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संज्ञाहरण शोध

An Official Journal of

BHARATIYA SANGYAHARAK ASSOCIATION

(Association of Anaesthetists of Indian Medicine)

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EDITORIAL

I am pleased to inform that our coming 13th National and 2nd International conference will be held on 6th – 8th Feb. 2010 at B.H.U., Varanasi in collaboration of Section of Sangyaharan, Faculty of Ayurveda, IMS, BHU, Varanasi and U.P. State Branch of A.A.I.M. It will be a unique conference in the sense that first time demonstration of Nerves on cadaver will be organized by an Ayurvedic institution. First time different nerves will be demonstrated on cadaver and then a live demonstration of Nerve block will be presented by a group of Ayurvedic & Allopathic Anesthesiologists. The Anatomy department of IMS, BHU, will provide all the help and expertise for success of this workshop. Five orations and five other workshops will be arranged with a best paper and best poster presentation session. A series of guest lectures on different topics e.g. Pain management, Palliative Care, Nerve Block, Agni Karma, Jalauka and Critical care will be attraction of this conference. A sight seeing of Ghats & Sarnath will be also arranged for accompanying persons & guests. A session on Yoga and visit of yoga centre will be arranged. The organizers hope for a large participation from India, Nepal, Srilanka, U.K. & U.S.A. The stay will be made comfortable, pleasant and fruitful.

A book on C.C.P.R. will be released at this poise occasion with an attractive souvenir. The regular February Issue of Sangyaharan Shodh will be also released.

On behalf of organizing committee I appeal to all the members to participate in this conference and transfer their knowledge and clinical experiences to update Ayurvedic Sangyaharak (Anesthesiologist).

Jai Hind **Jai Ayurved** **Jai Sangyaharan**

Devendra Nath Pande
Chief Editor

संज्ञाहरण दिवस दिनांक ६ फरवरी २००९

दिनांक ६ फरवरी २००९ को भारतीय संज्ञाहारक चिकित्सको द्वारा संज्ञाहरण दिवस समारोह, आपदा प्रबन्धन (साप्ताहिक) एवं सी०एम०ई० (Role of CT Scan in Diagnosis and Neonatal Resuscitation) का आयोजन संज्ञाहरण प्रभाग– शल्य विभाग, आयुर्वेद संकाय, चिकित्सा विज्ञान संस्थान धनवन्तरि भवन में आयोजित किया गया। इस अवसर पर भारतीय संज्ञाहारक चिकित्सक – शिक्षक एवं एसोशिएसन के पदाधिकारी तथा लगभग १५० प्रतिभागियों ने भाग लिया। समारोह के मुख्य अतिथि का०हि०वि०वि० के रेक्टर माननीय प्रो० बी०डी० सिंह, सम्मानित अतिथि प्रो० ए०के० पाल– भू०पू० विभागाध्यक्ष, नि: संज्ञा कलकत्ता मेडिकल कालेज, समारोह के अध्यक्ष प्रो० गजेन्द्र सिंह, निदेशक, चिकित्सा विज्ञान संस्थान, प्रो० वी०के० जोशी, संकाय प्रमुख आयुर्वेद, प्रो० एस०सी० वार्ष्णेय– शल्य तंत्र विभागाध्यक्ष थें।

समारोह की विषयवस्तु पर प्रकाश डालते हुए संज्ञाहरण प्रभाग के प्रभारी डा० डी०एन० पान्डे ने अतिथितियों का स्वागत किया। डा० वी०के० जोशी ने संज्ञाहरण विज्ञान की महत्ता पर प्रकाश डालते हुए उनके अन्य आयामों पर अनुसंधान करते हुए लोकोपयोगिता पर कार्य करने की प्रेरणा दी। प्रो० गजेन्द्र सिंह ने प्रभाग के कार्यो को प्रति संतोष व्यक्त करते हुए आयुर्वेद में प्राचीन काल के शल्य संज्ञाहरण पर प्रकाश डालते हुए वर्तमान में उनके संन्दर्भो पर अनुसंधान की महत्ता पर प्रकाश डाला तथा शीघ्र ही इसे स्वतंन्त्र विभाग बनाये जाने की बात की।

मुख्य अतिथि प्रो० बी०डी० सिंह ने संज्ञाहरण विषय की उपयोगिता पर उनके अन्य क्षेत्रो शिक्षण, अनुसंधान, चिकित्सा कार्य में आयुर्वेदिक सिद्धान्तों एवं औषधियों की महत्ता पर प्रकाश डाला। डा० डी०एन० पान्डे द्वारा लिखित पुस्तक "अनुशस्त्र कर्म" का विमोचन मुख्य अतिथि ने किया। समारोह का संचालन डा० आर०के० जायसवाल एवं धन्यवाद ज्ञापन डा० के०के० पान्डेय ने किया।

शैक्षिक सत्र प्रथम में कर्नल डा० के०के० सिंह एनेस्थीसियोलाजिस्ट ने सी०टी० स्कैन की उपयोगिता पर प्रो० एल०एम० सिंह मेमोरियल व्याख्यान दियें। अध्यक्षता प्रो० एस०सी० वार्ष्णेय ने की।

दूसरे सत्र में प्रो० ए०के०पाल ने नवजात श्वासावरोध पर प्रो०एम०एन० चौधरी स्मