia. This also can be used to study fibrillation and fasciculation potentials.

1.2.3 Treatment

In General treatment of spinal injuries is conservative and symptomatic. In all cases of suspected spinal injury the immediate concern is that there is no movement of the spine from the moment of the accident. Heavy sandbags or similar objects should be placed on each side of the head and neck and a small hard roll under the nape of neck. The

greatest risk to the patient with spinal cord injury is in the first 10 days when gastric dilatation, ileus shock and infection are the main problem.

The early management of spinal cord injury varies from centre to centre. Some advocate reduction of dislocated vertebra by traction and immobilization until skeletal mobility is obtained and then rehabilitation. Some advocate early surgical decompression, correction of bony displacement, removal of herniated disc tissue, intra medullary and extra medullary haemorrhage. Often the spine is fixed at the same time by a bone graft or wiring. Some centres delay operation or operate only when the spinal canal is narrowed by more than one third of its diameter (as shown by MRI CT or myelogram). The results of the conservative and aggressive surgical plans of management have been difficult to compare. The results of various methods of treatment concludes that the survival rate has increased as a result of early stabilization of fracture, prevention of respiratory, urinary and cutaneous complications and the early institution of rehabilitation measures but there is no evidence that the degree of neuralgic disability had been reduced. The after care of patients with paraplegia is concerned with management of bladder and bowel disturbances, care of the skin, maintenance of nutrition. Decubitus ulcers can be prevented by special skincare. Physiotherapy, muscle re-education and proper use of braces are important for rehabilitation.

Treatment of the demyelinative and necrotising myelitis consists of ACTH or prednisone. Improvement usually occurs but the relation to therapy is uncertain. Except for patients with post infectious form of myelitis there is always danger of later progression or relapse. Prevention of decubitus ulceration, early catheterization and bladder care and rehabilitation measures that are the usual procedure in the management of the paraplegic patients must also be employed.

The treatment of syringomyelia is difficult. The disease process may remain stationary for some months or years before progressing, makes evaluation of any mode of therapy difficult. Decompression of distended cord up to foramen magnum prevents progression of the symptoms and signs resulting from local compression of ascending and descending spinal tracts but relief is not always lasting.

2. DRUG PROFILE

DRUG PROFILE

As per Ayurvedic texts *Paingu* is considered as a *Nānātmaja Vātavyādhi* and the treatment prescribed for *Paingu* is similar to the recommended therapy of *Vātavyādhi*. The treatment of *Vātavyādhi* as mentioned in the texts are *Snehapāna*, *Abhyāga*, *Svedana*, different types of *Basti*, *Vātaśāmaka yogas*, *Āmapācaka* and *Koṣṭha śuddhikara Oṣadhis*. The patient should take the food which is unctuous, sour, saline and hot, added with ingredients which are *Pācana* (carminative) and *Dīpana* (digestive stimulants) in action. According to modern medicine, it can be correlated with Paraplegia where paralysis of both lower extremities is seen and treatment is mostly conservative and symptomatic. A clinical research work has been undertaken in four different studies to find out an effective therapy for the management of *Paingu* (Paraplegia) and to study the aetiopathology of *Paingu* (Paraplegia).

Study-I: Comprises of two groups:

Group-I:

Ekāṅgavīra Rasa (125 mg) along with *Eraṇḍa Taila* (5 to 10 ml) thrice daily and *Mahāmāṣa Taila* (50 ml. external use) for 60 days

Group-II:

Snehapāna, *Virecana* with *Eraṇḍa taila*, *Anuvāsana*, *Nirūha* and *Abhyāga* with *Mūrchhita Tila Taila* for 60 days

Study-II: Comprises of two groups:

Group-I: *Gorocanādi Gutikā* - 1 *gutikā* (250 mg) thrice daily for 60 days
Aśvagandhā Kvātha - 60 ml. thrice daily for 60 days
Balāshvagandhalākṣādi Taila -50 ml. for *Abhyāga* for 60 days

Group-II: *Pañcakarma* (*Virecana* and *Basti*) therapy with *Mūrchhita Taila*

Study-III: Comprises of two groups:

Group-I: *Pañcakarma* therapy (*Virecana* with *Eraṇḍa taila* and *Yoga*

Basti with *Daśamūlabalā taila*)

Group-II:

- (a) *Daśamūlabalā Kvātha* (60 ml.) two times in a day +
Candraprabhā Vati, one tablet (250mg) two times in a day for 45 days.
- (b) *Abhyanga* with *Daśamūlabalā taila* and *Mātrābasti* with *Daśamūlabalā taila* (50ml) (Alternative days).

Study-IV: Comprises of two groups:

Group-I: *Pañcakarma* therapy (*Virecana* with *Erandī taila* and *Yoga Basti* with *Daśamūlabalā taila*)

Group-II:

- (a) *Daśamūlabalā Kvātha* (60 ml.) two times in a day +
Candraprabhā Vati, one tablet (250mg) two times in a day for 45 days.
- (b) *Abhyanga* with *Daśamūlabalā taila* and *Mātrābasti* with *Daśamūlabalā taila* (50ml) (Alternative days) + Physiotherapy for 45 days.

The properties of all the drugs used in the present study are discussed below:

2.1 *EKAṄGA VĪRA RASA* (A. F. I., Part-I, Page 259)

(Brhat Rasarājasundara: Page 458)¹⁷

Sl. No	Name of the ingredient	English name/ Botanical name	Quantity
1	<i>Gandha (Gandhaka) Śuddha</i>	Purified Sulpher	1 part
2	<i>Mṛta Sūta (Pārada)</i> <i>Rasasindūra</i>	Processed Mercury	1 part
3	<i>Kānta lauha (Lauha)- Bhasma</i>	Calcined Magnatite/Hematite	1 part
4	<i>Vāṅga Bhasma</i>	Calcined Tin	1 part
5	<i>Nāga Bhasma</i>	Calcined Lead	1 part
6	<i>Tāmra Bhasma</i>	Calcined Copper	1 part
7	<i>Abhra (Abhraka) Bhasma</i>	Calcined Biotite/ Mica	1 part
8	<i>Tīkṣṇa (Lauha) Bhasma</i>	Calcined magnatite/hematite	1 part
9	<i>Nāgara (Śunṭhī)</i>	<i>Zingiber officinale</i> Rose. (Rz.)	1 part
10	<i>Marīca</i>	<i>Piper nigrum</i> Linn. (Fr.)	1 part
11	<i>Kanā (Pippalī)</i>	<i>Piper longum</i> Linn. (Fr.)	1 part
12	<i>Varā drava (Triphalā) kvātha</i> <i>Āmalakī</i> <i>Harītakī</i> <i>Bibhītaka</i>	Decoction made from <i>Emblica officinalis</i> Gaertn. (P.) <i>Terminalia chebula</i> Retz. (P.) <i>Terminalia bellerica</i> Roxb. (P.)	Q.S.*

¹⁷ शुद्धं गंधं मृतं सूतं कान्तं वडगं सनागकम् ।
ताम्रं चाभ्रं मृतं तीक्ष्णं नागरं मरिचं कणा ॥
सर्वमेकत्र सञ्चूर्य भावयेच्च पृथक्त्रयम् ।
वराव्योषकनिरुणिडीवह्निमार्कवजैर्द्रवैः ॥
शिग्युकुष्ठद्रवेणापि मनोधात्रा द्रवेण च ।
विषमुष्ट्यर्कहातैश्च आर्द्रकस्य रसैस्तथा ॥
रसश्चैकाङ्गवीरोऽसौ सुसिद्धो रसराट् भवेत् ।
पक्षाद्यातं चार्दितं च धनुर्वातं तथैव च ॥
अद्वांगं गृध्रसीं चापि विश्वाचीमपवाहुकम् ।
सर्ववातामयान्हन्ति सत्यं सत्यं न संशयः ॥(बृहत् रसराजसुन्दर पृष्ठ ४५८)

13	<i>Vyoṣa drava (Trikatu)-kvāṭha</i> <i>Śunṭhi</i> <i>Pippali</i> <i>Marīca</i>	Decoction made from <i>Zingiber officinale</i> Rose. (Rz.) <i>Piper longum</i> Linn. (Fr.) <i>Piper nigrum</i> Linn. (Fr.)	Q.S.*
14	<i>Nirgundi kvāṭha</i>	Decoction made from <i>Vitex negundo</i> Linn. (Lf.)	Q.S.*
15	<i>Vahni drava (Citraka) kvāṭha</i>	Decoction made from <i>Plumbago zeylanica</i> Linn. (Rt.)	Q.S.*
16	<i>Mārkavaja drava</i> (<i>Bhrṅgarāja</i>) svarasa	Juice of <i>Eclipta alba</i> Hassk. (Pl.)	Q.S.*
17	<i>Śigru Svarasa</i>	Juice of <i>Moringa pterygosperma</i> Gaertn. (Lf.)	Q.S.*
18	<i>Kuṣṭha kvāṭha</i>	Decoction made from <i>Saussurea lappa</i> C.B.Clarke (Rt.)	Q.S.*
19	<i>Dhātri drava (Āmalaki)</i> svarasa	Juice of <i>Emblica officinalis</i> Gaertn. (Fr.)	Q.S.*
20	<i>Viṣamūsti (Śuddha) kvāṭha</i>	Decoction made from purified <i>Strychnos nux-vomica</i> Linn. (Enm.)	Q.S.*
21	<i>Arka kvāṭha</i>	Decoction made from <i>Calotropis procera</i> (Ait) R. Br. (Lf./ Rt.)	Q.S.*
22	<i>Hata (Dhattūra) rasa</i>	Juice of <i>Datura metel</i> Linn. (Lf.)	Q.S.*
23	<i>Ārdraka rasa</i>	Juice of <i>Zingiber officinale</i> Rose.(Rz.)	Q.S.*

Q.S.*: Quantity sufficient for *bhāvanā* for 3 days

Dose : 125-375 mg

Anupāna : *Ārdraka svarasa*

Important therapeutic use: *Pakṣavadha; Arditā; Dhanurvāṭa; Vāṭaroga; Grdhrasī;*
Viśvācī

2.2 ERANDA TAILA: (*Bhāvaprakāśa - Guḍūcyādi Varga*)

Properties:

Rasa - *Madhura*

Guna - *Guru, Snigdha*

Vīrya - *Uṣṇa*

Important therapeutic uses – Constipation, Piles and other anorectal disorders, abdominal disorders, Pelvic disorders, Eye disorders and Headache

Dose : 1- 2 *tola* (12-24 ml)

2.3 MAHĀMĀṢA TAILAM (without *Māṃsa rasa*):

(Bhaisajyaratnāvalī Vātaroga cikitsā)¹⁸

Ingredients:

S. No.	Name of the ingredient	Botanical Name	Quantity
1	<i>Māṣa Kvāṭha</i>	<i>Phaseolus mungo</i> Linn. (Sd.)	768g.
		Water 3.2 Lt. reduced to 1/4 th Decoction	
2	<i>Balā Kvāṭha</i>	<i>Sida cordifolia</i> Linn. (Rt.)	768g.
		Water 3.2 Lt. reduced to 1/4 th Decoction	
3	<i>Rāsnā Kvāṭha</i>	<i>Alpinia galanga</i> Hance (Rz.)	768g.
		Water 3.2 Lt. reduced to 1/4 th Decoction	
4	<i>Daśamūla Kvāṭha</i>	Decoction made from group of ten medicinal	768g.

¹⁸ माषक्वाथे बलाक्वाथे रासनाया दशमूलजे ।
यवकोलकुलथानां छागमांसभवे पृथक् ॥२४१॥
प्रस्थे तैलस्य च प्रस्थं क्षीरं दत्वा चतुर्गुणम् ।
रासनात्मगुप्तसिन्धुथशताहैरण्डमुस्तकैः ॥२४२॥
जीवनीयबलाव्योषैः पचेदक्षसमैर्भिषक् ।
हस्तकम्पे शिरःकम्पे बाहुशोषेऽवबाहुके ॥२४३॥
बाधिर्ये कर्णशूले च कर्णनाद च दारुणे ।
विश्वाच्यामर्दिते कुञ्जे गृध्रस्यामपतानके ॥२४४॥
बस्त्यभ्यञ्जनमानेषु नावने च प्रयोजयेत् ।
माषतैलमिदं श्रेष्ठमूर्ध्वजत्रुगदापहम् ।
क्वाथप्रस्थाः षडेवात्र विभक्त्यन्तेन दर्शिताः ॥२४५॥

		plants	
		Water 3.2 Lt. reduced to 1/4 th Decoction	
5	<i>Yava Kvātha</i>	<i>Hordeum vulgare</i> Linn. (Fr.)	256g.
		Water 3.2 Lt. reduced to 1/4 th Decoction	
6	<i>Kola (Badari)</i> <i>Kvātha</i>	<i>Zizyphus mauritiana</i> Lamk. (Sd.)	256g.
		Water 3.2 Lt. reduced to 1/4 th Decoction	
7	<i>Kulattha Kvātha</i>	<i>Dolichos biflorus</i> Linn. (Sd.)	256g.
		Water 3.2 Lt. reduced to 1/4 th Decoction	
8	<i>Rāsnā</i>	<i>Alpinia galanga</i> Hance (Rt.)	12g.
9	<i>Ātmaguptā</i>	<i>Mucuna prurita</i> Hook (Sd.)	12g
10	<i>Saindhava</i>	Rock salt	12g
11	<i>Śatāhvā</i>	<i>Anethum sowa</i> Kurz. (Sd.)	12g
12	<i>Eranda</i>	<i>Ricinus communis</i> Linn. (Rt.)	12g
13	<i>Mustaka</i>	<i>Cyperus rotundus</i> Linn. (Rz.)	12g
14	<i>Jīvaka</i>	<i>Microstylis muscifera</i> Ridley (Rz.)	12g
15	<i>Rśabhaka</i>	<i>Microstylis wallichii</i> Lind. (Rz.)	12g
16	<i>Medā</i>	<i>Polygonatum verticillatum</i> All. (Rz.)	12g
17	<i>Mahāmedā</i>	<i>Polygonatum cirrifolium</i> Royle (Rz.)	12g
18	<i>Kākoli</i>	<i>Lilium polyphyllum</i> D.Don (Sub. Tr.)	12g
19	<i>Kṣīrakākoli</i>	<i>Pritillaria roylei</i> Hook (Sub. Tr)	12g
20	<i>Rddhi</i>	<i>Habenaria intermedia</i> D.Don (Sub. Tr)	12g
21	<i>Vrddhi</i>	<i>Habenaria intermedia</i> D.Don (Sub. Tr)	12g
22	<i>Madhuyastī</i>	<i>Glycyrrhiza glabra</i> Linn. (Rt.)	12g
23	<i>Jivanti</i>	<i>Leptadenia reticulata</i> W. & A. (Rt.)	12g
24	<i>Mudgaparnī</i>	<i>Phaselous trilobus</i> Ait. (Pl.)	12g
25	<i>Māśaparnī</i>	<i>Phaselous labialis</i> spreng. (Pl.)	12g
26	<i>Balā</i>	<i>Sida cordifolia</i> Linn. (Rt.)	12g
27	<i>Śunthī</i>	<i>Zingiber officinalis</i> Rose. (Rz.)	12g
28	<i>Marica</i>	<i>Piper nigrum</i> Linn. (Fr.)	12g
29	<i>Pippalī</i>	<i>Piper longum</i> Linn. (Fr.)	12g

Method of Preparation:

Step 1:

1. Take all the ingredients of Pharmacopoeial quantity.
2. Individually powder the materials of Sl. No. 1to 7 and pass through sieve no. 44.
3. Soak powdered materials in 4 times of water for 4 hours, heat and reduce the volume to 1/4th and make decoctions individually.
4. Filter through muslin cloth to obtain decoction individually.

Step 2:

1. Powder the materials of Sl. No. 8 to 29 to fine powder and pass through sieve no. 85
2. Add sufficient water to make into fine paste.

Step 3:

1. Transfer the decoctions (of step no. 1) and paste (of step no. 2) into a SS vessel and then add Tila taila.
2. Stir thoroughly and heat for 3 hours with constant stirring maintaining the temperature between 500C-800C during the first hour of heating.
3. After all the water content gets evaporated and the paste can be rolled with fingers (varti formation), stop the heating and confirm the following
 - a. Absence of moisture by exposing the varti to flame and
 - b. Absence of crackling sound indicating the absence of moisture.
4. Filter with muslin cloth while hot (at 800C) and pack in tightly closed containers.

Important therapeutic use: Hemiplegia, facial paralysis, deafness, torticollitis, earache, headache & cataract, lathyrism, paraplegia, sciatica, and all types of vata disorders.

Dosage: 6g.

It is also used externally for *vasti*, *abhyanga* and *nasya*.

Anupāna: Warm water, milk.

2.4 TAILA MŪRCCHANĀ (Bhaīṣajyaratnāvalī, Jvarādhikāra, AFI, page 351)¹⁹

S.No.	Name of the ingredient	Botanical Name	Quantity
1	<i>Mañjisthā</i>	<i>Rubia cordifolia</i> Linn. (Rt.)	1 part
2	<i>Harītakī</i>	<i>Terminalia chebula</i> Retz. (P.)	1 part
3	<i>Āmalakī</i>	<i>Emblica officinalis</i> Gaertn. (P.)	1 part
4	<i>Bibhītaka</i>	<i>Terminalia belerica</i> Roxb. (P.)	1 part
5	<i>Hrībera</i>	<i>Coleus vetti</i>	1 part
6	<i>Haridrā</i>	<i>Curcuma longa</i> Linn. (Rz.)	1 part
7	<i>Mustā</i>	<i>Cyperus rotundus</i> Linn. (Rz.)	1 part
8	<i>Lodhra</i>	<i>Symplocos racemosa</i> Roxb. (St. Bk.)	1 part
9	<i>Ketakīpuspa</i>	<i>Pandanus odoratissimus</i> Linn. (Fl.)	1 part
10	<i>Nyagrodha</i>	<i>Ficus benghalensis</i> Linn. (Sec. Rt.)	1 part
11	<i>Nalikā</i>	<i>Cinnamomum tamala</i> Nees. Eberm (St. Bk.)	1 part
12	<i>Tila taila</i>	<i>Sesamum Indicum</i> Linn. (Oil)	44 parts
13	Water		176 parts

Method of preparation: Take the ingredients (*Kalka dravyas*) numbered 1 to 11 in the composition, dry, powder and pass through sieve number 85. Transfer the powdered ingredients to wet grinder and grind with sufficient quantity of water to prepare *kalka* (homogeneous blend). Add increments of *Kalka* to water and stir thoroughly, then add *Tila taila*. Start heating constantly and check the *kalka* for formation of *varti* (*madhyāma pāka lakṣaṇa*) and observe the boiling mixture for appearance of froth. Stop heating when the *kalka* forms into *varti* and the froth emerges. Filter while hot through a muslin cloth and allow for cooling.

¹⁹ तैलं कृत्वा कटाहे दृढतरविमले मन्दमन्दानलैस्तत्।
तैलं निष्फेनभावं गतमिह च यदा शैत्यभावं समेत्य ।
मञ्जिष्ठारात्रिलौधैर्जलधरनलिकैः सामलैः साक्षपथ्यैः
सूचीपुष्णड्घ्ननीरैरुपहितकथितैर्गन्धयोगं जहाति ॥
तैलस्येन्दुकलांशिकैकविकसा भागोऽपि मूच्छाविधौ
ये चान्ये त्रिफलापयोदरजनीहीबेरलोध्रान्विताः ।
सूचीपुष्पवटावरोहनलिकास्तस्याश्च पादांशिका
दुर्गन्धं विनिहन्ति तैलमरुणं सौरभ्यमाकुर्वते ॥
(भैषज्यरत्नावली, ज्वराधिकार, १२८६ - १२८७)

2.5 GOROCANĀDI GUTIKĀ

(*Yoga Ratnāvalī*; published by The Indian Medical Practitioners Cooperative Pharmacy & Stores Ltd. Thiruvanmiyur, Madras -2000; *Sarvaroga Chikitsāratna: Sannipāta Chikitsā prakarana*, Page-302)

S.No.	Name of the ingredient	English name /Botanical Name	Quantity
1.	<i>Gorocana</i>	Bezoar	1 Part
2.	<i>Svarna bhasma</i>	Calx of gold	1 Part
3.	<i>Śrīnga bhasma</i>	Calx of deer antlers	1 Part
4.	<i>Pravāla bhasma</i>	Calx of corals	1 Part
5.	<i>Taṅkana bhasma</i>	Calx of borax	1 Part
6.	<i>Gandhamārjāravīrya</i>	Semen of Civet cat	1 Part
7.	<i>Amber</i>	Ambergris	1 Part
8.	<i>Añjana</i>	Galena	1 Part
9.	<i>Karpūra</i>	<i>Cinnamomum camphora</i> (Linn.) T.Nees & Eberm. (Ext.)	1 Part
10.	<i>Gairika</i>	Red ochre	1 Part
11.	<i>Rudrākṣa</i>	<i>Elaeocarpus ganitrus</i> Roxb. (seed)	1 Part
12.	<i>Candana</i>	<i>Santalum album</i> Linn. (Hard wood)	1 Part
13.	<i>Vacā</i>	<i>Acorus calamus</i> Linn. (Rz)	1 Part
14.	<i>Uśīra</i>	<i>Vetiveria zizanioides</i> (Linn.) Nesh. (Rt))	1 Part
15.	<i>Mustā</i>	<i>Cyperus rotundus</i> Linn. (Rz.)	1 Part
16.	<i>Trikatu</i>	(Fr)	1 Part
17.	<i>Aklarī</i>	<i>Lodoicea maldivica</i> Pers (Kernel)	1 Part
18.	<i>Atasī bīja</i>	<i>Linum usitatissimum</i> Linn. (Sd)	1 Part
19.	<i>Pūti gandha</i>	<i>Holoptelea integrifolia</i> Planch. (Rt)	1 Part
20.	<i>Ajaśrīnga</i>	Goat's horn	1 Part
21.	<i>Kṛṣṇa mṛgaśrīnga</i>	Stag's horn (black)	1 Part
22.	<i>Mṛgaśrīnga</i>	Stag's horn (white)	1 Part
23.	<i>Hasti danta</i>	Ivory	1 Part
24.	<i>Bhūnimba</i>	<i>Swertia chirata</i> Buch. Ham. (Wh.Pl.)	1 Part

25.	<i>Kṛṣṇa jīraka</i>	<i>Carum carvi</i> Linn. (Sd)	1 Part
26.	<i>Drona puspī</i>	<i>Leucas cephalotes</i> Sprang. (Wh.Pl.)	1 Part
27.	<i>Agnimantha</i>	<i>Premna integrifolia</i> Linn. (Rt)	1 Part
28.	<i>Śatapuṣpā</i>	<i>Anethum sowa</i> kurz. (Sd)	1 Part
29.	<i>Rakta kārpāsa bīja</i>	<i>Gossypium herbaceum</i> Linn.(Sd)	1 Part
30.	<i>Apāmārga</i>	<i>Achyranthes aspra</i> Linn.(Rt)	1 Part
31.	<i>Laśuna</i>	<i>Allium sativum</i> Linn. (Rz)	1 Part
32.	<i>Tripalā</i>	Three myrobalans (Fr.)	1 Part
33.	<i>Mālatī mūla</i>	<i>Jasminum officinale</i> Linn. var. <i>grandiflorum</i> Bailey (Rt)	1 Part
34.	<i>Pāthā</i>	<i>cissampelos pareira</i> Linn. (Rt)	1 Part
35.	<i>Aparājītā mūla</i>	<i>Clitoria ternatea</i> Linn. (Rt)	1 Part
36.	<i>Nīlī mūla</i>	<i>Indigofera tinctoria</i> Linn. (Rt)	1 Part
37.	<i>Jātīphala</i>	<i>Myristica fragrans</i> Houtt. (Fr)	1 Part
38.	<i>Mayakku</i>	<i>Quercus infectoria</i> Oliv.(gall)	1 Part
39.	<i>Musta</i>	<i>Larger Nut grass</i>	1 Part
40.	<i>Kandamṛga śrīṅga</i>	Rhinoceros horn	1 Part
41.	<i>Ārdraka</i>	<i>Zingiber officinalis</i> Rose. (Rz.)	1 Part
42.	<i>Jīraka</i>	<i>Cuminum cyminum</i> Linn. (Sd)	1 Part
43.	<i>Bhūnāga</i>	Earthworm	1 Part
44.	<i>Śunṭhī</i>	<i>Zingiber officinalis</i> Rose. (Rz.)	1 Part

Method of Preparation:

Powder the drugs separately. Grind 1-9 with *Ērdraka svarasa* (ginger juice) into a semisolid mass. Then add one by one the other drugs and grind with the addition of *Ērdraka svarasa* (ginger juice). When suitable consistency is reached, make 50 mg pills.

Action and uses:

It is Stimulant, hypo-tensive, anti-toxaemic and expectorant. Used in toxaemic states, allied state of pneumonia, coughs, bronchitis and whooping cough.

Dose:

1 to 2 pills daily twice or thrice with honey or breast milk. Some people may use betel leaf juice, ginger juice or *Daśamūlakatutrayādi* decoction as adjuvant.

2.6 *AŚVAGANDHĀ KVĀTHA:*

S.No.	Name of the ingredient	English name /Botanical Name	Quantity
1.	<i>Aśvagandhā</i>	<i>Withania somnifera</i> (Linn.) Dunal (Rt)	1 Part
2.	Water		16 Part

Take Roots of *Aśvagandhā* and wash with portable water, cut and crush in to small pieces.

Take these pieces in a vessel and add 16 parts of water. Boil to reduced to 1/4th. Thereafter, filter and store in another vessel.

2.7 *BALĀŚVAGANDHĀLĀKṢĀDI TAILA (AFI 8:36)*

*(Sahasrayoga, Tailaprakarana: 13)*²⁰

Sl. No.	Name of the ingredient	English name /Botanical Name	Quantity
1.	<i>Balā</i>	<i>Sida cordifolia</i> Linn. (Rt.)	256g.
		Water 4.096 Lt. reduced to 1/4 th Decoction	
2.	<i>Aśvagandhā</i>	<i>Withenia somnifera</i> (Linn) Dunal (Rt.)	256g.
		Water 4.096 Lt. reduced to 1/4 th Decoction	
3.	<i>Lākṣā</i>	<i>Laccifera lacca</i> (Resin.)	256g.
		Water 4.096 Lt. reduced to 1/4 th Decoction	

²⁰ बलाश्वगन्धालाक्षाणां कषाये पादावशेषिते ।
तैलप्रस्थं पचेद्वीमान्दधिमस्तुचतुर्गुणे ॥
रास्नाचन्दनमञ्जिष्ठादूर्वामधुकचोरकैः ।
सारिवोशीरजलदकुष्ठागरुसुरद्रुमैः ॥
हरिद्राकुमुदाकैन्तीशताह्नापद्मकेसरैः ।
कल्कैरेभिः सुसंयुक्तैः कार्षिकैश्च सुपेषितैः ॥
शनैर्मृद्गिनना पक्त्वा गालयेद् भाजने शुभे ।
प्रशस्ते तिथिनक्षत्रे तैलमेतत्तु शीलयेत् ॥
तेन सर्वज्वरोन्मादक्षयकासादयो गदाः ।
नश्यन्ति विविधाश्चैव वातरोगा न संशयः ॥
अश्विभ्यां निर्मितं तैलमेतत्पुष्टिकरं परम् ॥(सहस्रयोग, तैलप्रकरण; १३)

4.	<i>Taila</i>	<i>Sesamum indicum</i> Linn. (Oil)	768 g
5.	<i>Dadhi mastu</i>	(made from go dadhi)	3.072 kg.
6.	<i>Rāsnā</i>	<i>Alpinia galanga</i> Hance (Rt.) (Rt./Lf.)	12g
7.	<i>Candana (rakta candana)</i>	<i>Ptero carpus santalinus</i> Linn. f. (Ht. Wd.)	12g
8.	<i>Mañjiṣṭhā</i>	<i>Rubia cordifolia</i> Linn. (Rt.)	12g
9.	<i>Dūrvā</i>	<i>Cynodon dactylon</i> Linn. Pers. (Pl.)	12g
10.	Madhuka (yaṣṭī)	<i>Glycyrrhiza glabra</i> Linn. (Rt.)	12g
11.	<i>Coraka</i>	<i>Angelica glauca</i> Edgw. (Rz.)	12g
12.	Sāriva (śveta sārivā)	<i>Hemidesmus indicus</i> R.Br. (Rt.)	12g
13.	<i>Uśīra</i>	<i>Vetiveria zizanioides</i> (Linn.) Nesh. (Rt)	12 g
14.	<i>Jalada (mustā)</i>	<i>Cyperus rotundus</i> Linn. (Rz.)	12 g
15.	<i>Kuṣṭha</i>	<i>Saussurea lappa</i> C.B.Clarke (Rt.)	12 g
16.	<i>Agaru</i>	<i>Aquilaria agallocha</i> Roxb. (Ht. Wd.)	12 g
17.	<i>Suradruma (devadāru)</i>	<i>Cedrus deodara</i> (Roxb.) Loud. (Ht. Wd.)	12 g
18.	<i>Haridrā</i>	<i>Curcuma longa</i> Linn. (Rz.)	12 g
19.	<i>Kumuda</i>	<i>Nymphaea alba</i> Linn. (Rz.)	12 g
20.	<i>Kauntī (renuka)</i>	<i>Vitex agnus-castus</i> Linn. (Sd.)	12 g
21.	Śatāhvā	<i>Anethum sowa</i> kurz. (Sd)	12 g
22.	<i>Padma keśara (kamala)</i>	<i>Nelumbo nucifera</i> Linn. (Adr.)	12 g

Method of Preparation:

1. Take all the ingredients of pharmacopeial quantity. Wash and dry the materials except Sl. no. 4.
2. Powder *Balā*, *Aśvagandhā* and *Lākṣā* and pass through by mesh #44 individually.
3. Add 16 times of water to powders of *Balā*, *Aśvagandhā* and *Lākṣā* individually and then boil to reduce to 1/4th part and filter individually with muslin cloth.
4. Powder the materials of Sl. no. 6-22, add water and made into *kalka*.

5. Add all the decoctions, *Kalka*, *Mastu* and *Taila*; stir thoroughly and heat for 3 hours with constant stirring maintaining the temperature between 500C-800C during the first hour of heating.
6. After all the water content gets evaporated and the paste can be rolled with fingers (varti formation), stop the heating and confirm the following
 - a. Absence of moisture by exposing the varti to flame and
 - b. Absence of crackling sound indicating the absence of moisture.
7. Filter with muslin cloth while hot (at 80⁰C) and pack in tightly closed containers.

Important therapeutic uses: *Jvara*, *Unmāda*, *Kṣaya*, *Kāsa*, *Vāta roga*, *Kṛśatā*.

2.8 DAŚAMŪLABALĀ TAILA:

S. No.	Name of the ingredient	English name /Botanical Name	Quantity
1.	<i>Bilva</i>	<i>Aegle marmelos</i> Corr. (Rt.)	90.9gm (for <i>kvātha</i>) 15.18 (for <i>kalka</i>)
2.	<i>Kāśmarya</i> (<i>Gambhāri</i>)	<i>Gmelina arborea</i> Roxb. (Rt.)	90.9gm (for <i>kvātha</i>) 15.18 (for <i>kalka</i>)
3.	<i>Tarkāri</i> (<i>Agnimantha</i>)	<i>Clerodendrum phlomidis</i> Linn . (Rt.)	90.9gm (for <i>kvātha</i>) 15.18 (for <i>kalka</i>)
4.	<i>Pātalā</i>	<i>Stereospermum suaveolens</i> D. C. (Rt.)	90.9gm (for <i>kvātha</i>) 15.18 (for <i>kalka</i>)
5.	<i>Dunduka</i> (<i>Śyonāka</i>)	<i>Oroxylum indicum</i> Vent. (Rt.)	90.9gm (for <i>kvātha</i>) 15.18 (for <i>kalka</i>)
6.	<i>Prśniparnī</i>	<i>Uraria picta</i> Linn .(Rt.)	90.9gm (for <i>kvātha</i>) 15.18 (for <i>kalka</i>)
7.	<i>Śālaparnī</i>	<i>Desmodium gangeticum</i> D. C. (Rt.)	90.9gm (for <i>kvātha</i>) 15.18 (for <i>kalka</i>)

8.	<i>Bṛhatī</i>	<i>Solanum indicum</i> Linn. (Rt.)	90.9gm (for <i>kvātha</i>) 15.18 (for <i>kalka</i>)
9.	<i>Kaṇṭakārī</i>	<i>Solanum xanthocarpum</i> Burm. (Rt.)	90.9gm (for <i>kvātha</i>) 15.18 (for <i>kalka</i>)
10.	<i>Gokṣura</i>	<i>Tribulus terrestris</i> Linn. (Rt.)	90.9gm (for <i>kvātha</i>) 15.18 (for <i>kalka</i>)
11.	<i>Balā</i>	<i>Sida cordifolia</i> Linn. (Rt.)	90.9gm (for <i>kvātha</i>) 15.18 (for <i>kalka</i>)
12.	<i>Tila taila</i>	<i>Sesame oil</i>	1.0 Lt.

Method of Preparation:

Stage 1

1. Take the all the ingredients of pharmacopoeial quality for *kvātha*.
2. Then cut and crush in to small pieces, take in a vessel and add 16 litters of water. Boil to reduce to 4 litters then filter and store in another vessel.

Stage 2

1. Take the all the ingredient of pharmacopoeial quality for *kalka*, powder them and add water to make in to paste.

Stage 3

1. Take decoction of stage 1, *kalka* of stage 2 mix well and add *Tila taila*.
2. Stir thoroughly and heat for 3 hours with constant stirring maintaining the temperature between 500C-800C during the first hour of heating.
3. After all the water content gets evaporated and the paste can be rolled with fingers (varti formation), stop the heating and confirm the following
 - a. Absence of moisture by exposing the varti to flame and
 - b. Absence of crackling sound indicating the absence of moisture.
4. Filter with muslin cloth while hot (at 800C) and pack in tightly closed containers.

2.9 DAŚAMŪLABALĀ KVĀTHA:

S.No.	Name of the ingredient	English name /Botanical Name	Quantity
1.	Bilva	<i>Aegle marmelos</i> Corr. (Rt.)	1 part
2.	Kāśmarya (<i>Gambhārī</i>)	<i>Gmelina arborea</i> Roxb. (Rt.)	1 part
3.	Tarkārī (<i>Agnimantha</i>)	<i>Clerodendrum phlomidis</i> Linn. (Rt.)	1 part
4.	Pātalā	<i>Stereospermum suaveolens</i> D. C. (Rt.)	1 part
5.	Duṇḍuka (<i>Śyonāka</i>)	<i>Oroxylum indicum</i> Vent. (Rt.)	1 part
6.	Prśniparṇī	<i>Uraria picta</i> Linn. (Rt.)	1 part
7.	Śālaparṇī	<i>Desmodium gangeticum</i> D.C. (Rt.)	1 part
8.	Bṛhatī	<i>Solanum indicum</i> Linn. (Rt.)	1 part
9.	Kantakārī	<i>Solanum xanthocarpum</i> Burm. (Rt.)	1 part
10.	Goksura	<i>Tribulus terrestris</i> Linn. (Rt.)	1 part
11.	Balā	<i>Sida cordifolia</i> Linn. (Rt.)	1 part

Method of Preparation:

1. Take all the materials of equal and pharmacopeial quality, wash in portable water, dry and then powder them.
2. Add 16 times of water to the powders of above.
3. Boil to reduce to 1/4th Decoction.
4. Thereafter filter and store in another vessel.

2.10 *CANDRAPRABHĀ VATĪ* (A.F.I. 12: 10)

*(Śāringadharasamhitā, Madhyamakhaṇḍa, Adhyāya 7: 40-49)*²¹

S.No.	Name of the ingredient	English name /Botanical Name	Quantity
1.	<i>Candraprabhā (karpūra)</i>	<i>Cinnamomum camphora</i> (Linn.) T.Nees & Eberm. (Ext.) (Sub.Ext.)	3 g
2.	<i>Vacā</i>	<i>Acorus calamus</i> Linn. (Rz)	3 g
3.	<i>Mustā</i>	<i>Cyperus rotundus</i> Linn. (Rz.)	3 g
4.	<i>Bhūnimba (kirātātikta)</i>	<i>Swertia chirata</i> Buch. Ham.(Pl.)	3 g
5.	<i>Amṛta (gudūci)</i>	<i>Tinospora cordifolia</i> (Willd) micrs. (St.)	3 g

²¹ चन्द्रप्रभा वचा मुस्तं भूनिम्बामृतदारुकम् ।
हरिद्रातिविषा दार्का पिप्पलीमूलचित्रकौ ॥४०॥
धान्यकं त्रिफला चव्यं विडडगं गजपिप्ली ।
व्योषं माक्षिकधातुश्च द्वौ क्षारौ लवणत्रयम् ॥४१॥
एतानि शाणमात्राणि प्रत्येकं कारयेद बुधः ।
त्रिवृद्धन्तीपत्रकं च त्वगेला वंशरोचना ॥४२॥
प्रत्येकं कर्षमात्राणि कुर्यादेतानि बुद्धिमान् ।
द्विकर्षं हतलोहं स्याच्चतुष्कर्षं सिता भवेत् ॥४३॥
शिलाजत्वष्टकर्षं स्यादष्टौ कर्षाश्च गुगुलोः ।
एभिरेकत्र सद्भूषणैः कर्तव्या गुटिका शुभा ॥४४॥
चन्द्रप्रभेति विख्यातं सर्वरोगप्रणाशिनी।
प्रेमहान् विंशतिं कृच्छ्रं मूत्राघातं तथाशमरीम् ॥४५॥
विबन्धाऽनाहशूलानि मेहनं ग्रन्थिमर्बुदम् ।
अन्त्रवृद्धिं कटीशूलं श्वासं कासं विचर्चिकाम् ॥४६॥
अण्डवृद्धिं तथा पाण्डुं कामलां च हलीमकम् ।
कुष्ठान्यशर्सिं कण्डुं च प्लीहोदरभगन्दरम् ॥४७॥
दन्तरोगं नेत्ररोगं स्त्रीणामार्तवजां रुजम् ।
पुंसा शुक्रगतान् दोषान् मन्दाग्निमरुचिं तथा ॥४८॥
वायुं पित्तं कफं हन्याद् बल्या वृष्या रसायनी ।
चन्द्रप्रभायां कर्षस्तु चतुःशाणो विधीयते ॥४९॥
(शाङ्गधरसंहिता, मध्यमखण्ड, अध्याय ७;४०-४९)

6.	<i>Dāruka (devadāru)</i>	<i>Cedrus deodara</i> (Roxb.) Loud. (Ht. Wd.)	3 g
7.	<i>Haridrā</i>	<i>Curcuma longa</i> Linn. (Rz.)	3 g
8.	<i>Ativisā</i>	<i>Aconitum heterophy lum</i> Wall. (Rt. Tr.)	3 g
9.	<i>Dārvī̄ (dāruharidrā)</i>	<i>Barberis aristata</i> DC. (St.)	3 g
10.	<i>Pippalīmūla (Pippalī)</i>	<i>Piper longum</i> Linn. (Rt.)	3 g
11.	<i>Citraka</i>	<i>Plumbago zeylanica</i> Linn. (Rt.)	3 g
12.	<i>Dhānyaka</i>	<i>Coriandrum sativum</i> Linn. (Fr.)	3 g
13.	<i>Harītakī̄</i>	<i>Termenalia chebula</i> Retz. (P.)	3 g
14.	<i>Āmalakī̄</i>	<i>Embllick officinalis</i> Gaertn. (P.)	3 g
15.	<i>Bibhītaka</i>	<i>Termenalia belerica</i> Roxb. (P.)	3 g
16.	<i>Cavya</i>	<i>Piper chava</i> Hunter. (St.)	3 g
17.	<i>Vidaṅga</i>	<i>Embelia ribes</i> Burn.f. (Fr.)	3 g
18.	<i>Gajapippalī̄</i>	<i>Scindapsus officinalis</i> Schult. (Fr.)	3 g
19.	<i>Śunṭhī̄</i>	<i>Zingiber officinale</i> Rose. (Rz.)	3 g
20.	<i>Marica</i>	<i>Piper nigrum</i> Linn. (Fr.)	3 g
21.	<i>Pippalī̄</i>	<i>Piper longum</i> Linn. (Fr.)	3 g
22.	<i>Mākṣīka dhātu bhasma</i>	(<i>Mākṣīka</i>)	3 g
23.	<i>Yava kṣāra (yava)</i>	<i>Hordeum vulgare</i> Linn. (Pl.)	3 g
24.	<i>Sarjīkṣāra (svarjīkṣāra)</i>	<i>Fuller's earth</i>	3 g
25.	<i>Saindhava lavana</i>	Rock salt	3 g
26.	<i>Sauvarcala lavana</i>	Black salt	3 g
27.	<i>Vid lavana</i>	-	3 g
28.	<i>Trivṛt</i>	<i>Ipomoea turpethum</i> R.Br. (Rt.)	12 g
29.	<i>Dantī</i>	<i>Baliospermum montanum</i> Muell-Arg. (Rt.)	12 g
30.	<i>Patraka (tejapatra)</i>	<i>Cinnamomum tamala</i> Nees & Eberm. (Lf)	12 g
31.	<i>Tvak</i>	<i>Cinnamomum zeylanicum</i> Blume. (St. Bk.)	12 g
32.	<i>Elā (sūkṣmailā)</i>	<i>Elettaria cardamomum</i> Maton. (Sd.)	12 g
33.	<i>Vamsarochana (vamsa)</i>	<i>Bambusa bambos</i> Druce. (S.C.)	12 g
34.	<i>Louha bhasma</i>	Calx of Iron	24g
35.	<i>Sita</i>	Sugar	48 g
36.	<i>Śilājatu</i>	<i>Asphaltum panjabinum</i>	96 g
37.	<i>Guggulu</i>	<i>Commiphora wightii</i> (Arn.) Bhandari (Exd.)	96g

Dose: 250 to 500 mg

Anupāna : water, milk, gingily powder

Important therapeutic use: Vibandha; Ānāha; Śūla; Granthi; Pāṇḍu; Kāmalā; Mūtrakṛcchra; Prameha; Aśmarī; Arśa; Arbuda; Mūtrāghāta; Antravṛddhi; Kaṭiśula; Kuṣṭha; Kaṇḍū; Plīhodara; Bhagandara; Dantaroga; Netraroga; Aruci; Mandāgni; Strīroga; Ārtava rujā; Śukradosa; Daurbalya.

PHOTOGRAPHS OF SOME OF THE DRUGS USED IN PANGU

Amlaki

Emblica officinalis Gaertn



Bilva

Eagle marmelos Corr.



Ashvagndha

Withania somnifera Linn.



Atasi

Linum usitatissimum Linn.



Bala
Sida cordifolia Linn.



Bhunimba
Swertia chirayita Roxb. ex Flem



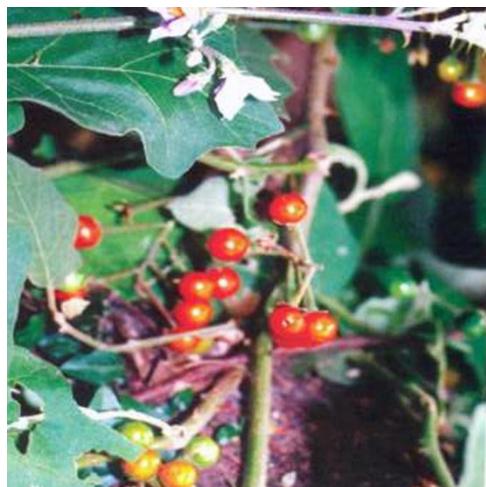
Bibhitaki
Terminalia bellirica Roxb.



Bidanga
Embelia ribes Burm. f.



Brihati
Solanum indicum Linn.



Chitrak
Plumbago zeylanica Linn.



Danti
Baliospermum montanum Muell. – Arg.



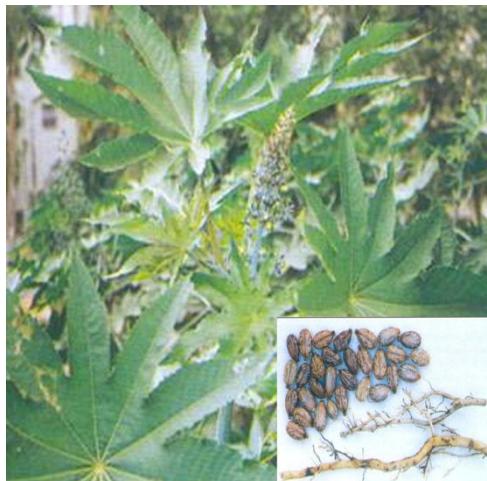
Daruharidra
Berberis aristata DC



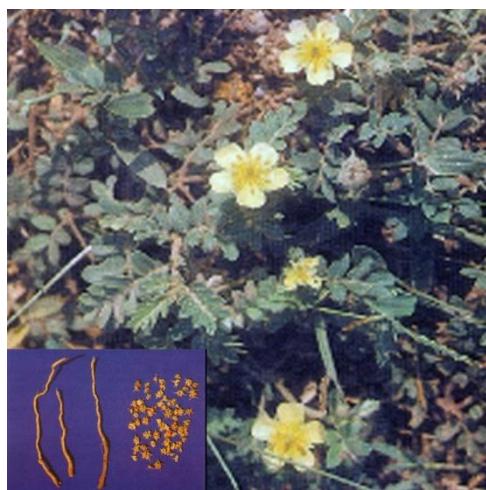
Dhanyaka
Coriandrum sativum Linn.



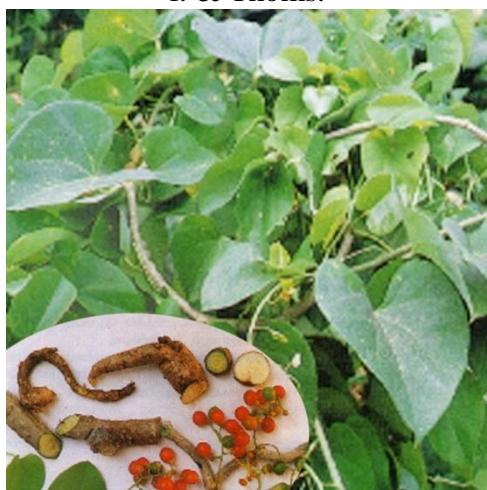
Eranda
Ricinus communis Linn.



Gokshura
Tribulus terrestris Linn



Guduchi
Tinospora cordifolia (Wild) Miers ex Hook.
f. & Thoms.



Guggulu
Commiphora mukul (Hook ex Stocks) Engl.



Haridra
Curcuma longa Linn.



Haritaki
Terminalia chebula Retz.



Kantakari
Solanum surattense Burm. f.



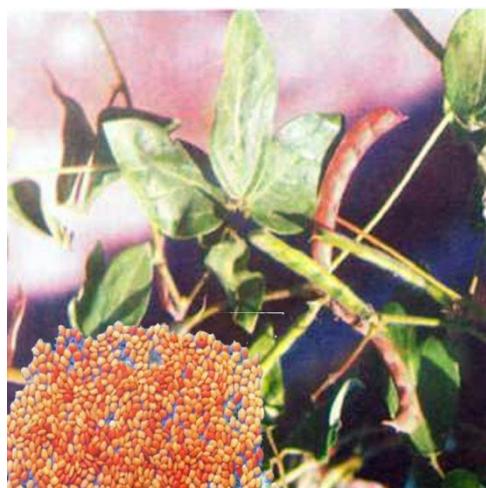
Kapikachu
Mucuna pruriens Linn.



Gambhari
Gmelina arborea Linn.



Kulattha
Dolichos biflorus Linn.



Lodhara
Symplocos racemosa Roxb.



Madhuка
Madhuca indica J.F.Gmel.



Maricha
Piper nigrum Linn.



Masa
Phaseolus mungo Linn.



Yashtimadhu
Glycyrrhiza glabra Linn.



Musta
Cyperus rotundus Linn.



Pippali
Piper longum Linn.



Shobhanjana
Moringa oleifera Lam.



Shalaparni
Desmodium gangeticum DC.



Shatavari
Asparagus racemosus Wild



Shyonaka
Oroxylum indicum Vent.



Shunthi
Zingiber officinale Roxb.



Trivrit
Operculina turpethum Linn.



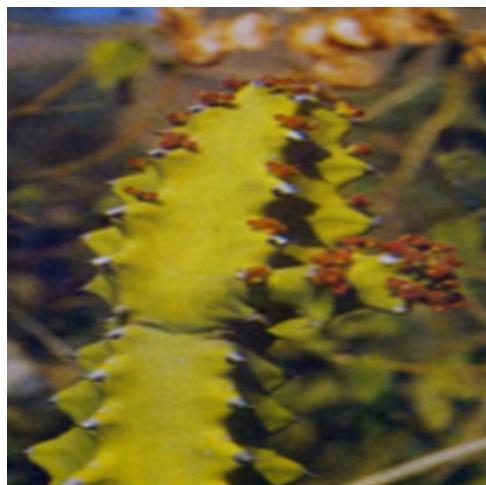
Vacha
Acarus calamus Linn.



Agnimantha
Clerodendrum phlomidis Linn.

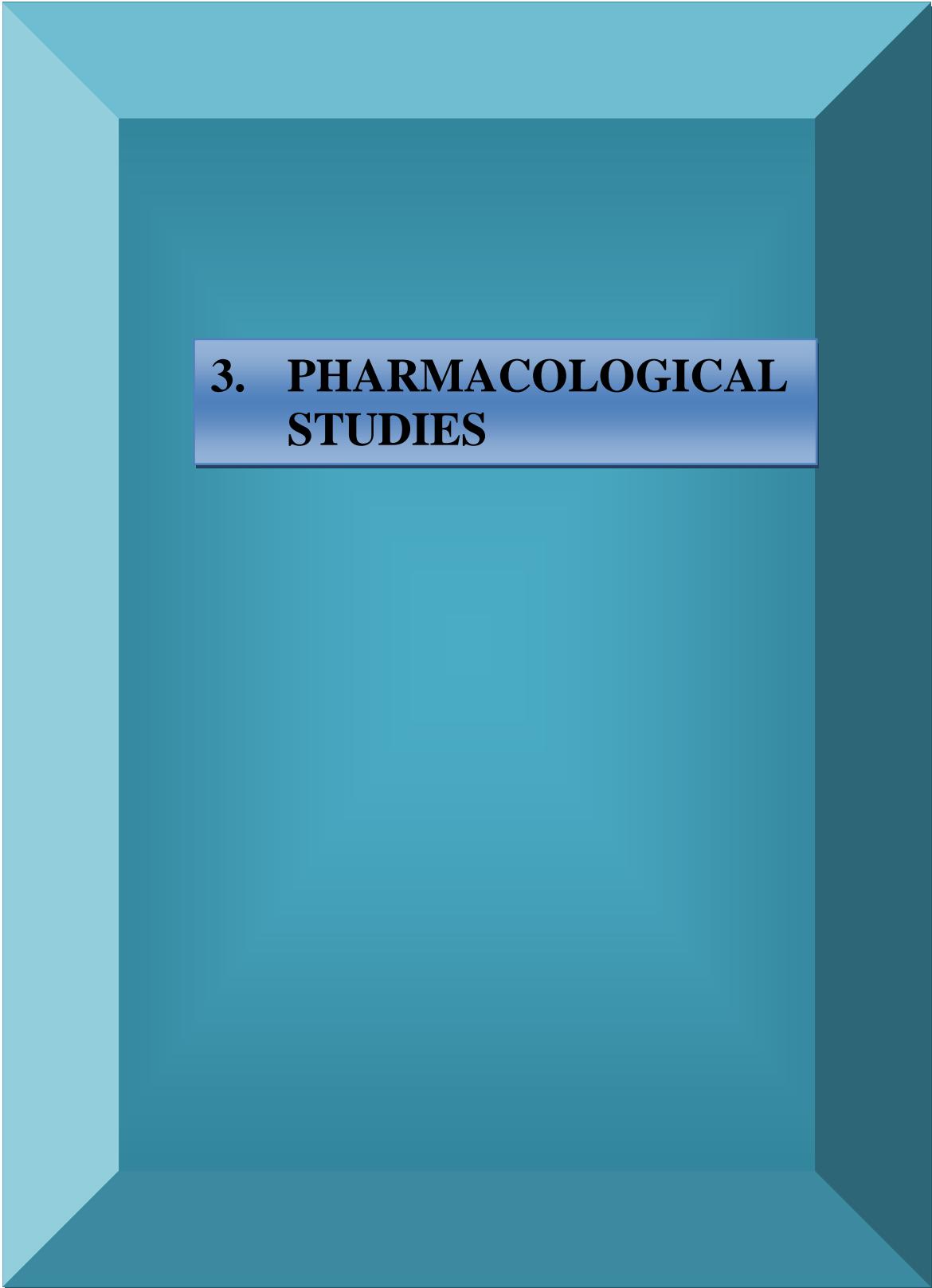


Snuhi
Euphorbia nerifolia Linn



Kushtha
Saussuria lappa C.B. Clarke





3. PHARMACOLOGICAL STUDIES

PHARMACOLOGICAL STUDIES

The plants used for clinical studies were subjected for pharmacological screening to ascertain their effectiveness. Detailed studies on *Sahacara* and *Nirgundi* were undertaken in the Pharmacology Department of the Institute.

A: Pharmacological studies on studies on *Strobilanthes heyneanus* Nees (*Sahacara*)

Strobilanthes heyneanus is a small aromatic shrub found all over Kerala (Family: Acanthaceae) and is used as *Sahacara* in various Ayurvedic preparations (Sahasrayoga, 1969). Identity of *Sahacara* is controversial. According to Ayurvedic Formulary of India (1978) *Barleria prionitis* Linn. *B. strigosa* Wild and *B. cristata* Linn. are the three species of *Sahacara*. Glossary of Indian Medicinal Plants (Chopra et.al.1956) and Indian Materia Medica (Nadkarni, 1976) described the above plants as *Vajradantç* and *Karunta*. However in South India, particularly in Kerala *Strobilanthes heyneanus* (Malayalam: Karim Kurinji) is used as *Sahacara*. According to Ayurvedic classics *Sahacara* is effective in neurological disorders (Caraka Samhitç, 1970), oedema, itching, skin diseases and healing of ulcer (Vaidya, 1936). The oil prepared from the whole plant is reported to be effective in *Gñdhrasç* (Sciatica) and in inflammatory conditions (Nair, 1985). It is also found to be beneficial in *Khaµja* (Paraplegia) and *Pa'gu*(Monoplegia), when given along with *Devad;ru* (Cedrus deodara) and áu,The (*Zingiber officinalis*). No detailed pharmacological studies on this plant are available (MAPIS, CSIR, New Delhi). Hence, pharmacological screening with various extracts of this plant was undertaken.

Materials and Methods

Shade dried coarsely powdered plant material from pharmacognostically identified (Botanical Survey of India, Coimbatore) plant was successively extracted with petroleum ether 60-80°

(PE), chloroform (CHE), ethanol 90% (ETE) and distilled water (AQE) using a soxhlet apparatus. ETE and AQE were dissolved and diluted to requisite concentration in distilled water. PE and CHE were macerated with few drops of Tween-80 to form a fine suspension and diluted to requisite concentration with distilled water. Extracts were administered intraperitoneally. Distilled water and Tween-80 were administered to control groups. In experiments involving drug interaction the extracts were administered 45 min. prior to drug administration. Dose was 100 mg Kg^{-1} of each extract unless otherwise mentioned. Wistar albino rats (120-150 g) and Swiss albino mice (20-30 g) of either sex maintained on Hindustan Lever rat feed and tap water and exposed to natural day and night cycles were used for all the experiments.

1. Acute toxicity: Graded doses of various extracts were administered to groups of mice (5 in each group) and were continuously observed for first four hours and afterwards at every 24 hrs for the next 72 hrs. Apparent toxicity and mortality were noted. LD₅₀ with 95% confidence limits was calculated as per Litchfield and Wilcoxon (1948).

2. Gross behaviour: Extracts were studied for their effect on gross behaviour in mice as described by Morpurgo (1971) in mice. Doses tested were 100, 200 and 400 mgKg^{-1} of each extract.

3. Hypnotic potentiation: Effect of various extracts on pentobarbitone (50 mgKg^{-1}) induced hypnosis in mice was studied as described by Bansinath *et.al.* (1982) latency of onset and duration of sleep were recorded.

4. Effect on muscle tone and Balance (forced locomotor activity, FMA) in mice:

Effect of various extracts on FMA was studied by noting their effect on the performance of trained mice on rotarod (MQ Lab.) as per the method of Kinnard and Carr. (1957).

5. Effect on spontaneous motor activity (SMA) in mice:

This was studied by noting the effect of various extracts on SMA in mice who have undergone habituation session in an actophotometer (Blue line activity cage) as described by Bansinath *et.al.* (1982).

6. Antipsychotic activity

- a) Effect on d-amphetamine induced stereotype in mice was studied for their effect in the extracts of the drug. Stereotype was scored as described by Valame and Gupta (1981).
- b) Effect on exploratory behaviour of mice: This was studied according to the method of Shillito (1970) using a tunnel board instrument. Mice exposed to natural day and night light cycle were used. Effect of the administration of various extracts on exploratory behaviour was measured by the number of different tunnels entered in the first minute and during the total observation period (5 min.) was noted. Total number of tunnels entered during the observation period was also noted.

7. Anti-depressant activity

- a) Anti-reserpine test was performed according to the method of Sheth *et.al.* (1972) by noting the effects of pretreatment with various extracts on reserpine (2.5 mgKg^{-1} I.P.) induced ptosis, catatonia and sedation in mice. None of the extracts (both stem and root extracts) could antagonize reserpine induced ptosis, catatonia and sedation.
- b) Behavioural despair test was carried out according to the method of Prosolit *et.al.* (1977) by noting the effects of the administration of various extracts on duration of mice immobility. Duration of mice immobility was not affected by pretreatment with various extracts (both stems and root extracts).

8. Anti-parkinsonism activity: All the extracts were screened for anti-parkinsonism activity by noting the effect of their prior administration on oxotremorine (500 mgKg^{-1} I.P.) induced tremors, head twitches, ataxia, salivation, lacrymation and diarrhoea as described by Plotnikoff and Kastin (1977). None of the extracts (both stem and root extracts) could

antagonize oxotremorine induced tremors, head twitches, ataxia, salivation, lacrymation and diarrhoea.

9. Anti-convulsant activity: These extracts were evaluated for anticonvulsant activity in mice by noting the effect of their pretreatment on electroconvulsion (induced by supra maximal electric shock 30 MA for 0.2 sec. Duration as described by Goodman *et.al.* 1953), pentylenetetrazol (100 mgKg^{-1} s.c. Winyard *et.al* 1952) and strychnine (2 mgKg^{-1} s.c. Patnaik *et.al.* 1979) induced convulsion. Number of mice protected and effect on seizure pattern were also noted.

Among stem extracts PE protected 2/6 mice and CHE 3/6 mice against electroconvulsion, ETE and AQE did not afford protection. Seizure pattern was not modified by any of the extracts. These extracts failed to protect mice against pentylenetetrazol and strychnine induced convulsions and also they did not modify seizure pattern. All the root extracts failed to protect mice against electroconvulsions as well as pentylenetetrazol and strychnine induced convulsions. They also did not modify seizure pattern. CHE prolonged the latency of onset of death against strychnine convulsions.

10. Analgesic activity

a) Evaluation by radiant heat method was carried out in mice using analgesiometer (INCO): Effect of various extracts on latency of tail flick response was noted at various time intervals.

b) Acetic acid writhing test in mice: Effect of the administration of various extracts on acetic acid (3% v/v, 0.1 ml./10 g body wt.) induced writhing was studied as described by Witkin *et.al.* (1961). Number of mice protected and number of stretching episodes in each mouse was noted.

11. Anti-inflammatory activity

a) Effect on carrageenan hind paw oedema: The extracts were tested for anti-inflammatory activity by noting their effect on carrageenin (1% in normal saline) induced hind paw oedema (acute inflammation) in rats. Carrageenan was injected beneath plantar aponeurosis as per

Winter *et.al.* (1962). Paw volume was measured by fluid displacement method of Singh and Ghosh (1968) before and 3 hr after carrageenin injection.

b) The effect of the stem extracts ETE and AQE was studied on cotton pellet granuloma formation in rats as per the method of Winter and Portar (1957). Sterilized cotton pellets weighing 10 mg were implanted (S.C.) in both the axillae and groins under ether anaesthesia. The extracts were administered daily for 6 consecutive days. On the 7th day the animals were sacrificed, cotton pellets were excised and dried in hot air oven at 60-70° C for 7 hours. Effect of these extracts on body wt., and weights of adrenal gland, liver and spleen as well as total and differential WBC counts were noted. Phenylbutazone group served as reference standard.

12. Immunosuppressant activity: The stem extracts were evaluated for immunosuppressant activity by noting the effect of their administration on humoral antibody response in mice following the procedure of Stone and Patet (1971). Betamethasone treated group served as reference standard. Effect of drug treatment on body wt. and wt. of adrenal, thymus and spleen were also noted.

13. Anti-microbial activity: All the extracts were screened for anti-bacterial activity against Pseudomonas aeruginosa, Escherichia coli and Staphylococcus aureus and for antifungal activity against Aspergillus fumigatus, T.rubrum, Violaceum and Microspora gypseum by disc diffusion method as described by Cruikshank *et.al.* (1975). Nutrient agar medium was used for antibacterial studies and Sabouraud's glucose agar medium for antifungal studies. All the extracts (both stem and root extracts) failed to inhibit the growth of bacterial and fungal organisms.

14. Studies on isolated tissues: Rat colon and rat uterus were set up for experimentation following standard procedures. Effect of extracts on tissues and their modifying effect, if any on the responses to sub-maximal doses of various spasmogen noted. Spasmogens used were acetylcholine (Rat colon) and 5-HT, oxytocin and prostaglandin F₂OC (Rat uterus).

RESULTS:

1. Acute Toxicity:

LD₅₀ mg Kg⁻¹ (95% confidence limits) of the various stem and root extracts were as follows:

Stem extract	Root extract
PE, 740.00 (328.89, 1665.00)	PE, 600.00 (300.00- 1200.00)
CHE, 1280.00 (800.00, 2048.00)	CHE, 650.00 (520.00 -.812.50)
ETE, 1400.00 (583.33, 3366.00)	ETE, No death observed upto 2000 mg.kg ⁻¹
AQE, 660.00 (388.24, 1122.00)	AQE, 1750 (813.95, 3762.50)

2. Gross behaviour:

Among stem extracts PE and CHE produced mild CNS depression at higher dose levels (400 mgkg⁻¹). ETE and AQE failed to produce any appreciable effect. Root extracts PE (200 MgKg⁻¹) and AQE (400 MgKg⁻¹) produced CNS depression characterized by hypo activity, ptosis and sedation. CHE did not affect gross behaviour while ETE (400 mgKg⁻¹) produced mild CNS stimulation.

3. Hypnotic potentiation:

Stem extract CHE shortened the duration of pentobarbitone sleep while PE, ETE and AQE failed to produce any effect. These extracts also failed to affect the latency of onset of sleep. Among root extracts PE and AQE significantly prolonged the duration of pentobarbitone sleep. CHE and ETE had no effect. None of the extracts affected latency of onset of sleep.

4. Effect on muscle tone and Balance (forced locomotor activity, FMA) in mice:

Both stem as well as root extracts (PE, CHE, ETE and AQE) failed to affect the performance of mice on rotarod.

5. Effect on spontaneous motor activity (SMA) in mice:

Stem extracts PE showed mild stimulation and AQE mild depression. CHE and ETE did not affect SMA. Root extracts AQE showed marked depression while PE, CHE and ETE had no marked effect.

6. Antipsychotic activity

a) Effect on d-amphetamine induced stereotype in mice:

None of the extracts (both stem as well as root extracts) could antagonize d-amphetamine stereotype.

b) Effect on exploratory behaviour of mice:

Among the stem extracts ETE depressed exploratory behaviour of mice. Number of different tunnels entered during first minute and during the entire observation period was significantly less and number of total tunnels entered was also less. AQE decreased only total number of tunnels entered during observation period. PE and CHE failed to affect exploratory behaviour significantly, Root extract PE increased the total number of tunnels entered during 5 min., observation period without significantly affecting number of different tunnels entered during the first minute and during total observation period, CHE, ETE and AQE did not affect exploratory behaviour.

7. Anti-depressant activity

a) Anti-reserpine test:

None of the extracts (both stem and root extracts) could antagonize reserpine induced ptosis, catatonia and sedation.

b) Behavioural despair test:

Duration of mice immobility was not affected by pretreatment with various extracts (both stems and root extracts).

8. Anti-parkinsonism activity:

None of the extracts (both stem and root extracts) could antagonize oxotremorine induced tremors, head twitches, ataxia, salivation, lacrymation and diarrhoea.

9. Anti-convulsant activity:

Among stem extracts PE protected 2/6 mice and CHE 3/6 mice against electroconvulsion, ETE and AQE did not afford protection. Seizure pattern was not modified by any of the extracts. These extracts failed to protect mice against pentylenetetrazol and strychnine induced convulsions and also they did not modify seizure pattern. All the root extracts failed to protect mice against electroconvulsions as well as pentylenetetrazol and strychnine induced convulsions. They also did not modify seizure pattern. CHE prolonged the latency of onset of death against strychnine convulsions.

10. Analgesic activity:

a) Evaluation by radiant heat method:

None of the stem extracts could raise the threshold of tail flick response. Among the root extracts PE, ETE and AQE failed to produce any significant effect. However, CHE produced a biphasic effect, initially a decrease in pain threshold which gradually increased with time (120, 180, 240 min. after the administration of extracts).

b) Acetic acid writhing test in mice:

Stem extracts ETE and AQE though failed to afford complete protection to any mice, significantly decreased the number of writhings. CE protected 2/6 mice and decreased the number of writhing, but the decrease is statistically non-significant. PE is inactive. Root extracts CHE and ETE treated groups showed significant decrease in acetic acid induced writhing episodes though none of the mice was protected completely PE failed to produce significant reduction.

11. Anti-inflammatory activity

a) Effect on carrageenan hind paw oedema:

Stem extracts PE, ETE and AQE produced significant suppression of carrageenin induced hind paw oedema in rats. CHE was inactive. Among root extracts CHE and AQE produced weak suppression of carrageenin induced hind paw oedema in rats. PE and ETE were inactive.

b) Effect on cotton pellet granuloma:

Stem extracts ETE and AQE significantly inhibited the development of granulomas formation. The inhibitory effect was almost similar to that of phenylbutazone. Both these extracts as well as phenylbutazone failed to affect body wt. and weights of adrenals and spleen. AQE and phenylbutazone group showed increase in the wt. of liver. Total leucocyte count in ETE, AQE and phenylbutazone treated rats was significantly increased compared to control rats. However there was no difference in differential count. Except in AQE treated group in which eosinophil count was significantly decreased.

12. Immunosuppressant activity:

Stem extract ETE markedly suppressed antibody formation when administered prior to immunization while enhancing antibody formation when administered after immunization. AQE markedly suppressed antibody formation in both the series like betamethasone. The effects could be compared to betamethasone which was however 100 times more potent. ETE in doses studied had no significant effect on the wt. of adrenal, thymus and spleen. AQE decreased the wt. of spleen without affecting the wt. of adrenal and thymus. In comparison wt. of all the three organs was significantly decreased in betamethasone treated group.

13. Anti-microbial activity:

All the extracts (both stem and root extracts) failed to inhibit the growth of bacterial and fungal organisms.

14. Studies on isolated tissues:

Stem extracts perse had no effect on rat colon. PE and CHE antagonized Ach induced spasm. ETE and AQE did not modify Ach spasm on rat colon. On rat uterus these extracts had no effect. However PE antagonized prostaglandin F₂OC effects. CHE antagonized oxytocin induced spasm. Other extracts were inactive. PE, CHE and ETE antagonized 5-HT induced spasm. AQE was inactive. All the root extracts failed to produce any effect on rat colon and also they did not modify responses to sub-maximal dose of ACH. They also did not produce any effect on rat uterus. PE, CHE and ETE antagonized 5-HT induced spasm while AQE was inactive. PE and CHE antagonized oxytocin induced contractions. ETE and AQE were inactive. PE antagonized prostaglandin F₂OC induced spasm while other three extracts were inactive.

DISCUSSION

Results of the studies show that the stem extracts have marked effect on CNS. However, they do not possess anti-psychotic, anti-depressant, anti-parkinsonian and central muscle relaxant activities as evident from their inability to modify d-amphetamine stereotype, reserpine effects, behavioural “despair”, oxotremorine induced tremors and FMA in mice. Though ETE depressed exploratory behaviour of mice, it can not be considered to represent anti-psychotic activity since it did not antagonize d-amphetamine stereotype, the primary screening test for anti-psychotics. PE and CHE showed weak anti-convulsant effect against electroconvulsion. However, both failed to protect mice against pentylenetetrazol and strychnine induced convulsions. ETE and AQE possess significant aspirin like analgesic effect since they antagonized acetic acid induced writhing in mice. These extracts also suppressed carrageenin hind paw oedema and granulation tissue formation in cotton pellet

granuloma indicating that they can influence both acute and sub-acute stages of inflammation. This is a significant finding when considered in the light of the fact that these extracts were prepared after thorough extraction of the plant material with petroleum ether (60-80° C) and chloroform which rules out the presence of steroid like constituents in them. Even though these extracts did not affect the wt. of the adrenals and AQE produced significant depletion of adrenal ascorbic acid suggesting that part of their effect might be through stimulation of adrenal activity. Depletion of adrenal ascorbic acid level is indicative of increased adrenal activity through adreno-corticotrophin release (Datta and Sanyal, 1978). It has been reported that lysosomal enzymes play an important role in acute and chronic inflammation (Lack, 1966, Houck, 1968). The activities of the enzymes are elevated in human rheumatoid synovia (Luscombe, 1963). Acidic anti-inflammatory drugs like non-steroidal anti-inflammatory agents may exert this beneficial effect by inhibiting activities of either released lysosomes or by stabilizing lysosomal membrane thereby inhibiting the release of materials responsible for inflammatory process (Tanaka and Lizuka, 1968 and Hariord and Smith, 1970). It has been reported that the inhibition of increased acid phosphatase in cotton pellet granuloma in rats with drugs aspirin, phenylbutazone, RG₄ and RG₇ (o-ethoxybenzamide derivatives) and indomethacin may be due to either inhibition of released acid phosphatase from the lysosomes or stabilizing the lysosomal membrane (Naik and Sheth, 1978). In the present study also there was significant increase of acid phosphatase activities in serum and liver of cotton pellet implanted rats. ETE produced significant decrease in acid phosphatase activity in serum just as PBZ. PBZ showed significant decrease in liver acid phosphatase activity while AQE and ETE failed to produce any effect. Since AQE did not affect acid phosphatase activity it does not seem to share the above mechanism of action. ETE was also found to produce significant decrease in serum alkaline phosphatase activity in cotton pellet implanted rats, the mechanism of action of which is not clear in relation to inflammatory process.

PBZ, ETE and AQE produced inhibition of glutamic pyruvic and glutamic oxaloacetic transaminases of serum of cotton pellet implanted rats. Also the drugs inhibited the GOT activities in the liver of the above groups. The drugs may be influencing the continuous formation of biologically active polypeptides like bradychinin. ETE and AQE suppressed antibody formation against SRBC in mice like betamethasone. Effect of betamethasone is due to its lymphoid mass reduction effect. It markedly decreased the weight of thymus and spleen. ETE did not affect the weight of thymus and spleen. AQE decreased the weight of spleen without affecting the weight of thymus significantly, indicating that they may not have similar mechanism of action. Increase in total leucocyte count in extracts and phenylbutazone treated groups is difficult to explain. PE and CHE produced antispasmodic effect against Ach in rat colon. On rat uterus PE antagonized PGF₂OC and 5-HT induced spasm. CHE antagonized oxytocin and 5-HT induced spasm while ETE antagonized 5-HT spasm only. Significance of this selective antagonism is difficult to interpret.

Among the root extracts AQE possess CNS depressant activity since it prolonged pentobarbitone induced sleeping and depressed SMA in mice though it did not affect forced locomotor activity. PE prolonged pentobarbitone sleep without affecting SMA and FMA indicating that it is devoid of true sedative effect. CHE raised the threshold of tail flick response and suppressed acetic acid writhing suggesting of analgesic activity while AQE suppressed acetic acid writhing without affecting the threshold of tail flick response. However, it does not seem to represent aspirin type of activity while considering the fact that they are inactive against carrageenin hind paw oedema in the same dose level. None of the extracts could inhibit the growth of bacteria and fungi studies indicating lack of antimicrobial activity. They also do not posses anti-inflammatory, anti-convulsant, anti-psychotic, anti-depressant and anti-parkinsonian activities as evident from their inability to modify the tests carried out to screen for their above activities. These extracts were inactive on isolated rat colon preparation. However, on rat uterus PE showed non-specific spasmolytic effect by

inhibiting 5-HT, PGF₂OC and oxytocin induced spasm. ETE inhibited only 5-HT, spasm while CHE antagonized 5-HT and oxytocin induced spasm. Though root extracts possess analgesic activity active principles in root does not seem to contribute to the therapeutic efficacy of the plant preparations in sciatica in view of lack of anti-inflammatory activity in them. Hence the reported efficacy of the medicinal preparations incorporating Strobilanthes heyneanus in sciatica and other inflammatory conditions seem to be due to the active principles present in its stem extracts and leaf extracts which are also found to possess analgesic and anti-inflammatory effect (Ravishankar, 1983).

B. Pharmacological studies on *Vitex negundo* Linn. (*Nirgundi*)

Plants belonging to genus *Vitex* are reputed to possess important therapeutic properties. Of the many species available in India, *Vitex negundo* is the species used extensively in the preparation of ayurvedic medicines. It is a large aromatic shrub, or small tree found throughout India. Important therapeutic properties described to it are vermifuge, tonic and antipyretic. An ointment prepared from the plant juice is used as hair tonic (Wealth of India). It is also claimed to be efficacious in curing inflammation of the joints due to rheumatoid arthritis. Bruised leaves are applied over sprained limbs, contusions, Leech bites and Scorpion stings; juice of the leaves is used to control fetid discharges and worms from ulcers (Nadkarni, 1976). *Nirgundi Taila* and *Nirgundi Kalpa* made out of the leaves are used as analgesics. It is also used as an important remedy in liver diseases, sciatica, migraine, mental deficiency, leprosy, skin diseases and as an anabolic agent (Vaidya, 1936). Anti-inflammatory (Singh, 1978) and anti-bacterial (Joshi and Magar, 1952) properties have also been reported with the leaves.

Root is also considered as tonic, febrifuge, expectorant and diuretic. Root bark tincture is recommended in cases of irritable bladder and rheumatism. Powdered root is used for treating piles and as a demulcent for dysentery. It is effective in dyspepsia, colic, worm

infestation, boils and leprosy (Nadkarni, 1976). A clinical trial of powder from the rhizome of *Alectra parasitica* which grows on the root of the plant is found to be effective in the treatment of leprosy without any toxic effects (Prasad, 1962). Analgesic (Srivastava and Sisodia, 1970), anti-inflammatory (Chaturvedi and Singh, 1965, Singh, 1978; Sharma and Singh, 1980 and Rathor, 1973) and diuretic properties (Vohra and Khan, 1981) with the plant extracts have been reported. Further, Nirgu, ;¢ preparations have been successfully employed for treating sciatica (Jain and Pande, 1976 and Tripathi, 1981) and rheumatism (Bhattacharya, 1981).

Though few studies are available in the different activities of the plant no systematic study has been attempted to define the pharmacological activity profile of the plant. As an attempt at delineating activity profile general pharmacological screening of different extracts of the leaves and root was undertaken in the pharmacology laboratory of this Institute.

Plant material used was collected from the Institutes medicinal plant garden. Shade dried, coarsely powdered plant material was successively extracted in a soxhlet in eluotropic series. Leaf extracts studied were petroleum ether 60-80°C (PE), chloroform (CHE), toluene (TLE), n-butanol (BE), 90% ethanol (ETE) and cold aqueous infusion (CAI) of the remaining mare.

Root extracts used were PE, CHE, BE, ETE and CAI. Animals Swiss albino mice (20 g, 30 g), Wistar albino rats (130, 160 g) of either sex, maintained on Hindustan lever's rat feed and tap water given ad libitum, exposed to natural day and night light cycles were used in the experiments.

RESULTS:

1. Acute toxicity: Graded doses of the extracts were administered to groups of mice (5-6 in a group) and continuously observed for 4 h. and then periodically for the next 72 h. Toxic effects and mortality if any were noted. Approximate LD₅₀ was calculated (Ghosh, 1971).

2. Gross behaviour: Effect of graded doses of extracts on gross behaviour in mice central nervous system was studied as described by Morpurgo (1971).

3. Hypnotic potentiation effect: This effect was studied by noting the effect of prior extract administration on pentobarbitone (50mg.Kg⁻¹ I.P.) and diazepam (25mg.Kg⁻¹) induced sleep in mice. Latency of onset and duration of sleep were noted as described by Baninath *et.al.* (1982).

4. Effect on Spontaneous Motor Activity, (SMA): Effect of extracts on SMA was studied as described by Bansinath *et.al.* (1982), in mice exposed to prior habituation sessions in an actophotometer (Blue line activity cage).

5. Effect on muscle tone and Balance (forced locomotor activity, FMA): The effect of extracts on FMA was studied by noting the effect of trained mice performance on rotord (MQ Lab.). None of the leaf and root extracts affected muscle tone and Balance in mice. However number of free rides were decreased in group treated with root extract CHE and increased in BE treated animals.

6. Anti-psychotic activity:

(i) Effect on exploratory behaviour of mice: Effect of extracts on the exploratory behaviour in mice was studied as described by Shillito (1970), using a tunnel board instrument. Number of different tunnels entered during first min. and the entire observation period was considered as index of exploration. Total number of tunnels entered was also noted to assess the effect on spontaneous activity.

(ii) Effect on d-amphetamine stereotype: Extract's effect on d-amphetamine (5 mgKg^{-1} I.P.) induced stereotype in mice was noted. Stereotype was scored as described by Valama and Gupta (1981).

(iii) Effect on secondary conditioned response (SCR), conditioned avoidance response (CAR) and unconditioned response (UR) in rats: Leaf extracts were evaluated for anti-psychotic effect by noting the effect of their administration of SCR, CAR and UR following the procedure described by Maffi (1958). Leaf extracts CAI and ETE inhibited SCR (2/6 rats).

7. Anti-depressant activity

(i) Anti-reserpine activity: Extracts effect on reserpine induced ptosis, catatonia and sedation in mice following the procedure of Sheth *et.al.* (1972).

(ii) Behavioural despair test: Effect of extracts on behavioural despair was studied as described by Porsolt *et.al.* (1977) by noting extracts effect on duration of mice immobility.

8. Anti-parkinsonian activity

Oxotremorine test: The effect of prior extract administration on oxotremorine (500mg.Kg^{-1} I.P.) induced tremors, head twitches, ataxia, lacrymation, salivation and diarrhoea were noted. The scoring was done as described by Plotnikoff and Kastin (1977).

9. Anticonvulsant activity

It was evaluated by employing three experimental models viz., supramaximal electric shock (MES, current strength 30 MA for 0.2 s.c. duration and Goodman *et.al.* 1953). Pentylenetetrazol (120 mgKg^{-1} s.c., Swinyard *et.al.* 1952) and strychnine (2 mgKg^{-1} s.c., Patnaik *et.al.* 1970) induced convulsions. Number of mice protected and affect on seizure pattern were noted.

10. Analgesic activity: Extracts was evaluated by employing two experimental models for analgsic activity.

a) Radiant heat method: Effect of extract on the threshold of tail flick response in mice was noted using an analgesiometer (INCO). Leaf extracts CHE and TLE raised the threshold of tail flick response moderately. PE, BE, ETE and CAI failed to produce any significant raise in pain threshold.

b) Acetic acid writhing test: This test was carried out as described by Witkin *et.al.* (1961) by noting the effect of extract administration in 3% acetic acid (v/v) induced writhing. Number of mice protected and number of writhing in each mouse was noted.

11. Anti-inflammatory activity

The extracts were screened for anti-inflammatory activity by noting their effect on carrageenin induced hind paw oedema in rats as described by Winter *et.al.* (1962). Paw volume was measured plethysmometrically as described by Singh and Ghosh (1968).

1. Acute toxicity:

The following is the approximate LD₅₀ of the leaf and root extracts respectively: PE 500 mgKg⁻¹ and 1000 mgKg⁻¹, CHE 500 mgKg⁻¹ and 1000 mgKg⁻¹, TLE 1000 mgKg⁻¹(leaf extract only) BE 1500 mgKg⁻¹ and 200 mgKg⁻¹, ETE 1000mg.Kg⁻¹ and 2000 mgKg⁻¹, CAI 3200 mgKg⁻¹ and 2000 mgKg⁻¹.

2. Gross behaviour:

Among the leaf extracts PE had no marked effect, mild CNS depression and writhing were noted at higher dose level (400 mgKg⁻¹), CHE and TLE produced CNS depression at 400 mgKg⁻¹, BE and ETE produced mild CNS stimulation at higher dose level (400 mgKg⁻¹), CAI had no significant effect at the dose tested (800 mgKg⁻¹). The root extracts CHE and ETE did not produce any apparent effect on CNS. PE and BE produced moderate CNS depression at higher dose level (400 mgKg⁻¹) and CAI at higher dose level (800 mgKg⁻¹) produced initial stimulation followed by depression.

3. Hypnotic potentiation effect:

The leaf extracts TLE, ETE and CAI prolonged both pentobarbitone and diazepam induced sleep. PE and BE prolonged the duration of pentobarbitone sleep without affecting diazepam induced sleep. None of the extracts affected latency of onset of sleep. The root extracts CAI did not prolong sleep induced by either diazepam or pentobarbitone. PE prolonged pentobarbitone sleep without significantly effecting diazepam sleep. CHE shortened the duration of diazepam sleep without affecting pentobarbitone sleep. BE and ETE prolonged diazepam sleep and did not affect pentobarbitone sleep. PE increased the latency of onset of diazepam induced sleep.

4. Effect on spontaneous motor activity, (SMA):

Leaf extracts PE, CAI and ETE depressed SMA in TLE treated group SMA was moderately stimulated. CHE and BE did not affect SMA significantly. Among root extracts ETE produced mild depression of SMA in mice while other extracts did not affect the same in mice.

5. Effect on muscle tone and Balance (forced locomotor activity, FMA):

None of the leaf and root extracts affected muscle tone and Balance in mice. However number of free rides were decreased in group treated with root extract CHE and increased in BE treated animals.

6. Anti-psychotic activity

(i) Effect on exploratory behaviour of mice:

The root extract PE significantly decreased the total number of tunnels entered during 5 min. period. However it did not affect number of different tunnels entered (1 min. and 5 min. duration). Other extracts did not affect exploratory behaviour of mice significantly.

(ii) Effect on d-amphetamine stereotype:

All the leaf and root extracts failed to antagonize d-amphetamine induced stereotype in mice.

(iii) Effect on secondary conditioned response (SCR), conditioned avoidance response (CAR) and unconditioned response (UR) in rats:

However they did not affect CAR and UR. Extracts PE, CHE, BE and TLE did not affect SCR, CAR and UR in rats.

7. Anti-depressant activity

(i) Anti-reserpine activity:

Leaf extracts PE and root extracts CHE, TLE, BE, ETE and CAI failed to modify reserpine induced ptosis, catatonia and sedation in mice.

(ii) Behavioural despair test:

Leaf extracts PE, CHE, BE and ETE did not affect duration of mice immobility. TLE and CAI prolonged the duration immobility. None of the root extracts affected duration of mice immobility.

8. Anti-parkinsonian activity

Oxotremorine test:

Root extracts BE and ETE produced marked antagonism of oxotremorine induced tremors, head twitches and lacrymation. BE antagonized diarrhoea and ataxia also. However, both these extracts did not antagonize oxotremorine induced salivation, whereas procyclidine antagonized all symptoms.

9. Anticonvulsant activity

Leaf extracts PE protected 3/5, BE 4/6 and other extracts 1/6 mice respectively against electroconvulsion. None of these extracts modified seizure pattern. All these extracts failed to protect pentylenetetrazol induced and strychnine induced convulsions. On the contrary PE, BE and CHE shortened the latency of onset of convulsions and death. All the root extracts failed to afford protection against electroconvulsion and pentylenetetrazol and strychnine induced convulsions except PE which protected 5/6 mice and also prolonged the latency of onset of seizures in pentylenetetrazol convulsions.

10. Analgesic activity:

a) Radiant heat method:

Leaf extracts CHE and TLE raised the threshold of tail flick response moderately. PE, BE, ETE and CAI failed to produce any significant raise in pain threshold. Only root extract ETE significantly increased the threshold of tail flick response while other extracts failed to produce this effect.

b) Acetic acid writhing test:

Though the leaf extracts PE, CHE, TLE and BE failed completely to protect any of the mice, marked decrease in the number of writhings were noted. ETE and CAI showed weak effect. Root extracts PE and ETE did not afford significant protection, CE showed moderate, BE and CAI marked antagonism towards acetic acid induced writhing.

11. Anti-inflammatory activity

Leaf extracts PE and CAI produced marked suppression of carrageenin induced hind paw oedema. TLE, BE and ETE produced moderate suppression while CHE had no effect. Root extract CE produced marked suppression of carrageenin induced hind paw oedema. BE, CAI and ETE produced weak to moderate suppression of oedema formation.

DISCUSSION AND CONCLUSION:

Of the different leaf extracts studied (PE, CHE, TLE, BE, ETE and CAI) only CHE and TLE produced discernible CNS depression at comparatively higher dose (400 mgKg^{-1}) as observed on gross behaviour in mice. TLE, ETE and CAI prolonged both diazepam and pentobarbitone sleep while PE and BE prolonged both diazepam and pentobarbitone sleep while PE and BE prolonged pentobarbitone sleep without affecting diazepam sleep. However these extracts did not affect the latency of onset of sleep indicating that their hypnotic potentiating effect is not

due to enhancement of permeability of hypnotic agents to CNS (Bansinath *et.al.* 1982). PE, CAI and ETE depressed SMA while in TLE treated mice moderate stimulation was noted. None of these extracts affected muscle tone and Balance in mice suggesting lack of central muscle relaxant effect.

These extracts lack anti-psychotic and anti-depressant effect as is evident from their inability to antagonize d-amphetamine induced stereotype in mice, to inhibit SCR and CAR in rats, antagonize reserpine effects and to reduce duration of immobility in mice (on the contrary CAI and TLE increased the duration of immobility). PE and BE showed moderate anti-convulsant effect against elector-convulsions while all these extracts failed to protect mice against pentylenetetrazol and strychnine induced convulsions. PE, CHE, TLE and BE significantly reduce the acetic acid induced writhing in mice while ETE and CAI showed weak effect. TLE and CHE raised the threshold of tail flick response significantly while other extracts failed in this respect. All these extracts except CHE produced marked suppression in carrageenin induced hind paw oedema indicating their anti-inflammatory activity. From this it is inferred that PE, TLE and BE possess aspirin like analgesic effect. CHE which is devoid of anti-inflammatory effect and did not antagonize acetic acid writhing indicate that its effect to increase the threshold of tail flick response might be through the CNS.

Among the root extracts studied (PE, CHE, BE, ETE and CAI) none possessed true sedative activity through PE and BE showed moderate CNS depressant activity in gross behaviour studies and PE prolonged pentobarbitone sleep without affecting diazepam sleep. None of them could affect SMA and FMA significantly unlike true sedatives, which depress both SMA and FMA. PE increased the latency of onset of diazepam sleep indicating that it may be delaying the passage of diazepam through CNS barriers. None of these extracts possess anti-psychotic activity since they failed to antagonize d-amphetamine stereotype in mice and did not suppress exploratory behaviour in mice. These extracts are also devoid of anti-depressant

activity as evident from their inability to antagonize reserpine effects and behavioural despair in mice. BE and ETE possess marked anti-parkinsonian activity since they produced significant antagonism of oxotremorine induced tremors, head twitches, ataxia, and lachrymation and BE in addition suppressed diarrhoea also. Even-though all these extracts failed to protect the animals against electroshock and strychnine induced convulsions PE protected the animals from pentylenetetrazol induced convulsion. ETE significantly raised the threshold of tail flick response without affecting acetic acid writhing in mice indicating that it may be due to its effect at CNS level. CHE, BE and CAI produced aspirin like analgesic activity by decreasing the number of acetic acid induced writhing without affecting the threshold of tail flick response. CHE produced significant anti-inflammatory effect while the other extracts produced weak to moderate effect.

Thus it is noticed that various extracts of the leaf and root of *V.negundo* Linn. possess anti-inflammatory and analgesic activities and the efficacy of this drug in rheumatism and other inflammatory conditions as reported in the Ayurvedic literature can be attributed to these properties.

CONCLUSION

The clinical research trials conducted in this Institute were found to be effective in management of *Khanja/Pangu* cases. The results were encouraging and statistically significant. On comparison of results obtained with different studies i.e. 4th study with *Sahacar;d¢* group was found to be the most effective and this encouraging result was observed in the *Sahacara* group also of the fifth study. In order to confirm the therapeutic potentialities of *Sahacara* and to find out the mechanism of action of this drug detailed pharmacological studies were undertaken in this Institute and the results obtained confirm its clinical claims in the management of *Pangu/Khauja*. The result obtained with *Nirgundi* group

in the fifth study was also encouraging and hence this drug was also taken up for the pharmacological studies. The results obtained were found to be supporting the clinical finds.

Bibliography

- Anonymous (1978): The Ayurvedic Formulary of India, Part I, Published by Govt. of India, Ministry of Health and Family Planning, Deptt. of Health.
- Anonymous (1987): Pharmacopoeial Standards for Ayurvedic Formulations, Published by Central Council for Research in Ayurveda and Siddha, New Delhi.
- Anonymous: Oushadhi Pharmacopoeia, Pharmaceutical Corporation of Indian Medicine.
- Alam, M.M., Joy, S. and Ali S.V. (1991): Screening of *Sida cordifolia* Linn. *Sida rhomboidea* Linn. and *Triumfetta rotundifolia* Lam. for Anti-inflammatory and Antipyretic Activities, Indian Drugs 28, 397-400.
- Amon, H.P.T., Safayhi, H., Mach, T. and Satiaraj, T. (1993): Mechanism of anti-inflammatory Action of Curcumin and Boswellic acids, J. Ethno Pharmacol.28, 113-119.
- Bansinath, M., Chandra Bose, A., Hema, S. and Guruswamy, M.N. (1982): Arch. Inter. Pharmacodyr. Therap.260 (1).
- Bhattacharya, C. (1981): Rheumatism, 16(3), p. 111-117.
- Bhavamishra (1969): Bhavaprakasha, Vidyodini Hindi Commentary by Sree Brahmashankara Shastri. The Chowkhamba Sanskrit Series Office, Gopalmandir Lane (P.O.), Chowkhamba, Post Box No.8, Varanasi, Vth Edition, page 393, Shloka 189-90.
- Bhavamishra (1969): Bhavaprakasha, Vidyodini Hindi Commentary by Sree BrahÀankara Shastri, The Chowkhamba Sanskrit Series Office, Gopalmandir Lane (P.O.), Chowkhamba, Post Box No.8, Varanasi, Vth Edition, Page 393, Shloka 189-90.
- Bhavamishra (1969): Bhavaprakasha, Vidyodini Commentary The Chowkhamba Sanskrit Series Office, Banaras, Page 366.

Bhavamishra (1969): Bhavaprakasha, Vidyodini Commentary, The Chowkhamba Sanskrit Series Office, Banaras, Page 199.

Borrie, P.C. (1971): Roxburg's Common Skin Diseases, 13th Edition, P. 6-7, Published by the English, Language Book Society and H.K. Lewis and Co. Ltd.

Caraka (1970): Caraka Samhita, Chikitsa, 28, 144, (Editor, Gangasahaya Pandeya), Chowkhamba Sanskrit Series Office, Varanasi-1.

Caraka (1970): Caraka Samhita 1st Edition, (Vidyotini Hindi Commentary by Kasinath S̄ishtri), Chowkhamba Sanskrit Series Office, Varanasi.

Caraka (1969): Caraka Part II, Siddhi I. Shloka 32, pp.886.

Caraka (1969): Caraka Samhita Part I, Vidyodini Hindi Commentary by Kashinadh Shastri, 1st Edition, Chowkhamba Sanskrit Series Office, Gopal Mandir Lane, P.O.Box-8, Varanasi, SootraSthana, Chapter-20, Shloka-11, page-269, ChikitsaSth̄na, Adhyaya-28, Shloka-11, page-662, ChikitsaSth̄na, Adhyaya-28, Shloka-72-74, page-703.

Caraka (1969): Caraka Samhita Part II Chapter I, Shloka 5, Page-801.

Caraka (1969): Caraka Samhita commentary Pt. Kasinatha Sastri, Edition I, The Chowkhamba Sanskrit Series Office, Varanasi, page: 174, 269, 713-14.

Chaturvedi, gN. and Singh, R.H. (1965): Indian J. Med. Res. 53(1), p. 71-80.

Chopra, R.N., Nayar, S.L. and Chopra, I.C. (1956): Glososary of Indian Medicinal Plants, Published by Publication and Information Directorate, CSIR, New Delhi, 33.

Cruicksjamk, R., Ouguid, T.P., Makmion, B.P. and Swain, R.H.A. (1975): Medical Microbiology, Vol.II, P. 202. Churchill Livingstone, London.

Deva, Radhakantha Raja (1967): Sabdakalpadruma, Chowkhamba Sanskrit Series Office, Varanasi.

Dutta, S. and Sanyal, S. (1978): Indian J. Exp. Biol., Vol. 16, p.166.

Gosh, M.N. (1971): Fundamentals of Experimental Pharmacology, P.41-42, Scientific Book Agency, Calcutta.

Goodman, L.S., Grewal, M.S., Broown, W.C. and Swinyard, E.A. (1953): J. Pharmac. Exp. Therap. 108, P.168-176.

Goodhart Robert, S and Shilo Mauriee, E. (1980): Modern nutrition in Health and Diseases, Edn. 6, K.M. Varghese Company, Bombay, page : 1265.

Govt. of India (1976): Ayurvedic Formulary of India, Govt. of India Press, Faridabad, First edition, *Vijagutika prakarana*, page-146.

Govindasa (1983): Baishyjaratnavali, Published by Chowkhamba Sanskrit Samsthan, Varanasi.

Hariford, D.J. and Smith, M.J.R. (1970): J. Pharma. Pharmacol., Vol. 22, P. 578.

Houck, J.C. (1968): Biochem. Pharmacol. Suppl., Vol.I.

Jain, P.K. and Panda, T.N. (1976): J. Res. Ind. Med. Yoga and Homoeo., 11 (2), P. 97-102.

Joshi, C.G. and Magar, N.G. (1952): Jour. Sci. Indus. Research, Vol. II-B, P. 261.

King, P.R.N. and King, E.J. (1954): J. Clin. Path., Vol.7, P.332.

Kinnard, W.J. and Carr, C.J. (1957): Journal of Pharmacology and Experimental Therapeutics, Vol. 121, P. 354-361.

Krishnan Vaidyan, K.V. and Gopala Pillai, S. (1969): Sahasrayoga, Sujanapriya commentary, Edn. 10, Vidyarambham Press and Book Depot Private Ltd., Alleppey, page : 315-316.

Krishnan Vaidyan, K.V. (1969): Sahasrayoga, (Sujana Priya Malayalam Commentary), Xth Edition, Vidyarambham Press and Book Depot Pvt. Ltd., Mullakkal, Alleppey.

Krishnan Vaidyan, K.V.(1969): Sahasrayoga, Sri Ramavilasam Press, Quilon (Kerala State), P.142.

Lack, C.H. (1966): Proc. R. Soc. Med., Vol.59, P.875.

Litchfield, J.T. and Wilcoxon, F. (1948): J. Pharmac. Exp., Therap., Vol. 96, P. 99-113.

Luscombe, M. (1963): Nature, Vol. 197, P. 1010.

Madhavakara (1955): Madhavanidana (Madhukosha Commentary byVijayarakshita and Srikantha Dutta), 5th Edition, Nirnayasagar Press, Bombay.

- Maffi, g (1958): Journ. Pharm.Pharmacology, Vol.II, P.129-139.
- Mehi, J.W. (1945): J. Diol. Chem., Vol. 157, P.173.
- Misra, B. (1961): Bhavaprakasha, Part II (Vidyotini Hindi Commentary by Pandit Sri Brahma Sankara Mishra), Chowkhamba Sanskrit Series Office, Varanasi.
- Morpurgo, C. (1971): Arzneim Forsch Drug Res., 21 (11), P. 1727-1734.
- Nadkarni, A.K. (1976): Indian Materia Medica, Vol.I, P.1171-1172, Popular Book Depot, Bombay.
- Naik, S.R. and Sheth, U.K. (1978): Indian J. Exp. Biol., Vol. 16, P. 1175.
- Patnaik, gK., Sabir, M. and Dhawan, B.N. (1979): Indian J. Exp. Biol., Vol. 17, P. 391-396.
- Parsolt, R.O., Bartir, A. and Jalfre, M. (1977): Arch. Int. Pharmacodyn., 229, P. 327-336.
- Petersdorf Adams, Braunwald and Isselbacher Martin, Wilson (1984): Harrison's Principles of Internal Medicine, McGraw Hill, New York.
- Plotnikoff, N.P. and Kastin, A.J. (1977): Advances in Bio-Chemical Psychopharmacology, Vol.17, P.92, Raven Press, New York.
- Prasad, B.N. (1962): Leprosy Rev., 33(3) P. 207-209.
- Pontis, V.V. and Grampurohit, N.D. (1994): Anti-inflammatory activity of the Creams Containing Turmeric and Red Sandal Wood, Indian Drugs 31, 117-118.
- Ramachandran Nair, P., Vijayan, N.P., Madhavikutty, P., Prabhakaran, V.A. and Indirakumari, S. (1985): Jour. Res. Ayu. Siddha, Vol.6, No.2, P. 121-131.
- Ramachandran Nair, P.(1984): Ancient Science of Life, Vol.IV, No.I, July 1984, pp. 20-26.
- Ramachandran Nair, P. (1986): Journal of Research in Indian Medicine, April-June, 1986.
- Ramachandran Nair, P. (1989): JRAS, Vol.X, No.1-2, pp. 30-40, 1989.
- Ramachandran Nair, P. (1992): JRAS, Vol.XIII, No.1-2, pp. 14-26, 1992.
- Ramachandran Nair, P. (1994): JRAS, Vol.XV, No.3-4, pp. 98-114, 1994.

Ramachandran Nair, P. (1994): A comparative study of Sahacaradi taila, Nirgundi taila in Khanja and Pangu, Journal of Research and Education in Indian Medicine, Banaras.

Ramachandran Nair, P., Vijayan, N.P., Bhagavathy Amma, K.C. and Madhavikutty, P. (1984): Action of Sahacaradiyoga in Khaja and *Pa'gu'*, Ancient Science of Life IV (1) : 20-27.

Ramachandran Nair, P., Vijayan, N.P., Bhagavathy Amma, K.C., Pillai, B.K.R, Ravindran, K.C. and Madhavikutty, P. (1987): Role of Sodhana treatment in Khanja and Pangu' Ayu July : 13-36.

Ramachandran Nair, P., Vijayan, N.P., Madhavikutty, P. and Indirakumari, S. (1986): A comparative study of Sahacaradi taila and Nirgundi taila in the management of Khanja (Monoplegia) and Pangu (Paraplegia). The Journal of Research and Education in Indian Medicine, 5 (2) : 13-16.

Ravishankar, B. *et.al.* (1987): Analgesic, anti-inflammatory and Immunosuppressant effect of Strobilanthes heyneanus Nees, Stem., J.R.A.S., Vol.VIII, No.1 and 2, pp.53-63.

Rathor, R.S. (1973): Nagarjun, 16(2), P. 25-29.

Ravishankar, B. (1983): Abstracts of Papers. XVI Annual Conference of Indian Pharmacological Society, Indian Journal of Pharmacology, Vol. 16, No.1, P. 46-47.

Raymond, D., Adams, M.D. and Maurice Victor, M.D. (1989): Principles of Neurology, McGraw Hill, New York.

Reitman, S. and Prankel, S. (1957): Amer. J. Clin. Path., Vol.28, P. 56.

Roe, J.J. and Kuether, C.A. (1943): J. Biol. Chem. Vol. 143, P. 399.

Sharngadhara (1931): Sargadhara Samhita, IIInd Edition, Adhamall's Dipika and Kasiram's Gudhartha Dipika (Sanskrit Commentary by Pandit Parasurama Shastri, Vidyasagar), Nirnaya Sagar Press, Bombay.

Sharma, Priyavrata (1969): Dravyaguna Vijnana, The Chowkhamba Vidya Bhavan, Varanasi, pp. 81-82, 69-71, 550-560.

- Satyavati, G. V., Raina, M.K. and Sharma, M. (1976): Medicinal Plants of India, Vol. I, P 314-315 and 524, Published by Indian Council of Medical Research, New Delhi.
- Sharma, A.K. and Singh, R.H. (1980): Bull. Med. Ethno. Bot. Res., 1(2), P. 262-271.
- Sheth, U.K., Dadkar, N.K. and Usha G Kamat (1972): Selected Topics in Experimental Pharmacology, P. 151.
- Shillito, E. (1970): Brit. J. Pharmac., 40, P.113-128.
- Singh, R.H. (1978): Rheumatism, Vol. 13, P.99.
- Singh, H. and Ghoosh, M.N. (1968): J. Pharm. Pharmacol., 21, P. 126.
- Shrivastava, S.C. and Sisodia, C.S. (1970): Indian Vet. J., 47 (2), P. 170-175.
- Stone, R.L. and Paget, C.J. (1971): Screening Methods in Pharmacology (Ed: R.A.Turner, and Peter), N. Academic Press, New York, Vol.II, P. 145-163.
- Sushruta (1966): Susrutha Samhit; (Commentary by Kaviraj Ambikadutta Shastri), Chowkhamba Sanskrit Series Office, Varanasi.
- Sushruta (1966): Sushruta Samhita Nidana 1 : 77 (Ayurveda tatva sandeepika commentary), Chowkhamba Sanskrit Series Office, Banaras.
- Sushruta (1966): Sushruta Samhita, Ayurveda Tatva Sandeepika commentary: Kaviraj Dr. Ambikadatta Shashtri. Edition 11, Chowkhamba Sanskrit Series Office, Varanasi.
- Swinyard, E.A., Brown, W.C. and Goodman, L.S. (1952): J. Pharmac. Exp. Therapy, 106, P. 319-330.
- Tanaka, K. and Lisuka, Y. (1968): Biochem. Pharmacol., Vol. 17, P.2023.
- Taranath Tharka Vachaspati (1970): Vachaspathyam, Chowkhamba Sanskrit Series Office, P.B.8, Varanasi.
- Tripathi, C.P. (1981): Sachitra Ayurved, 33(7), P. 498-499.
- Vagbhatta (1956): Ashtangahrdaya. Bhageerathi Pt. Sri. Taradatta panta Ayurvedacharya, Chowkhamba Sanskrit Series Office, Varanasi, Page : 5, 23, 85, 86, 226, 236, 352, 355, 567.

Vagbhata (1957): Ashtangahrdaya.Sutra 6: 167, 197 and Chikitsa 22 : 23. Chowkhamba Sansrit Series Office, Banaras.

Vagbhata (1957): Ashtangahrdaya.Nidana 15 : 45 Chowkhamba Sanskrit Series Office, Banaras.

Vagbhata (1957): Ashtangahrdaya. Sutra 12 : 9 Chowkhamba Sanskrit Series Office, Banaras.

Vagbhata (1957): Ashtangahrdaya.Nidanam 15 : 5, 6. Chowkhamba Sanskrit Series Office, Banaras.

Vagbhata (1957): Ashtangahrdaya.Shareera 3 : 68, 69, 4 : 5, 19. Chowkhamba Sanskrit Series Office Banaras.

Vagbhata (1957): Ashtangahrdaya.Sutra 15 : 5, Chowkhamba Sanskrit Series Office, Banaras.

Vagbhata (1957): Ashtangahrdaya.Sutra 19 : 64. Chowkhamba Sanskrit Series Office, Banaras.

Vagbhata (1956): Ashtangahrdaya., Moolam, Chowkhamba Sanskrit Series Office, P.O.Box-8, Varanasi, NidanaSthana, Chapter-1, Shloka 14-15, page 271.

Vagbhata (1956): Ashtangahrdaya., NidanaSthana, Chapter-15, Shloka-5-45, page 355.

Vagbhata (1956): Ashtangahrdaya., Moolam, SutraSthana, Adhyaya-12, Shloka-9, page 86.

Vagbhata (1956): SareeraSthanam, Adhyaya-4, Shloka-19-20, page 236.

Vagbhata (1956): SutraSthana, Adhyaya-17, Shloka-29-30, page 122.

Vagbhata (1956): SootraSthana, Adhyaya-19, Shloka-1, page 129.

Vagbhata (1956): SootraSthana, Adhyaya-19, Shloka-85-86, page 138.

Vallame, S.P. and Gupta, K.C. (1981): Indian J. Pharmac., 13 (2) P. 203-204.

Vaidya, K.M. (1936): The Ashtangahrdaya Kosha. The Mangalodayam Press, Trichur, pp. 601.

Vangasena (12th Centuary): Vangasena (Vatavyadhi Adhikara), 126.

Velayudha Kurup (1968): Sahasrayogam, Vaidhapriya Malayalam Commentary, Sri Rama Vilasam Press, Kollam, Kerala, VIIIth Edition, Tailapradakaranam 67, page 320-321.

Vohra, S.B. and Khan, M.S.Y. (1981): Indian Drugs Plarm. Ind., 16 (1), P. 39-40.

Winter, C.A. and Porter, C.C. (1957): Jour. Amar. Pharm. Assn., Vol. 46.

Winter, C.A., Riseley, E.A. and Nyss, G.W. (1962): Proc. Soc. Expt. Biol. Med. III, P. 544-547.

Witkin, L.B., Huebner, C.P., Galdi, F., O'Keefe, E., Spitaletta, F. and Plummer, A.J. (1961): J. Pharmac. Expt. Therap., P. 133,400.

4. CLINICAL STUDY

Materials and Methods:

Aims and Objectives:

1. To find out an effective therapy for the management of *Pangu* (Paraplegia)

Centres of study: CRI (A), Cheruthuruthy; RRI (A), Nagpur

No. of Studies: 04 (Four)

Study-I

Center of study: CRI (A), Cheruthuruthy

In this study 20 patients suffering from *Pangu* were selected for study and divided randomly in two groups, having 10 patients in each group. Single blind I.P.D. level study was conducted.

Treatment schedule: The regimen of treatment had been prescribed below:

Group I: Šamana therapy:

- i. *Ekāṅgavīra Rasa* (125 mg) along with *Erandā Taila* (5 to 10 ml) tds for 60 days
- ii .*Mahāmāsa Taila* (50 ml. Every day, externally used for *Abhyanga*) for 60 days

Group II: - Šodhana therapy:

Snehapāna, Yogavasti (Anuvāsana & Nirūha) and Abhyanga with Mūrcchhita Tila Taila for 60 days.The following procedure was adopted for *Šodhana* purpose:

Procedure	Drug and duration
Patient was kept under observation	1 to 7 days
<i>Snehapāna</i>	Oral administration of <i>Mūrcchhita Tila Taila</i> , starting with 50 ml. initially and increased by 50 ml. on consecutive days(8th to 14th day) or till <i>Samyaka snigdha Lakshanas</i> were observed.
<i>Vāspasveda</i> (Steam fomentation)	On 15th, 16th, 17th days

<i>Virecana</i> (Purgation therapy)	On 18th day oral administration of <i>Erandā Taila</i> 30 ml. on empty stomach in the morning at 6AM.
<i>Samsarjana karma</i> (Specific diet schedule)	3 times every day <i>Kanji</i> diet (rice gruel) with <i>Mudga</i> (Green gram) ie. Easily digestable diet for 7 days. (19th, 20th, 21st 22nd 23rd, 24th, 25th, 26th days), it depends upon the type of Suddhi.
<i>Abhyāṅga</i> (massage)	With <i>Mūrchhita Tila Taila</i> on 27th, 28th, 29th, 30th, 31st, 32nd & 33rd days
<i>Anuvāśana Vasti</i>	With 240 ml. of <i>Mūrchhita Tila Taila</i> on 34th, 36th, 38th, 40th & 41st days
<i>Nirūhavasti Vasti</i>	With <i>Erandā Mūla Kasāya</i> (480 ml.) + <i>Mūrchhita Tila Taila</i> (240 ml.) + honey (240 ml.) + <i>Śatāhvā kalka</i> (30 gm) and <i>Saindhava</i> (15 gm.) on 35th, 37th & 39th days
Follow up	From 42nd day to 60th day

Study-II

Centre of study: CRI (A), Cheruthuruthy

In this study, 80 patients suffering from *Panigu*, 40 patients in each group were taken randomly. Single blind I.P.D. level study was conducted.

Treatment schedule: The regimen of treatment has been prescribed below:

Group I - Patients were treated with *Śamana* therapy:

- i. *Gorocanādi Guṭikā*: 1tablet (250 mg) thrice daily along with *Aśvagandhā Kvāṭha*, 60 ml. till 60 days.
- ii. *Balāshvagandhalākṣādi Taila*: 50 ml. every day for *Abhyāṅga* till 60 days.

Group II –*Śodhana (Pañcakarma)* therapy was given in following way:

Procedure	Drug/Therapy and duration
1. <i>Snehapāna</i>	Oral administration of <i>Mūrchhita TilaTaila</i> starting with 50 ml. and increased by 50ml. every day up to 7 days or till the appearance of <i>Samyak Snigdha Laksana</i>
2. <i>Vāspasveda</i>	Applied for 3 days
3. <i>Virecana</i>	On 7th day oral administration of <i>Eranda Tail</i> , 30 ml. on empty stomach in the morning at 6AM.
4. <i>Samsarjana</i> (Specific diet schedule)	<i>Kanji</i> diet (rice gruel) 3 times every day with <i>Mudga</i> (Green gram) ie. Easily digestable diet up to 7 days (from next day after <i>Virechana</i>).
5. <i>Vasti-karma</i>	<p><i>YogaVasti</i> - 5 <i>Anuvāsana</i> and 3 <i>Nirūha vasti</i> were administered to each patient in the following manner-</p> <p><i>AnuvāsanaVasti</i> - <i>Mūrchhita TilaTaila</i> (240 ml.) along with <i>Saindhava</i> (15gm.)</p> <p><i>Nirūha Vasti</i> - <i>Erandamūla Kvātha</i> (600 ml.)+ <i>Mūrchhita TilaTaila</i> (180 ml.) + <i>Honey</i> (180 ml.)+ <i>Satapushpa Kalka</i> (30 gm.) + <i>Saindhava Lavana</i> (15 gm.).</p> <p>Order of Vasti administration:</p> <p><i>Anuvāsana Vasti</i> : 1st, 3rd, 5th, 7th, and 8th day</p> <p><i>Nirūha Vasti</i>: 2nd, 4th and 6th day</p>

Abhyanga with *Mūrchhita TilaTaila* was given for the rest of the days ie. 33 days after completion of the panchakarma treatment. *Abhyanga* was applied for 30 minutes every day.

Diet Schedule: *Kāñjī* diet (rice gruel) two times a day was given during *snehapāna* period; *Kāñjī* diet (rice gruel) three times a day was given during *Vāspa sveda*, *Samsarjana krama* period, and *Vasti* period.

Following diet schedule was apllied for *Abhyanga* period in group II (60 days) and group I, last 33 days.

Morning Milk (200 ml.) + Sugar 10 gm.

Kāñjī diet (Rice gruel) 150 gm. + Pickles
Noon - Rice (250 gm.) + Vegetables (200 gm.)

Evening *Kāñjī* (Prepared from Rice) 150 gm. + Green gram (25 gm.)

During the period of *Snehapāna*, light diet with *Kāñjī* or *Manda* (if appetite increases gradually).

Study-III

Centres of study: CRI (A), Cheruthuruthy

In this study 99 patients suffering from *Pāngu* were taken in two groups. 41 patients have completed the study in Group I and 58 patients have completed the study in Group II.

Treatment schedule: The regimen of treatment has been prescribed below:

Group I:

- i. *Daśamūlabalā Kvātha* (60 ml.) two times in a day and *Candraprabhā Vati*, 250 mg two times in a day for 45 days.
- ii. *Abhyanga* with *DaśamūlaBalā taila* and *Mātrābasti* with *DaśamūlaBalā*

Taila (50ml) on alternate days till 45 days.

Group II: - *Sodhana* therapy was applied in following way-

Procedure	Drug/Therapy and duration
<i>Snehapāna</i>	Oral administration of <i>DaśamūlaBalā taila</i> starting with 50 ml. and increased by 50 ml. everyday for 7 days or till appearance of <i>Samyak Snigdha Laksana</i> . Thereafter patient were advised to take <i>Kāñjī</i> diet (Rice gruel 2 times in a day).
<i>Vashpa sweda</i>	After massage (Abhyanga) of <i>DaśamūlaBalā taila</i> all over the body for 3 days. Thereafter the patients were given <i>Kāñjī</i> three times a day.
<i>Virecana</i>	Oral administration of <i>Erandā taila</i> (20-30ml.) for one day only, early in the morning at 6 AM. on empty stomach. Thereafter the patients were advised to take <i>Kāñjī</i> as pathya
<i>Samsarjana krama</i> (diet schedule)	Light and easily digestible <i>Kanji</i> diet (rice gruel) with <i>Mudga</i> (green gram) 3 time in a day up to 7 days.
<i>Vasti karma</i>	<p><i>Yogabasti</i>- 8 <i>Vasti</i> (5 <i>Anuvāsana</i> and 3 <i>Nirūha basti</i>) were administered to each patient in the following manner-</p> <p>Order of <i>Vasti</i> administration:</p> <p>1st day <i>Sneha vasti</i>: <i>DaśamūlaBalā taila</i> (240ml.) + <i>Saindhava lavana</i>(15gm.)</p> <p>2nd day <i>Nirūha vasti</i>: <i>DaśamūlaBalā Kvātha</i> (600ml.) + <i>DaśamūlaBalā Taila</i> (180ml.) + Honey (180ml.) + <i>Śatāhvā Kalka</i> (30gm.) + <i>Saindhava</i> (15gm.)</p> <p>3rd, 5th, 7th and 8th day <i>Sneha vasti</i> (Same as on 1st day)</p> <p>4th, 6th, day <i>Nirūha vasti</i> (Same as on 2nd day)</p>
<i>Abhyanga</i>	With <i>Daśamūla Balā Taila</i> 50 ml. every day for 15 days.
<i>Daśamūlabalā Kvātha</i>	Oral Administration in dose of 60 ml twice a day for 45 days.

Study-IV

In this study 36 patients suffering from *Pangū* were taken in two groups. 20 patients have completed the study in Group I and 16 patients have completed the study in Group II.

Centers of study: - CRI (A), Nagpur

Treatment schedule: The regimen of treatment has been prescribed below:

Group I :

Duration of treatment: Total duration of treatment was 45 days with following regimen -

- i. *DaśamūlaBalā Kvātha* 60 ml.+ *CandraprabhāVati* 250 mg B.D.
- ii. *Abhyanga* with *Daśamūla Balā taila* 50 to 100 ml.
- iii. *Mātrā Vasti* with *Daśamūla Balā taila* 30 to 60 ml
- iv. Physiotherapy x 6 weeks (was done by a Physiotherapist of Sri Ayurveda

Mahāvidyālaya, the modalities of Physiotherapy are Passive exercise, Active exercise, Prospective Neuromuscular facilitation (PNF) exercise, Electromagnetic stimulation if needed, heat to relieve pain, Ankle tone movement, cycling if needed, hand mobilization, sensory strategies, etc.)

Group II:

In this group Panchakarma therapy was applied as follows

Procedure	Drug/Therapy and duration
<i>Snehapāna</i>	<i>Daśamūlalabala taila</i> was given orally in 30 ml. quantity in increasing order on consecutive 7 days i.e. 30 ml, 60 ml, 90 ml., 120 ml, 150 ml, 180 ml, 210 ml. (Till <i>Samyak Snigdha lakṣana</i> -3-7days)
<i>Svedana</i>	<i>Vāspa sveda</i> till 3 days.
<i>Virecana</i>	Oral administration of <i>Erandā taila</i> , 30ml for 1 day only, early in the morning at 6 AM. on empty stomach.
<i>Samsarjana</i>	Light and easily digestible <i>Kāñjī diet</i> (rice gruel) with <i>Mudga</i> (green gram)

<i>krama-</i>	3 time in a day for 7 days. This depends upon <i>Śuddhi lakṣaṇa</i>
<i>Vasti karma</i>	<p><i>Yoga Vasti</i> (8 days)-</p> <p><i>Nirūha Vasti</i> with <i>Daśamūla Balā Kvātha</i> (3 days) and <i>Anuvāsana</i> with <i>Daśamūla Balā taila</i> (5 days)</p> <p><i>Anuvāsana vasti:</i> <i>DaśamūlaBalā taila</i> (240ml.) + <i>Saindhava lavana</i>(15gm.)</p> <p><i>Nirūha vasti:</i> <i>DaśamūlaBalā Kvātha</i> (600ml.) + <i>DaśamūlaBalā Taila</i> (180ml.) + Honey (180ml.) + <i>Śatāhvā Kalka</i> (30gm.) + <i>Saindhava</i> (15gm.)</p> <p>Order of Vasti administration:</p> <p><i>Anuvāsana Vasti:</i> 1st, 3rd, 5th, 7th and 8th day (after light diet)</p> <p><i>Nirūha Vasti:</i> 2nd, 4th, 6th, day.(in empty stomach)</p>

Criteria for Inclusion

1. Age between 20 to 70 years of either sex
2. Chronicity < 2 years
3. Inability to raise the lower limbs
4. Inability to walk
5. Presenting signs of paralysis
6. Hypotonia/Hypertonia of lower limbs
7. Exaggeration/Absence of knee jerks and ankle jerks
8. Impaired sensation
9. Retention/Incontinence of urine and/or faeces

Criteria for exclusion:

1. Age, below 12 years and above 70 years
2. Chronicity beyond 2 years.
3. Tuberculosis of the hip joint with complications of high fever, etc.
4. Pelvic pathology, if any
5. Any deformity/neoplasm of the spine (Lumbo sacral region)

6. Post fracture sequel.
7. Any systemic disease such as Hypertension, Peptic Ulcer, etc.
8. History of Liver diseases in recent past.
9. History of Renal diseases.
10. Serious complications associated with any other systemic disease.
11. Bed sores

Criteria for assessment:

Detail clinical history and physical examination of all the patients (taken for trial) has been recorded as per the proforma. The clinical, pathological and biochemical investigations also have been done. The result of the study has been assessed on the basis of improvement in the signs and symptoms of the disease.

Grading of symptoms:

1. Pain	Gradation
Normal	0
Mild	1
Moderate	2
Severe	3
2. Control over Urinary bladder	
Normal	0
Loss of control at times	2
Partial control	3
With sensation but without control	4
Complete retention	5
3. Control over rectum	
Normal	0
With sensation but without control	3
Complete retention	5
4. Sensory changes	
Normal	0
Can not appreciate fully but almost normal	4
Impaired sensation	8
Partially appreciated	12

No sensation	16
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5. Muscle Power

Normal	0
Can attain and regain squatting position	2
Can climb upstairs without any help	4
Can walk without support and can climb	6
Upstairs by using the banister	
Can walk with support	8
Patient can stand with support	10
Straight leg raising test about 10°	12
Semi flexion in all joints	14
Can perform slight flexion in all joints (Withdrawal on pricking)	16
Slight lateral and medial rotation of limb	18
Flickering movements	20
No movement	22

6. Fasciculation

Normal	0
Mild	1
Moderate	2
Severe	3

7. Electric Chorea

Normal	0
Moderate	1
Severe	2

8. Clonus

Absent	0
Ankle (Rt.-2+Lt.-2)	4

9. Tone

Normal	0
Hypertonia	2
Hypotonia	4

10. Knee reflexes

Normal	0
Exaggerated	2
No response	4

11. Ankle reflexes

Normal	0
Exaggerated	2
No response	4

12. Plantar reflexes

Normal	0
Extensor	4
No response	6

13. Abdominal reflexes

Present	0
Absent	2

14. Pressing power

25kg and above	0
20-25 kg	2
10 to 20 kg	4
Up to kg	6

15. Wasting

Normal	0
Mild	3
Moderate	6
Severe	9

16. Walking speed

Up to 20 second	0
21-30 second	1
31-40 second	2
41-50 second	3
51-60 second	4
60 and above	5

Criteria assessment of responses of therapy:

Assessment of the results is made mainly on the basis of relief in clinical symptomatology of the disease.

a. Good Response:

- i) Complete recovery from the disease.
- ii) Significant improvement in the well being of the Patients specially with reference to working capacity of the affected part.
- iii) Alteration in the Laboratory findings.

- b. **Fair Response:**
 - i) 50% and more improvement in symptoms and signs of disease, initially recorded.
 - ii) Satisfactory improvement in the well being of the patient.
 - iii) Improvement in the working capacity of the affected parts.

- c. **Poor response:** 25% and above improvement in symptoms.

- d. **No response:** No improvement in any of the symptoms.

Criteria for withdrawal:

The patient will be withdrawn from the study if he/she falls in one or more of the following criteria:

1. Left against Medical Advice.
2. Development of complications due to present illness or otherwise.
3. Aggravation of symptoms.

Type of Study: Single blind

Level of Study: In Patient Department (IPD) only

Follow Up:

For a period of 3 months on a regular interval of fortnight, either through postal correspondence or on personal attendance of the patients at the field station where he/she has been registered for the trial.

Source of the Procurement of the Drug:

Daśamūla Balā Kvātha and *Daśamūla Balā Taila* have been prepared in the Institute,s Pharmacy according to the Ayurvedic Formulary of India. However, *Chandraprabha Vati* has been procured from IMPCL, Mohan, Tarikhett. *Ekāngavīra Rasa* was procured from CRI Patiala. *Goracanadi Gutika* was procured from Oushadhi, Thrissur. *Mahāmāṣa Taila* was prepared at CRIP, Cheruthuruthy and *Erandā Taila* was procured from local Market. The drug were prepared as per references of Ayurvedic Formulary of India(AFI).

LABORATORY INVESTIGATIONS

A. Haematological

- a) Total Leucocyte count
- b) Differential Leucocyte count
- c) Haemoglobin percentage
- d) E.S.R.

MONITORING OF STUDY:

The progress of the study has been monitored by the team comprising of Clinicians, Pathologist, Radiologist, Neurologist (if available) and Statistician.

STATISTICAL ANALYSIS:

Before treatment and after the treatment, data on Signs/ Symptoms and laboratory parameters have been taken into account for diagnosis. Assessment of Result of Treatment is tabulated and analysed using suitable statistical methods.

DEMOGRAPHIC DATA:

Distribution of Patients according to age:

Age groups	No. of patients	Percentage (%)
12-20	20	8.92
21-30	31	13.83
31-40	37	16.51
41-50	57	25.44
51-60	52	23.21
61-70	27	12.05
Total	224	100.00

In these studies it was observed that, more number of patients (25.44%) were belong to the age group of 41-50 years, followed by 23.21% in 51-60 years of age group, 16.51% in 31-40 years of age group and 13.83% patients in 21-30years of age group.

Distribution of patients according to sex:

Gender	No. of patients	Percentage (%)
Male	155	69.19
Female	69	30.80
Total	224	100.00

As per sex wise distribution, more number of patients (69.19%) were male, followed by female patients (30.80%).

Distribution of patients according to Duration of diseases:

Duration in Days	No. of Patients	Percentage (%)
<180	43	29.86
<360	25	17.36
<540	33	22.91
<720	16	11.11
<900	27	18.75
Total	144	100.00

As per the chronicity of the disease, maximum patients i.e. 29.86% were reported with the duration of < 180 days followed by 22.91% patients with the chronicity of disease of <540 days and 18.755 patients reported with in <900 days.

RESULT

Study-I

Distribution of patients as per the severity of pain while walking:

Pain	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	5	50%	6	60%	5	50%	9	90%
Mild	0	0%	3	30%	1	10%	1	10%
Moderate	5	50%	1	10%	4	40%	0	0%
Total	10	100%	10	100%	10	100%	10	100%

In group-I, before treatment, 5 (50%) patients were suffering from moderate pain during walking and 5 (50%) patients were normal. After completion of the therapy it was found that, 6 (60%) patients became normal followed by 3(30%) patients suffering from mild pain and only one patient (10%) was suffering from moderate pain.

In group-II before treatment, moderate pain was found in 4(40%) patients followed by 1(10%) patients suffering from mild pain. After completion of treatment, 9(90%) patients became normal, followed by mild pain in 1(10%) of patients.

Distribution of patients according to the control over bladder:

Bladder Control	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	3	30%	7	70%	7	70%	7	70%
Loss of Control	0	0%	1	10%	0	0%	1	10%
Partial Control	4	40%	1	10%	3	30%	2	20%
With sensation but without control	0	0%	1	10%	0	0%	0	0%
Complete retention or complete incontinence	3	30%	0	0%	0	0%	0	0%
Total	10	100%	10	100%	10	100%	10	100%

In group I before treatment, 3(30%) patients were normal, 4(40%) patients were suffering from partial control over bladder, followed by 3(30%) were having complete retention of urine. After completion of treatment it was found that 7(70%) patients became normal, followed by loss of control, partial control and with sensation but without control of bladder in 1(10%) patients.

In group II before treatment 7(70%) patients were normal followed by 3(30%) patients were having partial control over the bladder. After completion of the treatment it was found that 7(70%) were normal, 2(20%) patients were having partial control and 1(10%) patient were having loss of control over the bladder.

Distribution of patients according to the control over rectum:

Rectum Control	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	3	30%	5	50%	6	60%	9	90%
With sensation but without control	4	40%	4	40%	3	30%	1	10%
Complete retention or complete incontinence	3	30%	1	10%	1	10%	0	0%
Total	10	100%	10	100%	10	100%	10	100%

In group I, before treatment 3(30%) patients were normal, 4(40%) patients were having sensation but without control over rectum and 3(30%) were having complete retention. After completion of therapy it was found that 5(50%) patients were normal, 4(40%) were having sensation but without control over rectum and 1(10%) was having complete retention of stool.

In group II, 6(60%) patients were normal before treatment, followed by 3(30%) patients were having with sensation but without control over rectum and 1(10%) patient was suffering from complete retention of stool. After treatment it was revealed that, 9 (90%) patients became normal and 1(10%) having with sensation but without control over rectum.

Distribution of patients according to the sensory changes:

Sensory changes	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	6	60%	7	70%	7	70%	7	70%
Can not appreciate fully but almost normal	1	10%	1	10%	0	0%	1	10%
Impaired Sensation	0	0%	0	0%	2	20%	1	10%
Partially appreciated	1	10%	2	20%	1	10%	1	10%
No sensation	2	20%	0	0%	0	0%	0	0%
Total	10	100%	10	100%	10	100%	10	100%

In group I, before treatment 6(60%) patients were normal, 1(10%) patient could not appreciate the sensation fully but almost normal, 1(10%) could partially appreciate the sensation and 2(20%) were having no sensation at all. After completion of therapy 7(70%) patients were normal, 1(10%) patient could not appreciate the sensation fully but almost normal and 2(20%) patient could partially appreciate the sensation.

In group II 7(70%) patients were normal before the treatment, 2(20%) patients were having impaired sensation and 1(10%) patient could partially appreciate the sensation. After completion of therapy it was found that, 7(70%) patients became normal and 1(10%) patient could not appreciate the sensation fully, could partially appreciate the sensation and having impaired sensation each.

Distribution of patients according to the Muscle Power:

Muscle power	Group I				Group II			
	BT		AT		BT		AT	
	No. of Pts	%						
Normal	0	0%	1	10%	0	0%	1	10%
Can attain and regain squatting position	0	0%	2	20%	0	0%	2	20%
Can climb upstairs without any help	0	0%	1	10%	0	0%	1	10%
Can walk without support and can climb upstairs by using the banister	1	10%	4	40%	4	40%	2	20%
Can walk with support	5	50%	0	0%	0	0%	0	0%
Patient can stand with support	0	0%	1	10%	3	30%	1	10%
Straight leg raising test about 10° (SLR)	1	10%	1	10%	1	10%	1	10%
Semi-flexion in all joints	1	10%	0	0%	0	0%	0	0%
Can perform slight flexion in all joints	0	0%	0	0%	0	0%	1	10%
Slight lateral and medial rotation of limb	0	0%	0	0%	0	0%	1	10%
Flickering movement	0	0%	0	0%	1	10%	0	0%
No movement	2	20%	0	0%	1	10%	0	0%
Total	10	100%	10	100%	10	100%	10	100%

In group I, 5(50%) patients could walk with support, 2(20%) patients had no movement of the lower limbs and 1(10%) patient could walk without support and could climb upstairs by using the banister, had straight leg raise test about 10° and semi flexion in all joints before treatment. After treatment it was observed that 1(10%) became normal, followed by 4(40%) patients could walk without support and could climb upstairs by using the banister, 2(20%) patient could attain and regain squatting position and 1(10%) patient could climb upstairs without any help, could stand with support as well as straight leg raising test was about 10° each.

In group II, before treatment, 4(40%) patients could walk without support and could climb upstairs by using the banister, 3(30%) patients could stand with support, 1(10%) had straight leg raising test about 10^0 , flickering movement and no movement of legs each. After treatment it was observed that, 1(10%) patient was normal, 2(20%) could attain and regain squatting position and could walk without support and could climb upstairs by using the banister each and 1(10%) could climb upstair without any help, can stand with support, straight leg raising test was about 10^0 , could perform slight flexion in all joints and slight & medial rotation of limb each.

Distribution of patients according to the Fasciculations:

Fasciculation	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	6	60%	6	60%	5	50%	7	70%
Mild	0	0%	2	20%	1	10%	1	10%
Moderate	4	40%	2	20%	4	40%	2	20%
Total	10	100%	10	100%	10	100%	10	100%

In group I before treatment, 6(60%) patients were normal and 4(40%) had moderate degree of fasciculation. After completion of treatment 6(60%) became normal and 2(20%) each had mild and moderate degree of fasciculation.

In group II, before treatment 5(50%) patients were normal, followed by 4(40%) had moderate fasciculation and 1(10%) with mild fasciculation. After completion of treatment it was found that, 7(70%) patients were normal, followed by 2(20%) had moderate fasciculation and 1(10%) had mild fasciculation.

Distribution of patients according to the Electric chorea:

Electric chorea	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	8	80%	10	100%	8	80%	9	90%
Moderate	2	20%	0	0%	0	0%	1	10%
Severe	0	0%	0	0%	2	20%	0	0%
Total	10	100%	10	100%	10	100%	10	100%

In Group I, before treatment, 8(80%) patients were normal (without any electric chorea) followed by 2(20%) patients were reported with moderate chorea. After treatment all patients 10(100%) became normal. While in group II, before treatment 8(80%) patients were reported as normal followed by 2(20%) patients of severe chorea. After treatment total 9 (90%) patients became normal & only 1(10%) patient was reported with moderate chorea.

Distribution of patients according to the clonus:

Clonus	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Absent	8	80%	8	80%	8	80%	9	90%
Present	2	20%	2	20%	2	20%	1	10%
Total	10	100%	10	100%	10	100%	10	100%

In group I, before treatment, clonus' was absent in 8(80%) patients and present in 2(20%) patients where as no changes was recorded after completion of the treatment.

In group II, before treatment, clonus' was absent in 8(80%) patients and present in 2(20%) patients, which became absent in 9(90%) patients and present in 1(10%) patient after completion of the treatment.

Distribution of patients according to the Muscle tone:

Muscle Tone	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	0	0%	5	50%	0	0%	2	20%
Hypertonia	6	60%	4	40%	5	50%	4	40%
Hypotonia	4	40%	1	10%	5	50%	4	40%
Total	10	100%	10	100%	10	100%	10	100%

In group I before treatment, hypertonia was present in 6(60%) patients followed by hypotonia in 4(40%) patients. After treatment, it was observed that 5 (50%) patients became normal, followed by hypertonia in 4(40%) patients and hypotonia in 1(10%) patients.

In Group II before treatment, hypertonia was recorded in 5 (50%) patients & rests of the 5 (50%) were having hypotonia. After treatment, 2(20%) patients became normal, followed by 4(40%) patients were recorded with hypertonia and hypotonia each.

Distribution of patients according to the Knee Reflexes:

Knee Reflex	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	1	10%	5	50%	1	10%	2	20%
Exaggerated	6	60%	4	40%	7	70%	6	60%
No response	3	30%	1	10%	2	20%	2	20%
Total	10	100%	10	100%	10	100%	10	100%

In group I before treatment, 1(10%) patient was normal, 6(60%) were reported with exaggerated knee reflex and 3(30%) patients with no response. After treatment it was found that, 5(50%) patients became normal followed by 4(40%) patients with exaggerated knee reflex and 1(10%) patient with no response.

In group II before treatment, 1(10%) patient was reported as normal, 7(70%) patients were reported with exaggerated knee reflex followed by 2(20%) patients with no response. After completion of the treatment it was found that, 2(20%) patients were normal, 6(60%) patients were reported with exaggerated knee reflex and 2(20%) patients with no response to the knee reflex.

Distribution of patients according to the Ankle Reflexes:

Ankle Reflex	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	0	0%	3	30%	0	0%	2	20%
Exaggerated	6	60%	6	60%	5	50%	4	40%
No response	4	40%	1	10%	5	50%	4	40%
Total	10	100%	10	100%	10	100%	10	100%

In group I before treatment, exaggerated ankle reflex was found in 6(60%) patients and no response to the Ankle reflexes was found in 4(40%) patients. After completion of the

therapy, it was found in normal condition in 3(30%) patients, in exaggerated form in 6(60%) and 1(10%) patient with no response.

In group II before treatment, 5(50%) of the patients had exaggerated ankle reflex and 5(50%) was found with no response. After completion of the therapy 2(20%) patients had shown normal response, 4(40%) patients had exaggerated ankle reflex & no response each.

Distribution of patients according to the Plantar Reflexes:

Plantar Reflex	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	0	0%	3	30%	0	0%	2	20%
Extensor	8	80%	6	60%	7	70%	5	50%
No response	2	20%	1	10%	3	30%	3	30%
Total	10	100%	10	100%	10	100%	10	100%

In group I before treatment, extensor planter reflex was found in 8(80%) patients and 2(20%) patients were found with no response. After completion of the therapy, 3(30%) patients were reported as normal, 6(60%) with extensor planter reflex and 1(10%) with no response.

In Group II before treatment, extensor planter reflex was found in 7(70%) patients, followed by 3(30%) with no response. After treatment 2(20%) patient were recorded as normal, 5(50%) were presented with extensor plantar response and 3(30%) patients with no response.

Distribution of patients according to the Abdominal Reflexes:

Abdominal Reflex	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Present	1	10%	7	70%	7	70%	8	80%
Absent	9	90%	3	30%	3	30%	2	20%
Total	10	100%	10	100%	10	100%	10	100%

In group I before treatment, abdominal reflex was absent in 9(90%) patients and present in 1(10%) patients. After treatment it was found present in 7(70%) patients and absent in 3(30%) patients.

In Group II before treatment, 7(70%) patients had abdominal reflex present and 3(30%) were reported with absence of abdominal reflex. After completion of therapy, 8(80%) had shown presence of abdominal reflex and 2(20%) patients had no abdominal reflex.

Distribution of patients according to the Pressing power:

Pressing Power	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
25 kg and above	0	0%	4	40%	0	0%	4	40%
20-25 Kg	0	0%	0	0%	1	10%	2	20%
10 to 20 Kg	3	30%	4	40%	4	40%	1	10%
Up to 10 Kg	7	70%	2	20%	5	50%	3	30%
Total	10	100%	10	100%	10	100%	10	100%

In Group I before treatment, 7(70%) patients were recorded having Pressing power up to 10 kg weight and 3(30%) patients were having pressing power of 10-20 kg. weight. After completion of treatment, 4(40%) of patients became enable to press 25 kg and above weight and 4(40%) of patients could press 10-20 kg weight, followed by 2(20%) could press up to 10 kg. only.

In group II before treatment, 5(50%) patients were recorded having pressing power up to 10 kg weight, followed by 4(40%) patients with pressing power of 10-20 kg weight and 1(10%) patient with 20-25 kg weight. After treatment, 4(40%) patients were found having pressing power of 25 kg weight and above, 3(30%) patient having pressing power of 10 kg. weight, 2(20%) patients could press 20-25 kg weight & 1(10%) patient had capacity to press 10-20 kg weight.

Distribution of patients according to the Wasting of Muscles:

Wasting of Muscles	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	7	70%	10	100%	10	100%	10	100%
Mild	1	10%	0	0%	0	0%	0	0%
Moderate	1	10%	0	0%	0	0%	0	0%
Severe	1	10%	0	0%	0	0%	0	0%
Total	10	100%	10	100%	10	100%	10	100%

In Group I before treatment, Wasting of muscle was absent in 7(70%) patients, 1(10%) patients were reported with severe, moderate and mild wasting of muscles respectively. After treatment all patients 10 (100%) became normal.

In case of group II, no patient was reported with wasting of muscle before and after the treatment.

Distribution of patients according to the Walking Speed:

Walking Speed	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Up to 20 second	0	0%	2	20%	0	0%	4	40%
21-30 second	0	0%	3	30%	0	0%	2	20%
31-40 second	0	0%	2	20%	4	40%	0	0%
41-50 second	3	30%	1	10%	0	0%	0	0%
51-60 second	1	10%	0	0%	0	0%	0	0%
60 second & above	6	60%	2	20%	6	60%	4	40%
Total	10	100%	10	100%	10	100%	10	100%

In Group I before treatment, 6(60%) patients took 60 second and more time to cover 20 meter of distance, followed by 3(30%) patients, who took 41-50 seconds and 1(10%) patients can walk in 51-60 seconds. After the treatment 3(30%) patients could cover the specified distance within 21-30 seconds, followed by 2(20%) could cover within 20 seconds, 31-40 seconds, 60 seconds and above respectively.

In group II, 6(60%) patients were able to cover the specified distance within 60 seconds & above followed by 4(40%) patients who covered within 31-40 seconds. After treatment 4(40%) patients became able to cover the distance within 20 seconds and within 60 seconds & above each and 2(20%) could cover within 21-30 seconds.

Effect of treatment on various Symptoms:

Symptoms	Group I			Group II		
	Z	p-value	Statistical Significance	Z	p-value	Statistical Significance
Pain (BT-AT)	-1.89	>0.05	Not Significant	-2.07	<0.05	Significant
Bladder Control (BT-AT)	-2.22	<0.05	Significant	-1.00	>0.05	Not Significant
Rectum Control (BT-AT)	-1.41	>0.05	Not Significant	-1.63	>0.05	Not Significant
Sensory changes (BT-AT)	-1.63	>0.05	Not Significant	-1.00	>0.05	Not Significant
Muscle power (BT-AT)	-2.67	<0.05	Significant	-2.46	<0.05	Significant
Fasciculations (BT-AT)	-1.41	>0.05	Not Significant	-1.41	>0.05	Not Significant
Electric chorea (BT-AT)	-1.41	>0.05	Not Significant	-1.34	>0.05	Not Significant
Clonus (BT-AT)	0.00	>0.05	Not Significant	-1.00	>0.05	Not Significant
Tone (BT-AT)	-2.07	<0.05	Significant	-1.34	>0.05	Not Significant
Knee (BT-AT)	-1.86	>0.05	Not Significant	-1.00	>0.05	Not Significant
Ankle Reflex (BT-AT)	-1.86	>0.05	Not Significant	-1.34	>0.05	Not Significant
Plantar Reflex (BT-AT)	-1.89	>0.05	Not Significant	-1.41	>0.05	Not Significant
Abdominal Reflex (BT-AT)	-2.45	<0.05	Significant	-1.00	>0.05	Not Significant
Pressing Power (BT-AT)	-2.56	<0.05	Significant	-2.23	<0.05	Significant
Wasting of muscles (BT-AT)	-1.60	>0.05	Not Significant	0.00	>0.05	Not Significant
Walking Speed (BT-AT)	-2.55	<0.05	Significant	-2.23	<0.05	Significant

Effect of *Shamana* therapy (group I) was found statistically significant ($p<0.05$) in case of Control over bladder, Muscle power, Tone, Abdominal reflex, Pressing power and Walking speed while group II(*Shodhana* therapy) was found statistically significant ($p<0.05$) in improving Pain, muscle power, Pressing power and walking speed only.

Effect of Treatment in various Lab parameters:

Groups	Parameters	Mean	SD	t-value	p-value	Statistical Significance
Group I	Hb (BT)	9.940	1.4455	-.173	>0.05	Not Significant
	Hb (AT)	10.040	1.2920			
	TLC (BT)	7250.00	1509.231	-1.153	>0.05	Not Significant
	TLC (AT)	8025.00	1396.275			
	Polymorphs (BT)	54.80	9.796	-0.421	>0.05	Not Significant
	Polymorphs (AT)	56.80	9.818			
	Lymphocytes (BT)	40.70	7.846	0.399	>0.05	Not Significant
	Lymphocytes (AT)	39.10	8.543			
	Eosinophils (BT)	4.50	3.100	0.303	>0.05	Not Significant
	Eosinophils (AT)	4.10	2.234			
	ESR (BT)	40.20	9.102	3.713	<0.05	Significant
	ESR (AT)	24.90	13.428			
	Glucose (BT)	95.70	20.624	.234	>0.05	Not Significant
	Glucose (AT)	94.40	27.520			
	Cholesterol (BT)	178.00	26.721	.926	>0.05	Not Significant
	Cholesterol (AT)	172.10	19.621			
Group II	Hb (BT)	9.960	.8579	1.229	>0.05	Not Significant
	Hb (AT)	9.540	1.1853			
	TC (BT)	8480.00	1338.988	.862	>0.05	Not Significant
	TC (AT)	8065.00	1506.661			
	Polymorphs (BT)	52.80	15.179	-1.556	>0.05	Not Significant
	Polymorphs (AT)	60.00	7.930			
	Lymphocytes (BT)	39.70	12.056	1.363	>0.05	Not Significant
	Lymphocytes (AT)	34.40	9.902			
	Eosinophils (BT)	6.10	6.523	-.222	>0.05	Not Significant

	Eosinophils (AT)	6.60	5.700			
	ESR (BT)	24.20	21.847	1.004	>0.05	Not Significant
	ESR (AT)	20.90	21.512			
	Glucose (BT)	102.40	24.459	.360	>0.05	Not Significant
	Glucose (AT)	100.00	13.752			
	Cholesterol (BT)	188.90	29.486	.470	>0.05	Not Significant
	Cholesterol (AT)	183.00	46.814			

As per various laboratory parameters statistically significant result was found only in ESR, in **Group I**.

In Group II effect of treatment on various laboratory parameters was found statistically not significant.

Statistical Significance for total score within the groups:

Parameter	Group I			Group II		
	Z	p-value	Statistical Significance	Z	p-value	Statistical Significance
Total Score (BT-AT)	- 2.67	<0.05	Significant	- 2.52	<0.05	Significant

Both the groups have shown statistically significant effect (<0.05), in over all effect of the therapy.

Study-II

Distribution of patients according to the severity of pain in walking:

Pain in walking	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	11	27.5%	25	62.5%	14	35%	28	70%
Mild	11	27.5%	10	25%	6	15%	7	17.5%
Moderate	12	30%	5	12.5%	12	30%	4	10%
Severe	6	15%	0	0%	8	20%	1	2.5%
Total	40	100%	40	100%	40	100%	40	100%

In Group-I, before treatment, 11(27.5%) patients were found having no pain and having mild pain during walking, 12(30%) were having moderate pain and 6(15%) patients were having severe pain. After completion of treatment it was found that, 25(62.5%) became normal followed by 10(25%) patients with mild pain and 5(12.5%) had moderate pain.

In Group-II before treatment, 14(35%) patients had no pain, moderate pain was found in 12(30%) patients, severe pain was noticed in 20.0% patients and 6 (15%) had mild pain. After treatment 28(70%) patients became normal, followed by mild pain in 7(17.5%) of patients, 4(10%) patients with moderate pain and 1(2.5%) patients with severe pain.

Distribution of patients according to the control over bladder:

Control over bladder	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	12	30.0%	27	67.5%	13	32.5%	28	70%
Loss of Control	2	5%	3	7.5%	4	10%	6	15%
Partial Control	9	22.5%	4	10%	5	12.5%	1	2.5%
With sensation but without control	10	2%	3	7.5%	6	15%	3	7.5%
Complete retention	7	17.5%	3	7.5%	12	30%	2	5%
Total	40	100%	40	100%	40	100%	40	100%

In group-I before treatment, in 12(30%) patients had no retention of urine, followed by partial control over bladder in 9(22.5%) patients, complete retention in 7(17.5%) patients and 2(5%) had loss of control on bladder function. After treatment 27(67.5%) patients became normal followed by 9(10%) patients had partial control over bladder and 7.5% patients had complete retention, Retention with sensation but without control and loss of control on bladder function each.

In Group II before treatment, 13 (32.5%) of patients were reported as normal, followed by 12 (30%) patients with complete retention, 6(15%) patients were reported with sensation but without control over bladder, 5(12.5%) patients had partial control over Bladder and 4(10%) had loss of control on bladder function. After treatment, 28(70%) became normal, 6(15%) patients with loss of control, 3(7.5%) patients, 2(5%) patients and 1(2.5%) patient were found with sensation but without control, complete retention and Partial control on bladder function respectively.

Distribution of patients according to the control over rectum:

Control over rectum	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	10	25%	23	57.5%	13	32.5%	33	82.5%
With sensation but without control	12	30%	10	25%	13	32.5%	4	10%
Complete retention	18	45%	7	17%	14	35%	3	7.5%
Total	40	100%	40	100%	40	100%	40	100%

In Group-I before treatment, 10(25%) patients had normal control over rectum, 18(45%) patients were recorded with complete retention and 12(30%) patients with sensation but without control over rectum function. After completion of the treatment, 23(57.5%) patients become normal followed by 10(25%) patients with sensation but without control over rectum and 7(17.5%) patients had complete retention.

In Group-II, before treatment 13(32.5%) patients had normal control over rectum as well as rectal function with sensation but without control each and 14(35%) patients were reported with complete retention. After treatment maximum i.e.33 (82.5%) patients became normal followed by 4(10%) patients with sensation but without control over rectum and only 3(7.5%) having complete retention.

Distribution of patients according to the sensory changes:

Sensory changes	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	2	5%	12	30%	4	10%	19	47.5%
Can not appreciate fully but almost normal sensation	5	12.5%	12	30%	6	15%	10	25%
Impaired Sensation	14	35%	10	25%	16	40%	6	15%
Partially appreciated	6	15%	3	7.5%	6	15%	3	7.5%
No sensation	13	32.5%	3	7.5%	8	20%	2	5%
Total	40	100%	40	100%	40	100%	40	100%

In group-I before treatment, 2(5%) patients were normal without any sensory change 14(35%) patients were reported with impaired sensation, 13(32.5%) patients had no sensation, 6(15%) patients with partially appreciated sensation and 5(12.5%) could not appreciate fully but almost normal sensation. After treatment 12(30%) patients were reported as normal and 12(30%) patients could not appreciate fully but almost normal, followed by 10(25%) patients with impaired sensation and 3(7.5%) partially appreciated and having no sensation.

In group II, before treatment, 4(10%) were normal, 16(40%) patients were reported with impaired sensation, 8(20%) patients with no sensation followed by 6(15%) patients with sensory changes who could be partially appreciated and could not appreciate fully but almost normal each. After treatment 19(47.5%) patients became normal, 10(25%) patients could not appreciate fully but almost normal and 6(15%) patients with impaired sensation,(7.5%) patients were partially appreciated and 2(5%) patients were having no sensation.

Distribution of patients according to the Muscle Power:

Muscle Power	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	1	2.5%	4	10%	0	0%	6	15%
Can attain and regain squatting position	1	2.5%	6	15%	0	0%	1	2.5%
Can climb upstairs without any help	0	0%	6	15%	1	2.5%	6	15%
Can walk without support and can climb upstairs by using the banister	7	17.5%	5	12.5%	3	7.5%	7	17.5%
Can walk with support	10	25%	7	17.5%	11	27.5%	14	35%
Patient can stand with support	7	17.5%	4	10%	10	25%	1	2.5%
Straight leg raising test about 10°	2	5%	1	2.5%	2	5%	0	0%
Semi-flexion in all joints	2	5%	0	0%	6	15%	1	2.5%
Can perform slight-flexion in all joints	1	2.5%	0	0%	1	2.5%	1	2.5%
Slight lateral and medial rotation of limb	2	5%	3	7.5%	1	2.5%	2	5%
Flickering movement	2	5%	1	2.5%	1	2.5%	0	0%
No movement	5	12.5%	3	7.5%	4	10%	1	2.5%
Total	40	100%	40	100%	40	100%	40	100%

In Group-I before treatment, 10(25%) patients were reported as they could walk with support, 7(17.5%) patients could walk without support and could climb upstairs by using the banister as well as Patient could stand with support each, 5(12.5%) patients had no movement, 2(5%) patients having straight raised leg test up to 10°, Semi-flexion in all joints, Slight lateral and medial rotation of limb, as well as No movement each and 1(2.5%) patients

can attain and regain squatting position as well as can perform slight-flexion in all joints each. After treatment it was reported that, 4(10%) patients became normal as well as Patient can stand with support each, 7(17.5%) patients were able to walk with support, 6(15%) patients Can attain and regain squatting position as well as can climb upstairs without help each, only 5(12.5%) patients Can walk without support and can climb upstairs by using the banister, 3(7.5%) patients had Slight lateral and medial rotation of limb as well as had no movement and only 1(2.5) patients had Straight leg raising test about 10° as well as Flickering movement each.

In Group-II before treatment, maximum patients 11(27.5%) patients could walk without support, 10(25%) patients could walk with support, 6(15) patients had semi flexion in all joints, 4(10%) patients had no movement and 3(7.5%) patients Can walk without support and can climb upstairs by using the banister, 1(2.5%)patients Could climb upstairs without any help, Could perform slight-flexion in all joints, had slight lateral and medial rotation of limb, as well as had Flickering movement each and no patient was reported as normal. After treatment, 6(15%) patients became normal as well as can climb upstairs without any help each, 14(35%) patients can walk with support, 7(17.5%) patients can walk without support and can climb upstairs by using the banister, 2(5%) had slight lateral and medial rotation of limb and 1(2.5%) patients could stand with support, semi-flexion in all joints, can perform slight-flexion in all joints as well as had no movement each.

Distribution of patients according to the Fasciculations:

Fasciculation	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	17	42.5%	29	72.5%	19	47.5%	29	72.5%
Mild	5	12.5%	4	10%	2	5%	6	15%
Moderate	15	37.5%	6	15%	15	37.5%	4	10%
Severe	3	7.5%	1	2.5%	4	10%	1	2.5%
Total	40	100%	40	100%	40	100%	40	100%

In Group-I, before treatment, 17(42.5%) patients were normal followed by 15(37.5%) patients, 5(12.5%) patients, 3(7.5%) patients had moderate, mild and severe fasciculation respectively. After treatment 29(72.5%) patients became normal followed by 6(15%) patients

with moderate fasciculation, 4(10%) patients with mild and only 1(2.5%) patient with severe fasciculation.

In Group II, before treatment, 19(47.5%) patients were reported as normal (without any fasciculation) followed by 15(37.5%) patients with moderate fasciculation, 4(10%) patients with severe fasciculation and 2(5%) patients with mild fasciculation. After treatment, 29(72.5%) patients became normal followed by 6(15%), 4(10%) and 1(2.5%) patients with mild, moderate and severe fasciculation respectively.

Distribution of patients according to the Electric chorea:

Electric chorea	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	16	40%	25	62.5%	17	42.5%	29	72.5%
Moderate	15	37.5%	11	27.5%	11	27.5%	7	17.5%
Severe	9	22.5%	4	10%	12	30%	4	10%
Total	40	100%	40	100%	40	100%	40	100%

In Group I, before treatment, 16(40%) patients were normal followed by 15(37.5%) patients were reported with moderate chorea and 9(22.5%) patients with severe chorea. After treatment 25(62.5%) patients became normal, 11(27.5%) had moderate chorea and 4(10%) had severe chorea.

In group II before treatment, 17(42.5%) patients were reported as normal, 11(27.5%) patients had moderate chorea and 12 (30%) had severe chorea. After treatment, 29 (72.5%) patients became normal, 7(17.5%) patient were reported with moderate chorea and 4(10%) patients had severe chorea.

Distribution of patients according to the clonus:

Clonus	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Absent	28	70%	31	77.5%	28	70%	30	75%
Present	12	30%	9	22.5%	12	30%	10	25%
Total	40	100%	40	100%	40	100%	40	100%

Before treatment, clonus was absent in 28(70%) patients. and it was present in 12(30%) patients in both the groups. After treatment in group I, 31(77.5%) patients were recorded with having clonus, and clonus was absent in 9(22.5%) patients. In Group II, 30(75%) patients had no clonus and 10(25%) patients with presence of clonus.

Distribution of patients according to the Tone:

Tone	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	2	5%	18	45%	3	7.5%	19	47.5%
Hypertonia	22	55%	16	40%	20	50%	10	25%
Hypotonia	16	40%	6	15%	17	42.5%	11	27.5%
Total	40	100%	40	100%	40	100%	40	100%

In group I, before treatment, hypertonia was present in 22(55%) patients and hypotonia in 16(40%) patients; after treatment it was observed that 18(45%) patients became normal followed by hypertonia in 16(40%) patients and hypotonia in 6(15%) patients.

In case of group II, hypertonia was recorded in 20(50%) patients & 17(42.5%) patients had hypotonia; after treatment 19(47.5%) patients were recorded as normal and 11(27.5%) patients had hypotonia and 10(25%) patients had hypertonia.

Distribution of patients according to the Knee Reflexes:

Knee Reflexes	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	5	12.5%	11	27.5%	1	2.5%	9	22.5%
Exaggerated	21	52.5%	20	50%	21	52.5%	17	42.5%
No response	14	35%	9	22.5%	18	45%	14	35%
Total	40	100%	40	100%	40	100%	40	100%

In Group I, before treatment, 5(12.5%) were recorded as normal, 21(52.5%) patients were recorded with exaggerated knee reflex and 14(35%) patients had no response; after completion of therapy, 11(27.5%) patients became normal, 20(50%) patients with exaggerated knee reflex and there were 9(22.5%) patients had no response.

In Group II before treatment, 21(52.5%) patients had exaggerated knee reflex and 18(45%) patients had no response. After treatment, 9(22.5%) patients became normal, 17(42.5%) patients had exaggerated knee reflex and 14(35%) patients had no response.

Distribution of patients according to the Ankle Reflexes:

Ankle Reflexes	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	2	5%	12	30%	1	2.5%	11	27.5%
Exaggerated	22	55%	17	42.5%	17	42.5%	15	37.5%
No response	16	40%	11	27.5%	22	55%	14	35%
Total	40	100%	40	100%	40	100%	40	100%

In Group I, before treatment, exaggerated ankle reflex was present in 22(55%) patients, and no response to ankle reflex was present in 16(40%) patients. After completion of the therapy, 12(30%) patients became normal, 17(42.5%) patients had exaggerated ankle reflex, and 11(27.5%) patient had no response.

In group II before treatment, 17(42.5%) patient had exaggerated knee reflex and 22(55%) patients had no response. After completion of the therapy, 11(27.5%) patients became normal, 15(37.5%) patients had exaggerated knee reflex & 14(35%) patients had no response.

Distribution of patients according to the plantar reflex:

Plantar reflex	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	1	2.5%	9	22.5%	4	10%	10	25%
Extensor planter reflex	25	62.5%	21	52.5%	16	40%	19	47.5%
No response	14	35%	10	25%	20	50%	11	27.5%
Total	40	100%	40	100%	40	100%	40	100%

In Group I, before treatment, 25(62.5%) patients had shown extensor planter response and 14(35%) patients had no response. After completion of the therapy extensor planter response was found normal in 9(22.5%) patients, extensor plantar reflex in 21(52.5%) patient and 14(35%) patients were having no response.

In Group II before treatment, extensor planter reflex was found in 16(40%) patients, and 20(50%) patients were reported to have no response to planter reflex. After completion of treatment 10(25%) patients were recorded with normal planter response while extensor response was found in 19(47.5%) patients and 11(27.5%) patients were found with no response.

Distribution of patients according to the Abdominal Reflexes:

Abdominal Reflexes	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Present	18	45%	26	65%	16	40%	23	57.5%
Absent	22	55%	14	35%	24	60%	17	42.5%
Total	40	100%	40	100%	40	100%	40	100%

In Group I, before treatment, abdominal reflex was absent 22(55%) of patients and present as normal in 18(45%) patients. After treatment it became present in 26(65%) patients and absent in 14(35%) patients.

In Group II before treatment, 16(40%) patients were reported with positive abdominal reflex as normal and 24(60%) were reported with absence of abdominal reflex. After completion of therapy, 23(57.5%) were reported with positive abdominal reflex abdominal reflex as normal and 17(42.5%) patients had shown absence of abdominal reflex.

Distribution of patients according to the Pressing power:

Pressing power	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%	No. of Patients	%	No. of Patients	%	No. of Patients	%
25 and above	2	5%	13	32.5%	1	2.5%	12	30%
20-25 Kg	2	5%	6	15%	1	2.5%	8	20%
10 to 20 Kg	6	15%	5	12.5%	3	7.5%	4	10%
Up to 10 Kg	30	75%	16	40%	35	87.5%	16	40%
Total	40	%	40	100%	40	100%	40	100%

In Group I, before treatment, 30(75%) patients were recorded having Pressing power up to 10 kg weight, 6(15%) patients were having 10-20 kg weight of Pressing power, and 2(5%) had pressing power of 20-25 kg as well as 25 kg and above weight each. After completion of treatment, 13(32.5%) patients became able to press 25 kg weight and above, 6(15%) had pressing power 20-25 kg weight, 5(12.5%) had 10-20 kg weight and 16(40%) had pressing power up to 10 kg only.

In group II before treatment, 35(87.5%) patients were had Pressing power up to 10 kg weight only, followed by 3(7.5%) patients with pressing power of 10-20 kg weight and 1(2.5%) patient could press 20-25 kg weight as well as 25kg weight and more of pressing power each. After treatment, 12(30%) patients were found having Pressing power of 25 kg weight and above, 8(20%) patients had pressing power of 20-25 kg. weight, 4(10%) patient could press 10-20 kg weight & 16(40%) patient had up to 10 kg weight of pressing power.

Distribution of patients in according to the Wasting of muscle:

Muscle wasting	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%	No. of Patients	%	No. of Patients	%	No. of Patients	%
Normal	37	92.5%	37	92.5%	38	95%	38	95%
Mild	2	5%	2	5%	2	5%	2	5%
Severe	1	2.5%	1	2.5%	0	0%	0	0%
Total	40	100%	40	100%	40	100%	40	100%

In Group I, before treatment, Wasting of muscles was absent in 37(92.5%) patients, 2(5%) patients were reported with mild and 1(2.5%) patients with severe wasting of muscles. After completion of treatment also, the result remain unchanged.

In case of group II before treatment, 38(95%) patients were reported with no wasting of muscles, 2(5%) patients were reported with mild wasting of muscles. After completion of treatment also, the result remain unchanged.

Distribution of patients according to the Walking Speed:

Walking Speed	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%	No. of Patients	%	No. of Patients	%	No. of Patients	%
Up to 20 second	0	0%	8	20%	0	0%	12	30%
21-30 second	2	5%	7	17.5%	0	0%	0	0%
31-40 second	3	7.5%	2	5%	0	0%	0	0%
41-50 second	2	5%	1	2.5%	4	10%	6	15%
51-60 second	1	2.5%	0	0%	0	0%	0	0%
60 second and above	32	80%	22	55%	36	90%	22	55%
Total	40	100%	40	100%	40	100%	40	100%

In Group I before treatment, 32(80%) patients took 60 second and more time to cover 20 meter of distance, followed by 3(7.5%) patients, who took 31-40 seconds and 2(5%) patients could cover in 41-50 seconds as well as 21-30 seconds each to cover the same

distance. After the treatment 8(20%) patient became able to cover the specified distance within 20 seconds, 22(55%) could cover in 60 seconds or more, 2(5%) patients covered within 31-40 seconds and 1(2.5%) covered within 41-50 seconds.

In group II before treatment, 36(90%) patients were able to cover the distance in 60 seconds & above and 4(10%) patients could cover within 41-50 seconds. After treatment 12 (30%) patients were able to cover the distance within 20 seconds, 6(15%) patients could cover the distance within 41-50 seconds and 22(55%) patients became able to cover the specified distance within 60 second and above.

Effect of treatment on Laboratory parameters:

Group	Lab. parameters	Mean	N	Std. Deviation	t-value	p-value	Statistical Significance
Group I	Hb (BT)	9.759	37	1.6539	-0.167	>0.05	Not Significant
	Hb (AT)	9.800	37	1.4424			
	TLC (BT)	8261.84	38	1398.524	-1.488	>0.05	Not Significant
	TLC (AT)	8600.26	38	804.264			
	Polymorphs (BT)	57.87	38	11.184	1.395	>0.05	Not Significant
	Polymorphs (AT)	55.13	38	12.338			
	Lymphocytes (BT)	39.29	38	12.098	-1.701	>0.05	Not Significant
	Lymphocytes (AT)	42.82	38	12.797			
	Eosinophils (BT)	2.58	38	3.185	0.985	>0.05	Not Significant
	Eosinophils (AT)	2.11	38	1.956			
	ESR (BT)	22.31	39	18.533	3.037	<0.05	Significant
	ESR (AT)	17.46	39	15.198			
	Glucose (BT)	114.00	39	45.941	0.598	>0.05	Not Significant
	Glucose (AT)	112.13	39	43.298			
	Cholesterol (BT)	174.92	39	34.560	0.355	>0.05	Not Significant
	Cholesterol (AT)	173.33	39	31.353			
Group II	Hb (BT)	9.643	40	1.4853	-1.537	>0.05	Not Significant
	Hb (AT)	10.003	40	1.3501			
	TLC (BT)	8606.25	40	1348.275	1.102	>0.05	Not Significant
	TLC (AT)	8360.00	40	1143.443			
	Polymorphs (BT)	54.20	40	13.249	0.265	>0.05	Not Significant
	Polymorphs (AT)	53.63	40	11.279			
	Lymphocytes (BT)	43.55	40	13.487	-0.537	>0.05	Not Significant
	Lymphocytes (AT)	44.68	40	11.360			

	Eosinophils (BT)	2.15	40	2.578	0.149	>0.05	Not Significant
	Eosinophils (AT)	2.08	40	2.080			
	ESR (BT)	27.43	40	22.916	0.620	>0.05	Not Significant
	ESR (AT)	25.35	40	24.915			
	Glucose (BT)	117.18	40	59.058	-0.888	>0.05	Not Significant
	Glucose (AT)	121.98	40	51.666			
	Cholesterol (BT)	177.70	40	41.910	0.872	>0.05	Not Significant
	Cholesterol (AT)	173.95	40	31.158			

In laboratory parameters statistically significant effect was found only in Group-I in case of ESR ($P < 0.05$).

Effect of treatment on various Symptoms:

Symptoms	Group I			Group II		
	Z	p-value	Statistical Significance	Z	p-value	Statistical Significance
Pain (BT-AT)	- 4.33	<0.05	Significant	- 3.76	<0.05	Significant
Control over bladder (BT-AT)	- 4.14	<0.05	Significant	- 4.13	<0.05	Significant
Control over Rectum (BT-AT)	- 3.99	<0.05	Significant	- 4.29	<0.05	Significant
Sensory changes (BT-AT)	- 4.88	<0.05	Significant	- 4.92	<0.05	Significant
Muscle power (BT-AT)	- 5.01	<0.05	Significant	- 4.97	<0.05	Significant
Fasciculations (BT-AT)	- 3.47	<0.05	Significant	- 3.53	<0.05	Significant
Electric chorea (BT-AT)	- 3.28	<0.05	Significant	- 3.54	<0.05	Significant
Clonus (BT-AT)	- 1.73	>0.05 Not Significant		- 1.41	>0.05 Not Significant	
Tone (BT-AT)	- 3.96	<0.05	Significant	- 4.12	<0.05	Significant
Knee Reflex (BT-AT)	- 3.05	<0.05	Significant	- 2.97	<0.05	Significant

Ankle Reflex (BT-AT)	- 3.42	<0.05	Significant	- 3.29	<0.05	Significant
Plantar Reflex (BT-AT)	- 3.03	<0.05	Significant	- 2.86	<0.05	Significant
Abdominal Reflex (BT-AT)	- 2.83	<0.05	Significant	- 2.65	<0.05	Significant
Pressing Power (BT-AT)	- 4.18	<0.05	Significant	- 4.26	<0.05	Significant
Wasting (BT-AT)	0.00	>0.05	Not Significant	0.00	>0.05	Not Significant
Walking Speed (BT-AT)	- 3.64	<0.05	Significant	- 3.62	<0.05	Significant

Effect of treatment in Group I and Group II was found statistically significant ($p<0.05$) in all the signs and symptoms except clonus and wasting of muscle.

Statistical Significance for Total Score with in the Groups:

Parameter	Group I			Group II		
	Z	p-value	Statistical Significance	Z	p-value	Statistical Significance
Total Score (BT-AT)	-5.30	<0.05	Significant	-5.44	<0.05	Significant

Both the groups have statistically significant effect (<0.05) on the total score of the disease.

Study-III

Distribution of patients according to the severity of pain in walking:

Pain	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	10	24.4%	25	61%	9	15.5%	35	60.3%
Mild	6	14.6%	13	31.7%	7	12.1%	15	25.9%
Moderate	16	39%	3	7.3%	27	46.6%	8	13.8%
Severe	9	22%	0	0%	15	25.9%	0	0%
Total	41	100%	41	100%	58	100%	58	100%

In Group-I, before treatment, moderate pain was present in 16(39%) patients followed by 9(22%) patients with severe pain and 6(14.6%) patients with mild pain. After treatment, 25(61%) patients became normal followed by 13(31.7%) patients with mild pain and 3(7.3%) with moderate pain.

In Group-II before treatment, moderate pain was found in 27(46.6%) patients followed by 15(25.9%) with severe pain and 7(12.1%) with mild pain. After treatment 35(60.3%) patients became normal, 15(25.9%) patients were found with mild pain and 8(13.8%) with moderate pain.

Distribution of patients according to the control over bladder:

Bladder control	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	6	14.6%	22	53.7%	6	10.3%	31	53.4%
Loss of Control	5	12.2%	6	14.6%	3	5.2%	14	24.1%
Partial Control	4	9.8%	5	12.2%	17	29.3%	9	15.5%
With sensation but without control	11	26.8%	2	4.9%	19	32.8%	2	3.4%
Complete retention	15	36.6%	6	14.6%	13	22.4%	2	3.4%
Total	41	100%	41	100%	58	100%	58	100%

In Group-I, before treatment, out of 41 patients, 6(14.6%) were found with normal control over bladder. 15 (36.6%) patients had complete retention of urine, 11(26.8%) were with sensation but without control, 5(12.2%) patients had loss of control over bladder and 4(9.8%) had partial control over bladder. After treatment, 22(53.7%) patients became normal, 6(14.6%) had loss of control as well as complete retention each, 5(12.2%) had partial control and 2(4.9%) were with sensation but without control over bladder.

In Group II before treatment, out of 58 patients, 6(10.3%) were found with normal control over bladder. 19(32.8%) patients were reported with sensation but without control, 17(29.3%) had partial control, 13(22.4%) had complete retention and 3(5.2%) had loss of control over bladder function. After treatment, 31(53.4%) became normal, 14(24.1%) had loss of control, 9(15.5%) had partial control and 2(3.4%) had with sensation but without control as well as complete retention each.

Distribution of patients according to the control over rectum:

Control over Rectum	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	8	19.5%	28	68.3%	16	27.6%	42	72.4%
With sensation but without control	17	41.5%	10	24.4%	21	36.2%	15	25.9%
Complete retention	16	39.0%	3	7.3%	21	36.2%	1	1.7%
Total	41	100%	41	100%	58	100%	58	100%

In Group-I, before treatment, out of 41 patients, 8(19.5%) were found with normal control over rectum. 17 (41.5%) patients were recorded with sensation but without control over rectum and 16(39%) had complete retention. After completion of the treatment, 28(68.3%) became normal, sensation but without control and 3(7.3%) had complete retention.

In Group-II before treatment, out of 58 patients, 16(27.6%) were found with normal control over bladder. 21(36.2%) patients were reported with sensation but without control over rectum as well as complete retention each. After treatment 42(72.4%) became normal, 15(25.9%) patients with sensation but without control and 1(1.7%) had complete retention.

Distribution of patients according to the sensory changes:

Sensory changes	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	4	9.8%	13	31.7%	2	3.4%	25	43.1%
Can not appreciate fully but almost normal	5	12.2%	17	41.5%	15	25.9%	21	36.2%
Impaired Sensation	13	31.7%	5	12.2%	16	27.6%	7	12.1%
Partially appreciated	12	29.3%	1	2.4%	18	31%	3	5.2%
No sensation	7	17.1%	5	12.2%	7	12.1%	2	3.4%
Total	41	100%	41	100%	58	100%	58	100%

In Group-I, before treatment, 4(9.8%) patients were noticed with normal sensation, 13(31.7%) patients were reported with impaired sensation, 12(29.3%) patients with partially appreciated sensation, 7(17.1%) patients with no sensation and 5(12.2%) could not appreciate fully but almost with normal sensation. After treatment, 13(31.7%) patients became normal, 17(41.5%) patients could not appreciate fully but almost normal followed by 5(12.2%) patients with impaired sensation as well as with no sensation each and 1(2.4%) with partially appreciated sensory changes.

In group II before treatment, 2(3.4%) patients presented with normal sensation, 18(31%) patientst almost normal, 16(27.6%) patients were having impaired sensation, 15(25.9%) could not appreciate fully but almost normal and 7(12.1%) patients were reported with no sensation. After treatment, 25(43.1%) patients became normal, 21(36.2%) patients could not appreciate fully but almost normal, 7(12.1%) patients reported with impaired sensation, 3(5.2%) patients were partially appreciated and 2(3.4%) patients were reported with no sensation.

Distribution of patients according to the Muscle power:

Muscle power	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	0	0%	3	7.3%	0	0%	3	5.2%
Can attain and regain squatting position	0	0%	3	7.3%	0	0%	11	19.0%
Can climb upstairs without any help	0	0%	7	17.1%	0	0%	6	10.3%
Can walk without support and can climb upstairs by using the banister	3	7.3%	6	14.6%	5	8.6%	13	22.4%
Can walk with support	9	22.0%	4	9.8%	17	29.3%	10	17.2%
Patient can stand with support	2	4.9%	6	14.6%	2	3.4%	2	3.4%
Straight leg raising test about 10	4	9.8%	2	4.9%	4	6.9%	3	5.2%
Semi-flexion in all joints	4	9.8%	1	2.4%	3	5.2%	0	0%
Can perform slight flexion in all joints	4	9.8%	3	7.3%	8	13.8%	6	10.3%
Slight lateral and medial rotation of limb	4	9.8%	1	2.4%	9	15.5%	2	3.4%
Flickering movement	4	9.8%	4	9.8%	2	3.4%	2	3.4%
No movement	7	17.1%	1	2.4%	8	13.8%	0	0%
Total	41	100%	41	100%	58	100%	58	100%

In Group-I, before treatment, 9(22.0%) patients were reported as they could walk with support followed by 7(17.1%) patients had no movement, 4(9.8%) patients had straight raised leg test positive up to 10° , semi flexion in all joints, slight lateral and medial rotation of limb as well as flickering movement each, 3(7.3%) could walk without support and could climb upstairs by using the banister and 2(4.9%) patients could stand with support. After treatment it was observed that 3(7.3%) patients became normal, could attain and regain squatting position as well as could perform slight flexion in all joints each, 7(17.1%) could climb upstairs without any help, 6(14.6%) could walk without support and could climb upstairs by using the banister as well as could walk with support, 4(9.8%) were able to walk with support as well as had flickering movement each and 1(2.4%) had slight lateral and medial rotation of limb as well as had no movement each.

In group-II before treatment, 17(29.3%) patients could walk with support, 9(15.5%) had slight lateral and medial rotation of limb, 8(13.8%) could perform slight flexion in all joints as well as had no movement each, 5(8.6%) could walk without support and could climb upstairs by using the banister, 4(6.9%) had straight leg raising test positive up to about 10^0 , 3(5.2%) had semi flexion in all joints, and 2(3.4%) patient could stand with support as well as had flickering movement each. After completion of treatment, 3(5.2%) became normal as well as had straight leg raising test about 10^0 each, 13(22.4%) could walk without support and could climb upstairs by using the banister, 11(19%) could attain and regain squatting position, 10(17.2%) could with support, 6(10.3%) could climb upstairs without any help as well as could perform slight flexion in all joints each and 2(3.4%) could stand with support, had slight lateral and medial rotation of the limb as well as had flickering movement each.

Distribution of patients according to the fasciculations:

Fasciculations	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	9	22%	20	48.8%	8	13.8%	21	36.2%
Mild	9	22%	16	39%	4	6.9%	29	50%
Moderate	13	31.7%	4	9.8%	26	44.8%	8	13.8%
Severe	10	24.4%	1	2.4%	20	34.5%	0	0%
Total	41	100%	41	100%	58	100%	58	100%

In Group-I, before treatment, 13(31.7%) patients had moderate fasciculation, 10(24.4%) patients had severe fasciculation and 9(22%) patients had mild fasciculation. After treatment, 20(48.8%) patients became normal, 16(39%) patients had mild fasciculation, 4(9.8%) patient with moderate fasciculation and 1(2.4%) had severe fasciculation.

In Group II before treatment, 26(44.8%) patients had moderate fasciculation, 20(34.5%) patient had severe fasciculation and 4(6.9%) had mild fasciculation. After treatment, 21(36.2%) became normal, 29(50 %) had mild fasciculation and 8(13.8%) had moderate fasciculation.

Distribution of patients according to the Electric chorea:

Electric chorea	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	15	36.6%	29	70.8%	19	32.8%	41	70.7%
Moderate	15	36.6%	11	26.8%	20	34.5%	16	27.6%
Severe	11	26.8%	1	2.4%	19	32.8%	1	1.7%
Total	41	100%	41	100%	58	100%	58	100%

In Group I, before treatment, 15(36.6%) patients were found normal, 15(36.6%) patients were reported with moderate chorea and 11(26.8%) patients with severe chorea. After treatment

29(70.8%) patients became normal, 11(26.8%) were reported with moderate chorea and 1(2.4%) had severe chorea.

In Group II before treatment, 19(32.8%) patients were found normal, 20(34.5%) patients were reported with moderate chorea and 19(32.8%) had severe chorea. After treatment, 41(70.7%) patients became normal, 16(27.6%) patient were reported with moderate chorea and 1(1.7%) patients with severe chorea.

Distribution of patients according to the clonus:

Clonus	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Absent	29	70.7%	32	78%	34	58.6%	41	70.7%
Present	12	29.3%	9	22%	24	41.4%	17	29.3%
Total	41	100%	41	100%	58	100%	58	100%

In Group I, before treatment, clonus was absent in 29(70.7%) and present in 12(29.3%) patients. After treatment, 32(78%) patients had absence of clonus and 9(22%) had presence of clonus.

In Group II before treatment, 34(58.6%) had absence of clonus and 24(41.4%) had presence of clonus. After treatment, 41(70.7%) had presence of clonus and 17(29.3%) had presence of clonus.

Distribution of patients according to the tone:

Tone	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	4	9.8%	13	31.7%	7	12.1%	24	41.4%
Hypertonia	19	46.3%	14	34.2%	28	48.3%	21	36.2%
Hypotonia	18	43.9%	14	34.1%	23	39.7%	13	22.4%
Total	41	100%	41	100%	58	100%	58	100%

In group I, before treatment, hypertonia was present in 19(46.3%) patients and hypotonia in 18(43.9%) patients. After treatment it was observed that 13(31.7%) patients became normal and 14(34.2%) patients had both hypertonia and hypotonia.

In case of Group II before treatment, hypotonia was recorded in 23(39.7%) and hypertonia was recorded in 28(48.3%) patients. After treatment, 24(41.4%) patients were

recorded with normal tone of muscles, 21(36.2%) with hypertonia and 13(22.4%) patients with hypotonia.

Distribution of patients according to the Knee Reflexes:

Knee Reflex	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	1	2.4%	6	14.6%	2	3.4%	13	22.4%
Exaggerated	20	48.8%	21	48.8%	32	55.2%	33	56.9%
No response	20	48.8%	14	31.6%	24	41.4%	12	20.7%
Total	41	100%	41	100%	58	100%	58	100%

In Group I before treatment, 20(48.8%) patients were reported with both exaggerated knee reflex no response each. After treatment, 6(14.6%) patients became normal, 21(48.8%) patients were found with exaggerated knee reflex and 14(31.6%) patients with no response.

In Group II before treatment, 32(55.2%) patients had exaggerated knee reflex followed by 41.4% patients with no response. After treatment, 13(22.4%) patients were reported as normal, 33(56.9%) patients were reported with exaggerated knee reflex and 12(20.7%) were reported with no response.

Distribution of patients according to the Ankle Reflexes:

Ankle Reflex	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	3	7.3%	7	17.1%	5	8.6%	17	29.3%
Exaggerated	18	43.9%	18	43.9%	28	48.3%	29	50%
No response	20	48.8%	16	39%	25	43.1%	12	20.7%
Total	41	100%	41	100%	58	100%	58	100%

In Group I, before treatment, 20(48.8%) patients were reported with exaggerated ankle reflex and 18(43.9%) patients were reported with no response. After completion of the therapy 7(17.1%) patient became normal, exaggerated ankle reflex remain unchanged in 18(43.9%) and 16(39.0%) patient had no response.

In Group II before treatment, 28(48.3%) patient had exaggerated knee reflex and 25(43.1%) patients had no response. After treatment 17(29.3%) patients were reported with normal ankle response, 29(50%) patients were reported with exaggerated knee reflex and 12(20.7%) patients were reported with no response.

Distribution of patients according to the Plantar reflexes:

Plantar Reflex	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	0	0%	8	19.5%	2	3.4%	16	27.6%
Extensor Plantar Reflex	21	51.2%	21	51.2%	37	63.8%	31	53.4%
No response	20	48.8%	12	29.3%	19	32.8%	11	19%
Total	41	100%	41	100%	58	100%	58	100%

In Group I, before treatment, 21(51.2%) patient had shown extensor planter reflex and 20(48.8%) patients had shown no response. After completion of the therapy it was noted that 8(19.5%) patients were reported as normal. 21(51.2%) patient had shown extensor planter reflex and 12(29.3%) had no response.

In group II before treatment, extensor planter reflex was present in 37(63.8%) patients followed by 19(32.8%) who had no response. After treatment, 16(27.6%) patients were recorded as normal, 53.4% patients were recorded with extensor response 11(19.0%) patients were recorded with no response.

Distribution of patients according to the Abdominal Reflexes:

Abdominal Reflex	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Present	18	43.9%	34	82.9%	20	34.5%	45	77.6%
Absent	23	56.1%	7	17.1%	38	65.5%	13	22.4%
Total	41	100%	41	100%	58	100%	58	100%

In Group I, before treatment, abdominal reflex was absent in 23(56.1%) patients and present in 18(43.9%) patients. After treatment, positive response to abdominal reflex was found in 34(82.9%) patients and negative response in 7(17.1%) patients.

In Group II, 20(34.5%) patients had shown positive response to abdominal reflex and 38(65.5%) had shown negative response. After completion of therapy 45(77.6%) patients had shown positive response and 13(22.4%) patients had shown negative response.

Distribution of patients according to the Pressing power:

Pressing Power	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
25 and above	0	0%	7	17.1%	0	0%	4	7%
20-25 Kg	1	2.4%	3	7.3%	0	0%	18	31%
10 to 20 Kg	3	7.4%	14	34.1%	3	5.2%	18	31%
Up to 10 Kg	37	90.2%	17	41.5%	55	94.8%	18	31%
Total	41	100%	41	100%	58	100%	58	100%

In Group I, before treatment, 37(90.2%) patients were recorded with having pressing capacity up to 10 kg weight followed by 3(7.4%) patients who were able to press 10-20 kg weight and 1(2.4%) had pressing capacity within 20-25 kg weight. After completion of treatment, 17(41.5%) patients had pressing capacity up to 10 kg weight, 14(34.1%) patients had pressing capacity of 10-20 kg weight, 7(17.1%) patients had pressing capacity of 25 kg and above weight and 3(7.3%) had pressing capacity of 20-25 kg weight.

In group II before treatment, 55(94.8%) patients had pressing capacity up to 10 kg weight and 3(5.2%) patients had pressing capacity of 10-20 kg weight. After treatment, 18(31%) patients had pressing power of 20-25 kg weight, 10-20 kg weight and up to 10 kg weight each and 4(7%) had pressing capacity of 25 kg weight or more.

Distribution of patients according to the Wasting of muscles:

Wasting of muscles	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Normal	41	100.0%	41	100.0%	58	100.0%	58	100.0%
Total	41	100.0%	41	100.0%	58	100.0%	58	100.0%

In this study wasting of muscle was found in neither of the groups.

Distribution of patients according to the Walking speed:

Walking Speed	Group I				Group II			
	BT		AT		BT		AT	
	No. of Patients	%						
Up to 20 second	0	0%	5	12.2%	0	0%	10	17.2%
21-30 second	0	0%	2	4.9%	0	0%	6	10.3%
31-40 second	1	2.4%	4	9.8%	0	0%	4	6.9%
41-50 second	1	2.4%	1	2.4%	0	0%	9	15.5%
51-60 second	1	2.4%	3	7.3%	2	3.4%	3	5.2%
60 second and above	38	92.8%	26	63.4%	56	96.6%	26	44.9%
Total	41	100%	41	100%	58	100%	58	100%

In Group I, before treatment, 38(92.8%) patients were able to cover the specified distance in 60 second and more and 1(2.4%) patients had taken 31-40 seconds, 41-50 seconds and 51-60 seconds each. After completion of the treatment, 26(63.4%) patients had taken 60 seconds or above, 5(12.2%) had taken up to 20 seconds, 4(9.8%) had taken 31-40 second, 3(7.3%) had taken 51-60 second, 2(4.9%) had taken 21-30second and 1(2.4%) had taken 41-50 second to cover the distance.

In Group II before treatment, 56(96.6%) patients had taken 60 second and above, and 2(3.4%) had taken 51-60 second to cover the distance. After treatment, 26(44.9%) patients were able to cover in 60 second & above, 10(17.2%) patients had taken up to 20 seconds, 9(15.5%) patients had taken 41-50 second, 6(10.3%) had taken 21-30 second, 4(6.9%) had taken 31-40 second and 3(5.2%) had taken 51-60 second to cover the distance.

Effect of Treatment on various laboratory parameters:

Group	Parameters	Mean	N	Std. Deviation	t-value	p-Value	Remarks
Group I	Hb (BT)	12.361	41	1.9269	-1.011	>0.05	Not
	Hb (AT)	12.607	41	1.4146			Significant
	TLC (BT)	8090.00	41	1163.862	-.351	>0.05	Not
	TLC (AT)	8165.85	41	899.058			Significant
	Polymorphs (BT)	52.71	41	12.079	1.557	>0.05	Not
	Polymorphs (AT)	49.66	41	9.964			Significant
	Lymphocytes (BT)	45.76	41	11.945	-1.591	>0.05	Not
	Lymphocytes (AT)	49.24	41	11.910			Significant
	Eosinophils (BT)	1.54	41	2.063	-.540	>0.05	Not
	Eosinophils (AT)	1.83	41	3.216			Significant
	ESR (BT)	22.51	41	16..744	2.138	<0.05	Significant
	ESR (AT)	18.10	41	11.736			
	Glucose (BT)	104.20	41	45.998	.499	>0.05	Not
	Glucose (AT)	100.71	41	43.911			Significant
	Cholesterol(BT)	188.98	40	45.047	1.640	>0.05	Not
	Cholesterol (AT)	177.55	40	37.589			Significant
	Serum. Protein(BT)	6.888	40	.7884	-.830	>0.05	Not
	Serum Protein (AT)	7.013	40	.6285			Significant
	Urea (BT)	19.44	39	6.373	4.602	<0.05	Significant
	Urea AT	15.33	39	4.349			
	Alkaline Phosphate (BT)	82.36	39	32.820	-1.597	>0.05	Not
	Alkaline Phosphate (AT)	90.51	39	48.207			Significant
	Acid Phosphate (BT)	3.01	20	1.743	-1.137	>0.05	Not
	Acid Phosphate (AT)	3.35	20	1.531			Significant
	SGOT (BT)	29.60	40	18.952	3.016	<0.05	Significant
	SGOT (AT)	24.35	40	13.677			
	SGPT (BT)	30.50	40	24.166	3.295	<0.05	Significant
	SGPT (AT)	22.18	40	16.803			
	Bilirubin (BT)	.796	16	.2472	1.246	>0.05	Not
	Bilirubin (AT)	.6975	16	.18749			Significant

Group II	Hb (BT)	12.220	58	1.6635	-.269	>0.05	Not
	Hb (AT)	12.273	58	1.5449			Significant
	TLC (BT)	8087.41	58	1017.071	.587	>0.05	Not
	TLC (AT)	8011.21	58	750.149			Significant
	Polymorphs (BT)	52.28	58	11.212	.815	>0.05	Not
	Polymorphs (AT)	50.76	58	10.834			Significant
	Lymphocytes (BT)	46.57	58	10.061	-.651	>0.05	Not
	Lymphocytes (AT)	47.81	58	11.103			Significant
	Eosinophils (BT)	1.60	58	2.420	.212	>0.05	Not
	Eosinophils (AT)	1.50	58	3.208			Significant
	ESR (BT)	22.93	58	19.125	1.478	>0.05	Not
	ESR (AT)	19.24	58	16.665			Significant
	Glucose (BT)	110.67	57	42.582	1.375	>0.05	Not
	Glucose (AT)	102.96	57	46.786			Significant
	Cholesterol (BT)	191.42	52	45.829	2.348	<0.05	Significant
	Cholesterol (AT)	179.83	52	38.774			
	Serum. Protein (BT)	6.843	53	.8965	-.569	>0.05	Not
	Serum Protein (AT)	6.909	53	.8258			Significant
	Urea (BT)	19.87	52	7.611	3.066	<0.05	Significant
	Urea (AT)	16.37	52	4.824			
	Alkaline Phosphate(BT)	77.86	49	24.701	-1.497	>0.05	Not
	Alkaline Phosphate (AT)	84.27	49	44.211			Significant
	Acid Phosphate (BT)	2.94	32	1.983	-2.704	<0.05	Significant
	Acid Phosphate (AT)	3.70	32	1.949			
	SGOT (BT)	26.88	50	15.986	.597	>0.05	Not
	SGOT (AT)	26.16	50	14.295			Significant
	SGPT (BT)	28.70	50	24.617	1.239	>0.05	Not
	SGPT (AT)	25.96	50	15.135			Significant
	Bilirubin (BT)	.883	23	.3419	2.281	<0.05	Significant
	Bilirubin (AT)	.7522	23	.34183			

In Group I, statistically significant effect ($p<0.05$) was found in improvement in ESR, biochemical tests like SGOT and SGPT. In Group II, statistically significant effect was found in Serum cholesterol level, Serum urea level, Acid phosphate level and Serum billirubin level.

Effect of treatment on various Symptoms:

Symptoms	Group I			Group II		
	Z	P-value	Statistical Significance	Z	p-value	Statistical Significance
Pain (BT-AT)	-	<0.05	Significant	-	<0.05	Significant
	4.64			5.96		
Control over bladder (BT-AT)	-	<0.05	Significant	-	<0.05	Significant
	4.61			6.01		
Control over Rectum (BT-AT)	-	<0.05	Significant	-	<0.05	Significant
	4.73			5.39		
Sensory changes (BT-AT)	-	<0.05	Significant	-	<0.05	Significant
	5.02			6.39		
Muscle power (BT-AT)	-	<0.05	Significant	-	<0.05	Significant
	5.40			6.54		
Fasciculations (BT-AT)	-	<0.05	Significant	-	<0.05	Significant
	4.40			5.94		
Electric chorea (BT-AT)	-	<0.05	Significant	-	<0.05	Significant
	4.02			5.34		
Clonus (BT-AT)	-	>0.05	Not Significant	-	<0.05	Significant
	1.73			2.33		
Tone (BT-AT)	-	<0.05	Significant	-	<0.05	Significant
	3.13			4.07		
Knee Reflex (BT-AT)	-	<0.05	Significant	-	<0.05	Significant
	3.21			3.91		
Ankle Reflex (BT-AT)	-	<0.05	Significant	-	<0.05	Significant
	2.83			4.20		
Plantar Reflex (BT-AT)	-	<0.05	Significant	-	<0.05	Significant
	3.35			4.03		
Abdominal Reflex (BT-AT)	-	<0.05	Significant	-	<0.05	Significant
	3.82			5.00		
Pressing Power (BT-AT)	-	<0.05	Significant	-	<0.05	Significant
	4.22			5.65		
Wasting (BT-AT)	0.00	>0.05	Not Significant	0.00	>0.05	Not Significant
Walking Speed (BT-AT)	-	<0.05	Significant	-	<0.05	Significant
	3.42			4.97		

In Group I, statistically significant effect ($p<0.05$) was found in all the symptoms except clonus and wasting of muscle. In Group II, statistically significant effect ($p<0.05$) was found in all the symptoms except wasting of muscle.

Statistical Significance for Total Score With in the groups:

Parameter	Group I			Group II		
	Z	p-value	Statistical Significance	Z	p-value	Statistical Significance
Total Score BT- Total Score AT	-5.513	<0.05	Significant	-6.625	<0.05	Significant

Both the groups have shown statistically significant effect (<0.05) in the total score of signs and symptoms.

Study-IV

Distribution of patients according to the severity of pain in walking:

Pain	Group							
	Group I				Group II			
	B.T		A.T.		B.T		A.T.	
	No. of Patients	%						
Normal	4	20%	4	20%	3	18.8%	3	18.8%
Mild	2	10%	15	75%	0	0%	12	75%
Moderate	13	65%	1	5%	8	50%	1	6.3%
Severe	1	5%	0	0%	5	31.2%	0	0%
Total	20	100%	20	100%	16	100%	16	100%

In group-I before treatment, 4 (20%) patients were normal, moderate pain was present in 13(65%) patients followed by 2(10%) patient was reported with mild pain and 1(5%) patients with severe pain. After treatment, 4(20%) remain normal. 15(75%) had mild pain and 1(5%) had moderate pain. In Group II before treatment, 3(18.8%) patients were normal, moderate pain was found in 8(50%) patients followed by severe pain in 5(31.2%) patients. After treatment, 3(18.8%) patients remain normal, 12(75%) patients had mild pain and 1(6.3%) patients had moderate pain.

Distribution of patients according to the control over bladder:

Control Over bladder	Group							
	Group I				Group II			
	B.T		A.T.		B.T		A.T.	
	No. of Patients	%						
Normal	11	55%	14	70%	9	56.3%	13	81.4%
Loss of Control	4	20%	3	15%	4	25%	3	18.8%
Partial Control	5	25%	3	15%	2	12.5%	0	0%
With sensation but without control	0	0%	0	0%	1	6.3%	0	0%
Total	20	100%	20	100%	16	100%	16	100%

In Group-I, before treatment, 5(25%) patients had partial control over bladder function and 4(20%) patients had loss of Control over bladder function. After treatment 14(70%) patients became normal followed by 3(15%) patient had partial control over bladder and loss of control over bladder function each.

In Group II before treatment, 4(25%) patients were reported with Loss of Control and 2(12.5%) patients were reported with partial control over Bladder function. After treatment, 13(81.4%) patients became normal and 3(18.8%) patients had loss of control over Bladder function.

Distribution of patients according to the control over rectum:

Control Over Rectum	Groups							
	Group I				Group II			
	No. of Patients	%						
Normal	12	60%	12	60%	10	62.5%	15	93.8%
With sensation but without control over rectum	8	40%	7	35%	6	37.5%	1	6.3%
Complete retention or complete incontinence	0	0%	1	5%	0	0%	0	0%
Total	20	100%	20	100%	16	100%	16	100%

In Group-I, before treatment, 12(60%) patients had normal control over rectum. 8(40%) patients were observed with sensation but without control over rectum. After completion of treatment, 7(35%) patients were reported with sensation but without control over rectum and 1(5%) patients had complete retention.

In Group-II before treatment, 6(37.5%) patients were found with sensation but without control over rectum and 10(62.5%) patients had normal control over rectal function. After treatment 15(93.8%) patients became normal and 1(6.3%) patients were observed with sensation but without control over rectum function.

Distribution of patients according to the sensory changes:

Sensory Changes	Groups							
	Group I				Group II			
	B.T		A.T.		B.T		A.T.	
	No. of Patients	%						
Normal	3	15%	4	20%	1	6.3%	4	25%
Can not appreciate fully but almost normal	3	15%	11	55%	2	12.5%	11	68.8%
Impaired Sensation	11	55%	3	15%	11	68.8%	1	6.3%
Partially appreciated	3	15%	2	10%	2	12.5%	0	0%
Total	20	100%	20	100%	16	100%	16	100%

In Group-I, before treatment, 11(55%) patients were reported with impaired sensation and 3(15%) patients were reported as they could not appreciate fully but almost normal as well as partially appreciated with the sensory change each. After treatment, 4(20%) patients were reported as normal, 11(55%) patients were reported as they could not appreciate fully but almost normal, 3(15%) patients with impaired sensation, and 2(10%) had partially appreciated sensory change.

In group II before treatment, 11(68.8%) patients were reported with impaired sensation and 2(12.5%) patients were reported as they could not appreciate fully but almost normal as well as could able to partially appreciate. After treatment 4(25%) patients became normal, 11(68.8%) patients were reported as they could not appreciate fully but almost normal and 1(6.3%) patients were reported with impaired sensation.

Distribution of patients in according to the Muscle Power:

Muscle Power	Group							
	Group I				Group II			
	B.T		A.T.		B.T		A.T.	
	No. of Patients	%						
Normal	0	0%	0	0%	0	0%	1	6.3%
Can climb upstairs without any help	0	0%	3	15%	0	0%	1	6.3%
Can walk without support and can climb upstairs by using the banister	0	0%	6	30%	0	0%	4	25%
Can walk with support	4	20%	7	35%	2	12.5%	6	37.5%
Patient can stand with support	7	35%	3	15%	8	50%	2	12.5%
Semi-flexion in all joints	2	10%	1	5%	0	0%	0	0%
Can perform slight-flexion in all joints (withdrawal on pain)	1	5%	0	0%	1	6.3%	2	12.5%
Slight lateral and medial rotation of limb	1	5%	0	0%	1	6.3%	0	0%
Flickering movement	3	15%	0	0%	2	12.5%	0	0%
No movement	2	10%	0	0%	2	12.5%	0	0%
Total	20	100%	20	100%	16	100%	16	100%

In Group-I, before treatment, 7 (35%) patients were reported as they could stand with support, followed by 4(20%) patients could walk with support, 3(15%) had flickering movement, 2(10%) patients having no movement and 1(5%) patients can perform slight flexion in all joints. After treatment it was reported that 3(15%) patients were able to stand with support & 7(35%) who could walk with support, 6(30%) patients could walk without support and could climb upstairs by using the banister, 3(15%) patient could stand with support as well as could climb upstairs without any help each and 1(5%) patient had semi flexion in all joints.

In group-II, before treatment 8(50%) could stand with support, 2(12.5%) patients who could walk with support, had flickering movement as well as no movement each, and 1(6.3%) patients could perform slight flexion in all joints as well as slight lateral and medial rotation of limb each. After treatment 6(37.5%) patients were able to walk with support, 4(25%) patients could walk without support and climb upstairs by using the banister followed by 1(6.3%) patients became normal as well as could climb upstairs without any help.

Distribution of patients in according to the Fasciculation:

Fasciculations	Group							
	Group I				Group II			
	BT		A.T.		BT		A.T.	
	No. of Patients	%						
Normal	0	0%	3	15%	1	6.3%	5	31.3%
Mild	8	40%	12	60%	7	43.8%	10	62.5%
Moderate	12	60%	5	25%	7	43.8%	1	6.3%
Severe	0	0%	0	0%	1	6.3%	0	0%
Total	20	100%	20	100%	16	100%	16	100%

In Group-I, before treatment, 12(60%) patients had moderate fasciculation and 8(40%) patients had mild fasciculation. After treatment 3(15%) patients became normal, 12(60%) patients had mild fasciculation and 5(25%) patient had moderate fasciculation.

In Group II, before treatment 7(43.8%) patients were reported with mild as well as moderate fasciculation and 1(6.3%) patient had severe fasciculation. After treatment 5(31.3%)

patients became normal, 10(62.5%) patients had mild fasciculation and 1(6.3%) patients had moderate fasciculation.

Distribution of patients according to the Electric chorea:

Electric Chorea	Group							
	Group I				Group II			
	B.T		A.T.		B.T		A.T.	
	No. of Patients	%						
Normal	0	0%	6	30%	0	0%	7	43.8%
Moderate	18	90%	13	65%	16	100%	9	56.4%
Severe	2	10%	1	5%	0	0%	0	0%
Total	20	100%	20	100%	16	100%	16	100%

In Group I, before treatment 18(90%) patients were reported with moderate chorea and 2(10%) patients with severe chorea. After treatment 6(30%) patients became normal, 13(65%) had moderate chorea and 1(5%) had severe chorea.

In group II before treatment, all the patients were reported with moderate chorea. After treatment, 7(43.8%) patients became normal and 9(56.4%) patient were reported with moderate chorea.

Distribution of patients according to the clonus:

Clonus	Group							
	Group I				Group II			
	BT		A.T.		BT		A.T.	
	No. of Patients	%						
Absent	8	40%	8	40%	8	50%	9	56.3%
Present	12	60%	12	60%	8	50%	7	43.8%
Total	20	100%	20	100%	16	100%	16	100%

In Group I, before treatment, 12(60%) patients were reported with presence of clonus and 8(40%) patients were reported with absence of clonus. After treatment also no changes were recorded.

In Group II before treatment, 8(50%) patients had absence of clonus and same numbers of patients were reported with presence of clonus. After treatment, 9(56.3%) patients had absence of clonus and 7(43.8%) had presence of clonus.

Distribution of patients according to the tone:

Tone	Groups							
	Group I				Group II			
	B.T		A.T.		B.T		A.T.	
	No. of Patients	%						
Normal	0	0%	5	25%	1	6.3%	5	31.3%
Hypertonia	7	35%	5	25%	7	43.8%	4	25%
Hypotonia	13	65%	10	50%	8	50%	7	43.8%
Total	20	100%	20	100%	16	100%	16	100%

In Group I, before treatment, hypotonia was present in 13(65%) patients and hypertonia was present in 7(35%) patients. After treatment it was observed that 5(25%) patients became normal and same numbers of patients were reported to have hypertonia. Hypotonia was present in 10(50%) patients.

In Group II before treatment, hypertonia was recorded in 8(50%) patients & hypotonia in 7(43.8%) patients. After treatment, 5(31.3%) patients became normal, 7(43.8%) patients had hypotonia and 4(25%) patients were recorded with hypertonia.

Distribution of patients according to the Knee reflex:

Knee Reflex	Groups							
	Group I				Group II			
	B.T		A.T.		B.T		A.T.	
	No. of Patients	%						
Normal	3	15%	7	35%	2	12.5%	8	50%
Exaggerated	12	60%	11	55%	10	62.5%	6	37.5%
No response	5	25%	2	10%	4	25%	2	12.5%
Total	20	100%	20	100%	16	100%	16	100%

In Group I, before treatment, 12(60%) patients were reported with exaggerated knee reflex and 5(25%) patients had shown no response. After treatment, 7(35%) patients became normal, 11(55%) patients had exaggerated knee reflex and 2(10%) patients had shown no response.

In Group II before treatment, 10(62.5%) patients had exaggerated knee reflex followed by 4(25%) patients had no response. After treatment 8(50%) patients became normal, 6(37.5%) patients had exaggerated knee reflex and 2(12.5%) patients had shown no response.

Distribution of patients according to the Ankle Reflex:

Ankle Reflex	Group							
	Group I				Group II			
	B.T		A.T.		B.T		A.T.	
	No. of Patients	%						
Normal	3	15%	9	45%	2	12.5%	9	56.3%
Exaggerated	12	60%	11	55%	10	62.5%	6	37.5%
No response	5	25%	0	0%	4	25%	1	6.3%
Total	20	100%	20	100%	16	100%	16	100%

In Group I, before treatment exaggerated ankle reflex was recorded in 12(60%) patients and no response to the ankle reflex was present in 5(25%) patients. After completion of the therapy 9(45%) patient became normal and 11(55%) patients had exaggerated ankle reflex.

In group II, before treatment 10(62.5%) of the patient had exaggerated ankle reflex and 4(25%) patients had no response. After completion of the therapy 9(56.3%) patients became normal, 6(37.5%) patients had exaggerated ankle reflex and 1(6.3%) had no response.

Distribution of patients according to the plantar reflex:

Planter Reflex	Group							
	Group I				Group II			
	B.T		A.T.		B.T		A.T.	
	No. of Patients	%						
Normal	9	45%	14	70%	7	43.8%	12	75%
Extensor	11	55%	6	30%	9	56.3%	4	25%
Total	20	100%	20	100%	16	100%	16	100%

In Group I, before treatment, 11(55%) patients had extensor planter reflex. After completion of the therapy extensor planter reflex was present in 6(30%) patient and 14(70%) patients were reported as normal.

In Group II before treatment, extensor planter was observed in 9(56.3%) patients. After treatment 12(75%) patient were recorded as normal and extensor planter reflex was present in 4(25%) patients.

Distribution of patients according to the abdominal reflex:

Abdominal Reflex	Groups							
	Group I				Group II			
	B.T		A.T.		B.T		A.T.	
	No. of Patients	%						
Present	19	95%	20	100%	9	56.3%	15	93.8%
Absent	1	5%	0	0%	7	43.8%	1	6.3%
Total	20	100%	20	100%	16	100%	16	100%

In Group I, before treatment, 19(95%) patients were showing presence of abdominal reflexes and 1(5%) of patients had absence of abdominal reflex. After treatment it was present in all the patients. In Group II before treatment, 9(56.3%) patients had presence of abdominal reflex and 7(43.8%) were reported with absence of abdominal reflex. After completion of therapy, 15(93.8%) had presence of abdominal reflex and 1(6.3%) patient had it absence of abdominal reflex.

Distribution of patients according to the Pressing power:

Pressing Power	Group							
	Group I				Group II			
	B.T		A.T.		B.T		A.T.	
	No. of Patients	%						
25 and above	0	0%	0	0%	1	6.3%	1	6.3%
20-25 Kg	3	15%	5	25%	2	12.5%	8	50%
10 to 20 Kg	4	20%	11	55%	5	31.3%	7	43.8%
up to 10 Kg	13	65%	4	20%	8	50%	0	0%
Total	20	100%	20	100%	16	100%	16	100%

In Group I, before treatment, 13(65%) patients were able to press up to 10 kg weight, 4(20%) patients who were able to press 10-20 kg weight and 3(15%) were able to press 20-25kg weight. After completion of treatment, 11(55%) patients were able to press 10-20 kg weight, 5(25%) could able to press 20-25kg weight and 4(20%) could press up to 10 kg weight only.

In group II before treatment, 8(50%) patients could press up to 10 kg weight, followed by 5(31.3%) patients who could press 10-20 kg weight, 2(12.5%) patient with pressing power of 20-25 kg and 1(6.3%) could press 25kg weight and above. After treatment, 8(50%) patients became able to press 20-25 kg weight, 7(43.8%) patient could press 10-20 kg weight and 1(6.3%) patient could press 25 kg & above.

Distribution of patients according to the Wasting of thigh muscles:

Wasting of thigh Muscles	Group							
	Group I				Group II			
	B.T		A.T.		B.T		A.T.	
	No. of Patients	%	No. of Patients	%	No. of Patients	%	No. of Patients	%
Normal	1	5%	1	5%	3	18.8%	3	18.8%
Difference of 2.5-3.5 cms	15	75%	16	80%	9	56.3%	12	75%
Difference of	4	20%	3	15%	4	25%	1	6.3%

3.5-4.5 cms								
Total	20	100%	20	100%	16	100%	16	100%

In Group I, before treatment, 1(5%) patient was recorded with no wasting of muscles or as normal. Wasting of muscles was present in 15(75%) patients with difference of 2.5-3.5 cms, 4(20%) patients were reported with wasting of muscles with difference of 3.5-4.5cms. After treatment 16(80%) had wasting of muscles between 2.5-3.5 cms and 3(15%) had wasting of muscles between 3.5-4.5 cms.

In group II before treatment, 3(18.8%) patients were recorded with no wasting of muscles or as normal. 9(56.3%) patients were reported with wasting of muscle with difference between 2.5-3.5 cms and 4(25%) patients with wasting of muscles with difference between 3.5-4.5 cms. After completion of therapy, 12(75%) patients had muscle wasting between 2.5-3.5 cms and 1(6.3%) was reported with muscle wasting with difference of 3.5-4.5 cms.

Distribution of patients according to the Wasting of Calf muscle:

Wasting of Calf muscle	Groups							
	Group I				Group II			
	B.T		A.T.		B.T		A.T.	
	No. of Patients	%						
Normal	1	5%	1	5%	4	25%	4	25%
Difference of 1.0-1.5 cms	17	85%	18	90%	8	50%	11	68.8%
Difference of 1.5-2.0 cms	2	10%	1	5%	4	25%	1	6.3%
Total	20	100%	20	100%	16	100%	16	100%

In Group I before treatment, 1(5.0%) patient was reported to has no wasting of calf muscles or as normal. 17(85%) patients had wasting of calf muscles with difference between 1.0-1.5 cms cms and 2(10%) patients were reported with wasting of calf muscles with difference between 1.5-2.0 cms. After treatment, 18(90%) patients had wasting of calf muscles between 1.0-1.5 cms.

In Group II, before treatment, 4(25.0%) patients were reported to have no wasting of calf muscles or as normal. 8(50%) patients were reported with wasting of calf muscles with difference between 1.0-1.5 cms and 4(25%) patients had wasting of muscles with difference

between 1.5-2.0 cms. After completion of therapy, 11(68.8%) patients had muscle wasting of calves between 1.0-1.5 cms. and 1(6.3%) had wasting of calf muscles between 1.5-2.0 cms.

Distribution of patients according to the Walking speed:

Walking Speed	Groups							
	Group I				Group II			
	No. of Patients	%						
up to 20 second	0	0%	1	5.0%	0	0%	4	25%
21-30 second	0	0%	4	20%	0	0%	2	12.5%
31-40 second	2	10%	4	20%	0	0%	3	18.8%
41-50 second	1	5%	5	25%	1	6.3%	3	18.8%
51-60 second	4	20%	1	5%	1	6.3%	2	12.5%
60 and above	13	65%	5	25%	14	87.5%	2	12.5%
Total	20	100%	20	100%	16	100%	16	100%

In Group I, before treatment, 13(65%) patients were taking 60 seconds and above time to cover distance of 20 meters followed by 4(20%) patients who had taken 51-60 seconds, 2(10%) patients could walk in 31-40 seconds and 1(5%) could walk in 41-50 seconds. After completion of treatment 5(25%) patients could cover the distance in 41-50 seconds and same number of patients became able to cover the same distance within 60 seconds or above, followed by 4(20%) patients could walk in 31-40 seconds and same number of patients in 21-30 seconds.

In Group II before treatment, 14(87.5%) patients were able to cover the specified distance in 60 seconds and above, followed by 1(6.3%) patient who could walk in 41-50 seconds and 1(6.3%) patient could cover in 51-60 seconds. After treatment 4(25%) patients had taken up to 20 seconds time to cover distance and 3(18.8%) patients were able to walk in 31-40 seconds and same numbers of patients covered in 41-50 seconds. 2(12.5%) could walk in 51-60 second and 2(12.5%) patients in 60 seconds and above .

Effect of treatment on various laboratory parameters:

Groups	Parameters	Mean	N	Std. Deviation	t-value	p-value	Statistical Significance	
<i>Group I</i>	TLC (BT)	10170.00	20	1096.454	1.696	>0.05	Not Significant	
	TLC (AT)	9795.00	20	980.051				
	Polymorphs(BT)	62.90	20	6.897				
	Polymorphs (AT)	64.10	20	6.958	-.792	>0.05	Not Significant	
	Lymphocytes(BT)	32.15	20	6.434				
	Lymphocytes(AT)	32.15	20	6.175		.000	>0.05	
	Monocytes (BT)	.40	20	.503				
	Monocytes(AT)	.70	20	.571	-1.831	>0.05		
	Eosinophils (BT)	4.50	20	6.109				
	E osinophils (AT)	3.05	20	4.872		3.067	<0.05	
	Hb (BT)	9.935	20	1.0113				
	Hb (AT)	10.788	20	1.1901	-3.623	<0.05		
	E.S.R (BT)	24.80	20	13.625				
	E.S.R (AT)	20.25	20	10.477		3.612	<0.05	
	Blood Sugar (BT)	94.97	20	10.408				
	Blood Sugar(AT)	92.90	20	6.935	1.190	>0.05		
<i>Group II</i>	TLC (BT)	10353.13	16	1134.529	5.689	<0.05	Significant	
	TLC (AT)	9315.63	16	934.651				
	Polymorphs (BT)	63.81	16	6.316				
	Polymorphs (AT)	65.75	16	4.987	-1.137	>0.05		
	Lymphocytes (BT)	32.44	16	6.066				
	Lymphocytes (AT)	32.19	16	4.679		.148	>0.05	
	Monocytes (BT)	.75	16	.683				
	Monocytes (AT)	.69	16	.479	.324	>0.05		
	E osinophils (BT)	3.00	16	4.195				
	E osinophils (AT)	1.69	16	.793		1.239	>0.05	
	Hb (BT)	9.288	16	.9695				
	Hb (AT)	10.144	16	.9647	-5.518	<0.05		
	E.S.R (BT)	33.56	16	19.575				
	E.S.R (AT)	24.31	16	11.050		3.091	<0.05	
	Blood Sugar (BT)	98.54	16	15.211	2.737	<0.05	Significant	
	Blood Sugar (AT)	91.95	16	8.052				

In Group I, statistically significant effect ($P>0.05$) was found in Eosinophil count, haemoglobin count and ESR count. In Group II, statistically significant effect ($P>0.05$) was found in TLC count, haemoglobin count, ESR count and Blood Sugar level.

Effect of treatment on various symptoms:

Symptoms	Group I			Group II		
	Z	p-value	Statistical Significance	Z	p-value	Statistical Significance
Pain (BT-AT)	-3.50	<0.05	Significant	-3.31	<0.05	Significant
Control over bladder (B.T. - .A.T)	-1.84	>0.05	Not Significant	-2.07	<0.05	Significant
Control Over Rectum (BT-AT)	-0.27	>0.05	Not Significant	-2.24	<0.05	Significant
Sensory Changes (BT-AT)	-3.05	<0.05	Significant	-3.69	<0.05	Significant
Muscle Power (BT-AT)	-3.85	<0.05	Significant	-3.43	<0.05	Significant
Fasciculations (BT-AT)	-2.89	<0.05	Significant	-3.21	<0.05	Significant
Electric Chorea (BT-AT)	-2.65	<0.05	Significant	-2.65	<0.05	Significant
Clonus (BT-AT)	0.00	>0.05	Not Significant	-0.58	>0.05	Not Significant
Tone (BT-AT)	-2.27	<0.05	Significant	-1.89	<0.05	Significant
Knee Reflex (BT-AT)	-2.65	<0.05	Significant	-2.83	<0.05	Significant
Ankle Reflex (BT-AT)	-3.05	<0.05	Significant	-3.16	<0.05	Significant
Planter Reflex (BT-AT)	-2.24	<0.05	Significant	-2.24	<0.05	Significant
Abdominal Reflex (BT-AT)	-1.00	>0.05	Not Significant	-2.45	<0.05	Significant
Pressing Power (BT-AT)	-3.05	<0.05	Significant	-3.28	<0.05	Significant
Wasting of Thigh muscle (BT-AT)	-1.00	>0.05	Not Significant	-1.73	>0.05	Not Significant
Wasting of Calf muscle (BT-AT)	-1.00	>0.05	Not Significant	-1.73	>0.05	Not Significant
Walking Speed (BT-AT)	-3.20	<0.05	Significant	-3.31	<0.05	Significant

In Group I, statistically significant effect ($P = <0.05$) was observed in all the symptoms except Control over bladder, Control over Rectum, Clonus, Abdominal Reflex, Wasting of Thigh muscle and Wasting of Calf muscle.

In Group II, statistically significant effect ($P = <0.05$) was observed in all the symptoms except Clonus, Wasting of Thigh muscle and Wasting of Calf muscle.

5. DISCUSSION

DISCUSSION

According to *Āyurveda*, *Pāṅgu* (Paraplegia) is one of the predominant *Vāta Vyādhi* among the 80 types of *Nānātmaja vāta Vyādhi*. In modern medical science, there is no suitable and satisfactory treatment for the management of *Pāṅgu* (Paraplegia). At the early stage of this disease, physiotherapy can provide some effective solution. Hence for the better and permanent solution for its management through Ayurvedic system, council has taken serious and humble efforts and completed 4 clinical studies with different classical preparations and procedures i.e. *Śamana* and *Śodhana* therapy. Each study has been carried out in two groups i.e. Group- I and Group- II.

The properties of the drugs used in all four studies in both *Śamana* and *Śodhana* groups are as follows-

Ekāṅgavīra Rasa is a known drug for *vāta vikāra* of single limb and it is having the *Rasāyana* property for the nourishing of the localized motor nerves which is affected by *vāta*. The drug *Eraṇḍa taila* is a best *vātānułomaka* and *Mrdu virecaka*. It is helpful for elimination of aggravated *vāta* through *Adhomārga*. *Gorocanādī gutīkā*, *Candraprabhā Vatī*, *Daśamūla Balā Kvātha* and *Aśvagandhā Kvātha* all are having *Rasāyana* and *Vātasāmaka* properties.

Tailas like *MahāMāṣa*, *Balā- Aśvagandhā-Lākṣādi* and *Daśamūla-Balā*, which are used for external application, all are having *Vāta śāmaka* and *Bṛmhāṇīya* properties.

In *Śodhana* groups; various types of *Vasti* were administered. These are the best and known therapy for all types of *Vāta Vyādhi*. *Virecana* is also act as the similar function like *Vastikarma*.

In these four studies, it was observed that more number of patients (25.44%) were belong to the age groups of 41-50 years followed by 23.21% patients in 51-60 years of age group. As per sex wise distribution, 69.20% patients were male and 30.80% were female. As per the chronicity of the disease, maximum patients (29.86%) were reported with the duration of less than 180 days.

Study-I

Total 20 patients were taken in this study, i.e 10 patients in each. In Group-I, *Ekāṅgavīra Rasa* alongwith *Eranda taila* were given orally and *Mahāmāṣa taila* was given for external application. In Group-II, *Śodhana* therapy i.e. *Virecana* with *Eranda taila* and *Yogavasti* (a combination of *Anuvāsana vasti* & *Nirūha vasti*) were given. The total duration of treatment in both the groups was 60 days.

After completion of the treatment it was observed that the effect of the therapies in both the groups was significant ($P<0.05$) in muscle power, pressing power and walking speed. In addition to this, the effect of therapy in Group-I was significant ($P<0.05$) in control over bladder, tone and abdominal reflex where as the therapy was found effective in pain in Group-II. On over all assessment, both the therapies were significant to manage the *Paṅgu*, but there is no major difference in the effect between two therapies.

After completion of the treatment it was observed that the effect of therapy on all laboratory parameters was not found significant in both the group. Only ESR value in group I was statistically significant (at $P<0.05$).

Study-II

Total 80 patients were equally distributed in two groups. In Group-I, *Gorocanādi* *gutikā* along with *Aśvagandhā Kvātha* was given orally and Balā- *Aśvagandhā-lākṣādi taila* was given for local application. In Group-II, *Śodhana* therapy i.e. *Virecana* and *Yogavasti* were given. The total duration of treatment on both the groups was 60 days.

After completion of treatment, it was observed that the effect of therapies on both the groups was found statistically significant ($P<0.05$) in all signs and symptoms except clonus and wasting of muscles.

In this study the effect of therapy on laboratory parameters in group I was significant (at $P<0.05$) for ESR value only but all other laboratory parameters were found not significant.

The over all effect of both therapies though found significant but there were no major differences in the effect of both the groups.

Study-III

Total 99 patients were treated in both the groups. In Group-I, 41 patients were treated with *Candraprabhā Vati* alongwith *Daśamūla Balā Kvātha* orally and *Daśamūla Balā taila* was given alternatively for *Mātrābasti* and also for external application. Alongwith these, physiotherapy also were given. In Group-II, 58 patients were administered with *Pañcakarma* therapy i.e. *Virecana* with *Erandā taila* and *Yogavasti*. The total duration of treatment was 45 days.

After completion of treatment, both the therapies were found statistically significant in all the signs and symptoms except wasting of muscles. In case of clonus, the effect of *Śamana* therapy was significant which was not significant on *Śodhana* therapy. In group I, significant effect ($P<0.05$) was found in laboratory parameters like ESR, SGOT & SGPT. But in group II, significant effect was found in Serum cholesterol, Serum urea, Acid phosphate and Serum billirubin level.

Study-IV

In this study a total 36 patients were taken under study. Group I, comprising of 20 patients, received *Candraprabhā Vati* along with *Daśamūla Kvātha* orally and *Daśamūla Balā taila* for local application as *Abhyanga* was used. This group was also treated with *Matravasti* and physiotherapy. Further in group II, 16 patients were administered with *Virecana* and *Yogavasti*. The total duration of treatment in this study was 45 days.

After completion of the treatment it was found that effect of both the therapies were statistically significant in all signs and symptoms except clonus and wasting of muscles. However in Group II the effect of therapy was significant in improving control over bladder, rectum and abdominal reflex which was found not significant in group I.

In group I, significant effect ($P<0.05$) of therapy was observed on Eosinophil, Haemoglobin and ESR. While in group II, significant effect noticed on TCL, Haemoglobin, ESR values and Blood sugar level.

6. CONCLUSION

CONCLUSION:

The word *Pāṅgu* is generally indicate limping gait. *Pitta*, *kapha*, *malas* and *dhātus* are said to be *Pāṅgu* while explaining the importance of *Vāta*. When we treat the *Pāṅgu* cases, then only we realize the seriousness of this disease properly. The lower part of the person,s body is paralyzed and cannot function wilfully. The patient is fully dependent on others for everything and become actual *Pāṅgu*. The studies reveal that there is good role for *Ayurveda* in the management of *Pāṅgu*.

1. There is marked relief in the symptoms as per assessment criteria in all the groups.
2. The drugs like *Bala*, *Asvagandha*, Milk, etc. are having the property of preventing the degeneration and strengthen the nervous system in *Pāṅgu*.
3. The better results were observed in *Pañcakarma* therapy, stresses the importance of *Srotoviśodhana* particularly in the management of *Pāṅgu*.
4. It was also noticed that there were no significant changes in blood sugar, serum cholesterol, serum protein, urea, serum alkaline phosphate, serum acid phosphate, SGOT, SGPT, bilirubin, etc. It denotes that Ayurvedic treatment is not harmful and not interfere the function of the system or organs.

7. APPENDAGES

Bibliography

- Anonymous (1978): The Ayurvedic Formulary of India, Part I, Published by Govt. of India, Ministry of Health & Family Planning, Deptt. of Health.
- Anonymous (1987): Pharmacopoeial Standards for Ayurvedic Formulations, Published by Central Council for Research in Ayurveda & Siddha, New Delhi.
- Anonymous: Oushadhi Pharmacopoeia, Pharmaceutical Corporation of Indian Medicine.
- Alam, M.M., Joy, S. and Ali S.V. (1991): Screening of *Sida cordifolia* Linn. *Sida rhomboidea* Linn. & *Triumfetta rotundifolia* Lam. for Anti-inflammatory and Antipyretic Activities, Indian Drugs 28, 397-400.
- Amon, H.P.T., Safayhi, H., Mach, T. & Satiaraj, T. (1993): Mechanism of anti-inflammatory Action of Curcumine and Boseswellic acids, J. Ethno Pharmacol.28, 113-119.
- Bansinath, M., Chandra Bose, A., Hema, S. and Guruswamy, M.N. (1982): Arch. Inter. Pharmacodyr. Therap.260 (1).
- Bhattacharya, C. (1981): Rheumatism, 16(3), p. 111-117.
- Bhavamishra (1969): Bhavaprakasha, Vidyotini Hindi Commentary by Sree Brahmashankara Shastri. The Chowkhamba Sanskrit Series Office, Gopalmandir Lane (P.O.), Chowkhamba, Post Box No.8, Varanasi, Vth Edition, page 393, Shloka 189-90.
- Bhavamishra (1969): Bhavaprakasha, Vidyotini Hindi Commentary by Sree BrahAankara Shastri, The Chowkhamba Sanskrit Series Office, Gopalmandir Lane (P.O.), Chowkhamba, Post Box No.8, Varanasi, Vth Edition, Page 393, Shloka 189-90.
- Bhavamishra (1969): Bhavaprakasha, Vidyotini Commentary, The Chowkhamba Sanskrit Series Office, Banaras, Page 366.
- Bhavamishra (1969): Bhavaprakasha, Vidyotini Commentary, The Chowkhamba Sanskrit Series Office, Banaras, Page 199.
- Borrie, P.C. (1971): Roxburg's Common Skin Diseases, 13th Edition, P. 6-7, Published by the English, Language Book Society and H.K. Lewis & Co. Ltd.
- Caraka (1970): Caraka Samhita, Chikitsa, 28, 144, (Editor, Gangasahaya Pandeya), Chowkhamba Sanskrit Series Office, Varanasi-1.
- Caraka (1970): Caraka Samhita 1st Edition, (Vidyotini Hindi Commentary by Kashinath S̄ishtri), Chowkhamba Sanskrit Series Office, Varanasi.
- Caraka (1969): Caraka Part II, Siddhi I. Shloka 32, pp.886.
- Caraka (1969): Caraka Samhita Part I, Vidyotini Hindi Commentary by Kashinadh Shastri, Ist Edition, Chowkhamba Sanskrit Series Office, Gopal Mandir Lane, P.O.Box-8, Varanasi, SootraSthana, Chapter-20, Shloka-11, page-269, ChikitsaSth̄na, Adhyaya-28, Shloka-11, page-662, ChikitsaSth̄na, Adhyaya-28, Shloka-72-74, page-703.
- Caraka (1969): Caraka Samhita Part II Chapter I, Shloka 5, Page-801.
- Caraka (1969): Caraka Samhita commentary Pt. Kasinatha Sastri, Edition I, The Chowkhamba Sanskrit Series Office, Varanasi, page: 174, 269, 713-14.
- Chaturvedi, G.N. and Singh R.H. (1965): Indian J. Med. Res. 53(1), p. 71-80.

- Chopra R.N., Nayar S.L. and Chopra I.C. (1956): Glososary of Indian Medicinal Plants, Published by Publication and Information Directorate, CSIR, New Delhi, 33.
- Cruicksjamk, R., Ouguid, T.P., Makmion, B.P. and Swain, R.H.A. (1975): Medical Microbiology, Vol.II, P. 202. Churchill Livingstone, London.
- Deva, Radhakantha Raja (1967): Sabdakalpadruma, Chowkhamba Sanskrit Series Office, Varanasi.
- Dutta, S. and Sanyal, S. (1978): Indian J. Exp. Biol., Vol. 16, p.166.
- Gosh M.N. (1971): Fundamentals of Experimental Pharmacology, P.41-42, Scientific Book Agency, Calcutta.
- Goodman L.S., Grewal M.S., Broown W.C. and Swinyard E.A. (1953): J. Pharmac. Exp. Therap. 108, P.168-176.
- Goodhart Robert, S and Shilo Mauriee, E. (1980): Modern nutrition in Health and Diseases, Edn. 6, K.M. Varghese Company, Bombay, page: 1265.
- Govt. of India (1976): Ayurvedic Formulary of India, Govt. of India Press, Faridabad, First edition, *Vijagutika prakarana*, page-146.
- Govindasa (1983): Baishyjratnavali, Published by Chowkhamba Sanskrit Samsthan, Varanasi.
- Hariford, D.J. & Smith, M.J.R. (1970): J. Pharma. Pharmacol. Vol. 22, P. 578.
- Houck, J.C. (1968): Biochem. Pharmacol. Suppl., Vol.I.
- Jain, P.K. and Panda, T.N. (1976): J. Res. Ind. Med. Yoga and Homoeo. 11 (2), P. 97-102.
- Joshi, C.G. and Magar, N.G. (1952): Jour. Sci. Indus. Research, Vol. II-B, P. 261.
- King, P.R.N. & King, E.J. (1954): J. Clin. Path., Vol.7, P.332.
- Kinnard, W.J. & Carr, C.J. (1957): Journal of Pharmacology and Experimental Therapeutics, Vol. 121, P. 354-361.
- Krishnan Vaidyan, K.V. and Gopala Pillai, S. (1969): Sahasrayoga, Sujanapriya commentary, Edn. 10, Vidyarambham Press & Book Depot Private Ltd., Alleppey, page: 315-316.
- Krishnan Vaidyan, K.V. (1969): Sahasrayoga, (Sujana Priya Malayalam Commentary), Xth Edition, Vidyarambham Press and Book Depot Pvt. Ltd., Mullakkal, Alleppey.
- Krishnan Vaidyan, K.V. (1969): Sahasrayoga, Sri Ramavilasam Press, Quilon (Kerala State), P.142.
- Lack, C.H. (1966): Proc. R. Soc. Med., Vol.59, P.875.
- Litchfield, J.T. and Wilcoxon, F. (1948): J. Pharmac. Exp., Therap., Vol. 96, P. 99-113.
- Luscombe, M. (1963): Nature, Vol. 197, P. 1010.
- Madhavakara (1955): Madhavanidana (Madhukosha Commentary byVijayarakshita and Srikantha Dutta), 5th Edition, Nirnayasagar Press, Bombay.
- Maffi, g (1958): Journ. Pharm.Pharmacology, Vol.II, P.129-139.
- Mehi, J.W. (1945): J. Diol. Chem., Vol. 157, P.173.
- Misra, B. (1961): Bhavaprakasha, Part II (Vidyotini Hindi Commentary by Pandit Sri Brahma Sankara Mishra), Chowkhamba Sanskrit Series Office, Varanasi.

- Morpurgo, C. (1971): Arzneim Forsch Drug Res., 21 (11), P. 1727-1734.
- Nadkarni, A.K. (1976): Indian Materia Medica, Vol.I, P.1171-1172, Popular Book Depot, Bombay.
- Naik, S.R. & Sheth, U.K. (1978): Indian J. Exp. Biol., Vol. 16, P. 1175.
- Patnaik, GK., Sabir, M. and Dhawan, B.N. (1979): Indian J. Exp. Biol., Vol. 17, P. 391-396.
- Parsolt, R.O., Bartir, A. and Jalfre, M. (1977): Arch. Int. Pharmacodyn., 229, P. 327-336.
- Petersdorf Adams, Braunwald and Isselbacher Martin, Wilson (1984): Harrison's Principles of Internal Medicine, McGraw Hill, New York.
- Plotnikoff, N.P. and Kastin, A.J. (1977): Advances in Bio-Chemical Psychopharmacology, Vol.17, P.92, Raven Press, New York.
- Prasad, B.N. (1962): Leprosy Rev., 33(3) P. 207-209.
- Pontis, V.V. & Grampurohit, N.D. (1994): Anti-inflammatory activity of the Creams Containing Turmeric and Red Sandal Wood, Indian Drugs 31, 117-118.
- Ramachandran Nair, P., Vijayan, N.P., Madhavikutty, P., Prabhakaran, V.A. and Indirakumari, S. (1985): Jour. Res. Ayu. Siddha, Vol.6, No.2, P. 121-131.
- Ramachandran Nair, P. (1984): Ancient Science of Life, Vol.IV, No.I, July 1984, pp. 20-26.
- Ramachandran Nair, P. (1986): Journal of Research in Indian Medicine, April-June, 1986.
- Ramachandran Nair, P. (1989): JRAS, Vol.X, No.1-2, pp. 30-40, 1989.
- Ramachandran Nair, P. (1992): JRAS, Vol.XIII, No.1-2, pp. 14-26, 1992.
- Ramachandran Nair, P. (1994): JRAS, Vol.XV, No.3-4, pp. 98-114, 1994.
- Ramachandran Nair, P. (1994): A comparative study of Sahacaradi taila, Nirgundi taila in Khanja and Pangu, Journal of Research and Education in Indian Medicine, Banaras.
- Ramachandran Nair, P., Vijayan, N.P., Bhagavathy Amma, K.C. and Madhavikutty, P. (1984): Action of Sahacaradiyoga in Khajuja and *Pa'gu'*, Ancient Science of Life IV (1) : 20-27.
- Ramachandran Nair, P., Vijayan, N.P., Bhagavathy Amma, K.C., Pillai, B.K.R, Ravindran, K.C. and Madhavikutty, P. (1987): Role of Sodhana treatment in Khanja and Pangu' Ayu' July: 13-36.
- Ramachandran Nair, P., Vijayan, N.P., Madhavikutty, P. and Indirakumari, S. (1986): A comparative study of Sahacaradi taila and Nirgundi taila in the management of Khanja (Monoplegia) and Pangu (Paraplegia). The Journal of Research and Education in Indian Medicine, 5 (2) : 13-16.
- Ravishankar, B. et.al. (1987): Analgesic, anti-inflammatory and Immunosuppressant effect of Strobilanthes heyneanus Nees, Stem. J.R.A.S., Vol.VIII, No.1 & 2, pp.53-63.
- Rathor, R.S. (1973): Nagarjun, 16(2), P. 25-29.
- Ravishankar, B. (1983): Abstracts of Papers. XVI Annual Conference of Indian Pharmacological Society, Indian Journal of Pharmacology, Vol. 16, No.1, P. 46-47.
- Raymond, D., Adams, M.D. & Maurice Victor, M.D. (1989): Principles of Neurology, McGraw Hill, New York.

- Reitman, S. & Prankel, S. (1957): Amer. J. Clin. Path., Vol.28, P. 56.
- Roe, J.J. & Kuether, C.A. (1943): J. Biol. Chem. Vol. 143, P. 399.
- Sharngadhara (1931): Sargadhara Samhita, IIInd Edition, Adhamall's Dipika and Kasiram's Gudhartha Dipika (Sanskrit Commentary by Pandit Parasurama Shastri, Vidyasagar), Nirnaya Sagar Press, Bombay.
- Sharma, Priyavrata (1969): Dravyaguna Vijnana, The Chowkhamba Vidya Bhavan, Varanasi, pp. 81-82, 69-71, 550-560.
- Satyavati, G. V., Raina, M.K. & Sharma, M. (1976): Medicinal Plants of India, Vol. I, P 314-315 and 524, Published by Indian Council of Medical Research, New Delhi.
- Sharma, A.K. and Singh, R.H. (1980): Bull. Med. Ethno. Bot. Res., 1(2), P. 262-271.
- Sheth, U.K., Dadkar, N.K. and Usha G Kamat (1972): Selected Topics in Experimental Pharmacology, P. 151.
- Shillito, E. (1970): Brit. J. Pharmac., 40, P.113-128.
- Singh, R.H. (1978): Rheumatism, Vol. 13, P.99.
- Singh, H. and Ghoosh, M.N. (1968): J. Pharm. Pharmacol., 21, P. 126.
- Shrivastava, S.C. and Sisodia, C.S. (1970): Indian Vet. J., 47 (2), P. 170-175.
- Stone, R.L. and Paget, C.J. (1971): Screening Methods in Pharmacology (Ed: R.A.Turner, and Peter), N. Academic Press, New York, Vol.II, P. 145-163.
- Sushruta (1966): Susrutha Samhit; (Commentary by Kaviraj Ambikadutta Shastri), Chowkhamba Sanskrit Series Office, Varanasi.
- Sushruta (1966): Sushruta Samhita Nidana 1: 77 (Ayurveda tatva sandeepika commentary), Chowkhamba Sanskrit Series Office, Banaras.
- Sushruta (1966): Sushruta Samhita, Ayurveda Tatva Sandeepika commentary: Kaviraj Dr. Ambikadatta Shashtri. Edition 11, Chowkhamba Sanskrit Series Office, Varanasi.
- Swinyard, E.A., Brown, W.C. and Goodman, L.S. (1952): J. Pharmac. Exp. Therapy, 106, P. 319-330.
- Tanaka, K. & Lisuka, Y. (1968): Biochem. Pharmacol. Vol. 17, P.2023.
- Taranath Tharka Vachaspati (1970): Vachaspathyam, Chowkhamba Sanskrit Series Office, P.B.8, Varanasi.
- Tripathi, C.P. (1981): Sachitra Ayurved, 33(7), P. 498-499.
- Vaghbhata (1956): Ashtangahrdaya. Bhageerathi Pt. Sri. Taradatta panta Ayurvedacharya, Chowkhamba Sanskrit Series Office, Varanasi, Page: 5, 23, 85, 86, 226, 236, 352, 355, 567.
- Vaghbhata (1957): Ashtangahrdaya.Sutra 6: 167, 197 and Chikitsa 22: 23. Chowkhamba Sansrit Series Office, Banaras.
- Vaghbhata (1957): Ashtangahrdaya.Nidana 15: 45 Chowkhamba Sanskrit Series Office, Banaras.
- Vaghbhata (1957): Ashtangahrdaya. Sutra 12 : 9 Chowkhamba Sanskrit Series Office, Banaras.

Vagbhata (1957): Ashtangahrdaya.Nidanam 15 : 5, 6. Chowkhamba Sanskrit Series Office, Banaras.

Vagbhata (1957): Ashtangahrdaya.Shareera 3 : 68, 69, 4 : 5, 19. Chowkhamba Sanskrit Series Office Banaras.

Vagbhata (1957): Ashtangahrdaya.Sutra 15 : 5, Chowkhamba Sanskrit Series Office, Banaras.

Vagbhata (1957): Ashtangahrdaya.Sutra 19 : 64. Chowkhamba Sanskrit Series Office, Banaras.

Vagbhata (1956): Ashtangahrdaya., Moolam, Chowkhamba Sanskrit Series Office, P.O.Box-8, Varanasi, NidanaSthana, Chapter-1, Shloka 14-15, page 271.

Vagbhata (1956): Ashtangahrdaya., NidanaSthana, Chapter-15, Shloka-5-45, page 355.

Vagbhata (1956): Ashtangahrdaya., Moolam, SutraSthana, Adhyaya-12, Shloka-9, page 86.

Vagbhata (1956): SareeraSthanam, Adhyaya-4, Shloka-19-20, page 236.

Vagbhata (1956): SutraSthana, Adhyaya-17, Shloka-29-30, page 122.

Vagbhata (1956): SootraSthana, Adhyaya-19, Shloka-1, page 129.

Vagbhata (1956): SootraSthana, Adhyaya-19, Shloka-85-86, page 138.

Vallame, S.P. and Gupta, K.C. (1981): Indian J. Pharmac., 13 (2) P. 203-204.

Vaidya, K.M. (1936): The Ashtangahrdaya Kosha. The Mangalodayam Press, Trichur, pp. 601.

Vangasena (12th Century): Vangasena (Vatavyadhi Adhikara), 126.

Velayudha Kurup (1968): Sahasrayogam, Vaidhapriya Malayalam Commentary, Sri Rama Vilasam Press, Kollam, Kerala, VIIth Edition, Tailaprapakaranam 67, page 320-321.

Vohra, S.B. and Khan, M.S.Y. (1981): Indian Drugs Pharm. Ind., 16 (1), P. 39-40.

Winter, C.A. and Porter, C.C. (1957): Jour. Amar. Pharm. Assn., Vol. 46.

Winter, C.A., Riseley, E.A. and Nyss, G.W. (1962): Proc. Soc. Expt. Biol. Med. III, P. 544-547.

Witkin, L.B., Huebner, C.P., Galdi, F., O'Keefe, E., Spitaletta, F. and Plummer, A.J. (1961): J. Pharmac. Expt. Therap., P. 133,400.

CENTRAL COUNCIL FOR RESEARCH IN AYURVEDA AND SIDDHA, NEW DELHI

CLINICAL EVALUATION OF THE EFFECT OF COMPOUND/ HERBOMINERAL COMPOUND DRUGS/PANCHAKARMA THERAPY IN THE MANAGEMENT OF

Paingu (PARAPLEGIA)

PROFORMA

FORM, I - SCREENING OF THE CASES

Name of the Patient:

Age: Sex:

Address:

1. Centre _____

2. Code No.

3. Patient No.

4. Group No.

First Second

CRITERIA OF SELECTION

5. Age between 12-70 years
either sex

Yes No

6. Chronicity < 2 years

Yes No

7. Inability to raise the lower limbs

Yes No

8. Inability to walk

Yes No

9. Presenting signs of paralysis

Yes No

10. Hypotonia/Hypertonia of lower limbs Yes 1 No 2

11. Exaggeration/Absence of knee Yes 1 No 2

12. Impaired sensation Yes 1 No 2

13. Retention/Incontinence of urine and/or faeces Yes 1 No. 2

CRITERIA FOR EXCLUSION

14. Age below 12 and above 70 years Yes 1 No 2

15. Chronicity beyond 2 years Yes 1 No 2

16. Tuberculosis of the hip joint with complications of high fever, etc. Yes 1 No 2

17. Pelvic pathology, if any Yes 1 No 2

18. Any deformity/neoplasm of the spine (Lumbo sacral region) Yes 1 No 2

19. Post fracture sequela Yes 1 No 2

20. Any systemic disease such as Hypertension, Peptic Ulcer, etc. Yes 1 No. 2

21. History of Liver diseases in recent past Yes 1 No. 2

22. History of Renal diseases Yes 1 No 2

23. Serious complications associated with any other systemic disease Yes 1 No 2

24. Bed sore Yes 1 No 2

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**CLINICAL EVALUATION OF COMPOUND/HERBOMINERAL COMPOUND
DRUGS/PANCHAKARMA THERAPY IN THE MANAGEMENT OF *Pangu*
(PARAPLEGIA)**

PART II, ADMISSION FORM

Name of the patient:

Address

Date of admission Date of discharge

1. Centre

2. Code No.
(of clinical trial)

3. Patient No.

4. Group 1 2

5. Age of patient in years

6. Sex M F

7. Educational status: Illiterate 1 Read & write 2

Primary School 3 Middle School 4

High School 5 College 6

Others specify 7 Information 8
not available

8. Occupation: Desk work 1 Field work 2

Field work with physical labour 3 Field work intellectual 4

Indicate nature of work 5

Total Income of the family in Rupees _____

9. Total family members

10. Income per capita per month in Rupees

11. Marital status Married 1 Unmarried 2

Widow 3 Divorced 4

Widower 5

CHIEF COMPLAINTS WITH DURATION (in days)

12. Weakness/Paralysis of both the lower limb Present Absent Duration

13. Inability to walk Present Absent Duration

14. Pain Present Absent Duration

15. Numbness Present Absent Duration

16. Pins and needle sensation Present Absent Duration
17. Burning sensation Present Absent Duration
18. Impaired sensation Present Absent Duration
19. Involuntary movements Present Absent Duration
20. Muscle wasting Present Absent Duration
21. Retention/Incontinence of urine Present Absent Duration
22. Retention/Incontinence of fasces Present Absent Duration

HISTORY OF PRESENT ILLNESS

23. Onset of disease Acute 1 Insidious 2
24. Duration of disease (in days):
25. Treatment given so far:
- | | |
|--------------------|----------------------------|
| Ayurvedic medicine | <input type="checkbox"/> 1 |
| Modern medicine | <input type="checkbox"/> 2 |
| Homoeopathy | <input type="checkbox"/> 4 |
| Any other | <input type="checkbox"/> 6 |
| Unani | <input type="checkbox"/> 3 |
| Naturopathy | <input type="checkbox"/> 5 |
| Specify _____ | |

Spell out the medicine given and results obtained _____

26. Factors aggravating the disease/Chief complaints: _____

27. Factors relieving main complaints: _____

28. History of past illness, having relation with present illness:

Yes No

 1 2

If yes, specify _____

FAMILY HISTORY

 1 2

29. Hypertension Yes No

30. Diabetes Mellitus Yes 1 No 2

31. Bronchial Asthma Yes 1 No 2

32. Mental disease Yes 1 No 2

33. Cancer Yes 1 No 2

34. Cardio vascular disease Yes 1 No 2

35. Tuberculosis Yes 1 No 2

36. Others (specify) _____

PERSONAL HISTORY

37. Diet Veg. 1 Non-veg. 2 Lacto-ova veg. 3

38. Sleep Good 1 Disturbed 2 Insomnia 3

39. Emotional stress Yes 1 No 2

If yes, specify _____

40. Bowel habit Regular 1 Constipation 2

Hard stool 3 Loose stool 4

Scyballeus 5

41. Deha prakrti V_ita 1 Pitta 2 Kapha 3

V_ita-Kaphaja 4 V_ita-Pittaja 5

Pitta-Kaphaja 6

42. Manas Prakrti Satwa 1 Rajas 2 Tamas 3

Satwa-rajas 4 Raja-tamas 5

Satva-tamas 6

PHYSICAL EXAMINATION

43. Built Lean 1 Medium 2 Heavy 3

44. Gait Normal 4 Abnormal 6

If abnormal, specify _____

45. Body weight (Kg.)

46. Body temperature

47. Pulse

48. Respiration

49. Blood pressure (Systolic)

50. Blood pressure (Diastolic)

51. Cynosis Present 1 Absent 2

52. Anaemia Present 1 Absent 2

53. Jaundice Present 1 Absent 2

54. Pigmentation Present 1 Absent 2

55. Clubbing of fingers Present 1 Absent 2

56. Deformities Present 1 Absent 2

If any, specify _____

57. Lymphadenopathy Present 1 Absent 2

If any, specify _____

SYSTEMIC EXAMINATION

58. C.V.S. with chest Normal 1 Abnormal 2

If abnormal, specify abnormalities _____

59. C.N.S. Normal 1 Abnormal 2

If abnormal, specify abnormalities _____

60. Respiratory system Normal 1 Abnormal 2

If abnormal, specify abnormalities _____

61. Digestive system Normal 1 Abnormal 2

If abnormal, specify abnormalities _____

62. Uro-Genital system Normal 1 Abnormal 2

If abnormal, specify abnormalities _____

LOCAL EXAMINATION

63. Muscular wasting Present 1 Absent 2

64. Swelling Present 1 Absent 2

65. Discolouration Present 1 Absent 2

66. Fasciculation Present 1 Absent 2

67. Foot drop Present 1 Absent 2

68. Muscular spasm Present 1 Absent 2

**SAMPRAPTI (PATHOGENESIS) OF THE DISEASE ACCORDING TO
AYURVEDIC CONCEPT:**

69. Anubandhya DoÀa V̄ita 1 Pitta 2 Kapha 3

70. Anubandha dosa V̄ita 1 Pitta 2 Kapha 3

71. DfÀya Rasa 1 Rakta 2 MjÆsa 3

Meda 4 Asthi 5 Majji 6

Shukra 7

72. Stages of disease (Roga Kriya kala)

Sanchaya 1 Prakopa 2 Prasara 3

Sthana, Sanshraya 4 Vyakti 5 Bheda 6

SHROTAS PARIKSHA

Medovaha-Shrotas

- | | | |
|--|--------------------------------|-------------------------------|
| 73. Maladhykya (Excess of excreta) | Yes <input type="checkbox"/> 1 | No <input type="checkbox"/> 2 |
| 74. Hastapada daha
(Burning sensation in the sole & palm) | Yes <input type="checkbox"/> 1 | No <input type="checkbox"/> 2 |
| 75. Hastapada suptata
(Numbness of the limbs) | Yes <input type="checkbox"/> 1 | No <input type="checkbox"/> 2 |
| 76. Tandra (Drowsiness) | Yes <input type="checkbox"/> 1 | No <input type="checkbox"/> 2 |
| 77. Dehacikkanata
(Greasiness of the skin) | Yes <input type="checkbox"/> 1 | No <input type="checkbox"/> 2 |
| 78. Alasya (Lethargy) | Yes <input type="checkbox"/> 1 | No <input type="checkbox"/> 2 |

Mootra Vaha Shrotas

- | | | |
|---|--------------------------------|-------------------------------|
| 79. Bahumootrata (Polyuria) | Yes <input type="checkbox"/> 1 | No <input type="checkbox"/> 2 |
| 80. Atibandata (Scanty urination) | Yes <input type="checkbox"/> 1 | No <input type="checkbox"/> 2 |
| 81. Prakupa (Defective urination/
Difficulty in micturition) | Yes <input type="checkbox"/> 1 | No <input type="checkbox"/> 2 |
| 82. Alpa-alpa
(Scanty urination) | Yes <input type="checkbox"/> 1 | No <input type="checkbox"/> 2 |
| 83. Abhikshna
(Constant/repeated urination) | Yes <input type="checkbox"/> 1 | No <input type="checkbox"/> 2 |
| 84. Bahul mootrata
(Urine with prostatic secretion) | Yes <input type="checkbox"/> 1 | No <input type="checkbox"/> 2 |
| 85. Sashoola mootrata (Painful micturition) | <input type="checkbox"/> 7 | |

Pureesha Vaha Shrotas

- | | | |
|---|--------------------------------|-------------------------------|
| 86. Alpa-alpa Pureesha
(Scanty defecation) | Yes <input type="checkbox"/> 1 | No <input type="checkbox"/> 2 |
|---|--------------------------------|-------------------------------|

87. Sashoola pureesha
(Painful defecation) Yes 1 No 2

88. Atidrava pureesha
(Diarrhea) Yes 1 No 2

89. Atigrathita pureesha
(ScyBal;) Yes 1 No 2

Other shrotases involved if any: _____

90. Provisional Diagnosis:

91. Final Diagnosis:

92. Medical Management:

93. Principal drug therapy:

Dose:

Vehicle:

Diet:

94. Summary of findings:

95. Result:

Good response

 1

Fair response

 2

Poor response

 3

No response

 4

Drop out

 5

LEMA

 6

Death

 7

Date:

Signature of Investigator

Counter signature of Head of Dept.

CENTRAL COUNCIL FOR RESEARCH IN AYURVEDA & SIDDHA, NEW DELHI

**CLINICAL EVALUATION OF THE EFFECT OF COMPOUND HERBOMINERAL
COMPOUND DRUGS/PANCHAKARMA THERAPY IN THE MANAGEMENT OF**

Pangu (PARAPLEGIA)

PART III, INVESTIGATION RECORD

Name of the patient:

Age:

Sex:

Address:

1. Centre

--	--

2. Code No.

(of clinical trial)

--	--

3. Patient No.

--	--	--	--

4. Group No.

--

	At the time of admission	After 15 days	30 days	45 days	60 days	75 days	90 days
1	2	3	4	5	6	7	8
Urine:							
5. Sugar	1	2	1	2	1	2	1
6. Albumin	1	2	1	2	1	2	1
7. Bile salt	1	2	1	2	1	2	1
8. Bile pigment	1	2	1	2	1	2	1
9. Microscopy	1	2	1	2	1	2	1
Stool:							
10. Ova	1	2	1	2	1	2	1
11. Cyst	1	2	1	2	1	2	1

Present = 1 Absent = 2

HAEMATOLOGICAL INVESTIGATIONS

12. Hb.

--	--

13. T.L.C.

--	--	--	--

14. D.L.C. P E L M B

15. E.S.R.

--	--	--

BIOCHEMICAL:

16. Protein total

--	--

17. Albumin Globulin ratio

--	--

18. Blood Glucose

--	--	--

19. Blood urea

--	--

20. Serum Bilirubin

--	--

21. Serum Cholesterol

--	--	--

22. Serum Alkaline Phosphatase

--	--

23. Serum Acid Phosphatase

--	--

24. S G O T

--	--

25. S G P T

--	--

26. V.D.R.L.

Pos.	Neg.
------	------

RADIOLOGICAL INVESTIGATIONS

27. X-Ray spine (AP & Lateral View)
(Thoracic and Lumbo sacral region) Nor. Abnormal

--	--

If abnormal, specify abnormalities _____
28. Both Hip joints Nor. Abnormal

--	--

If abnormal, specify abnormalities _____

CENTRAL COUNCIL FOR RESEARCH IN AYURVEDA AND SIDDHA, NEW DELHI

CLINICAL EVALUATION OF THE EFFECT OFCOMPOUND/ HERBOMINERAL
COMPOUND DRUGS/PANCHAKARMA THERAPY IN THE MANAGEMENT OF Pangu
(PARAPLEGIA)

FORM IV, For Periodical Observation and Assessment

Name of the patient:

Address:

Centre

--	--

Code No. (of clinical trial)

--	--

Patient No.

--	--	--	--

Group No.

--

Parameters to be taken for assessment of response of therapy	Initially at the time of admission	1 st	2 nd	3 rd	4 th	Follow
		Asses.	Asses.	Asses.	Asses.	up
(1)	(2)	15 days	30 days	45 days	60 days	
		(3)	(4)	(5)	(6)	(7)
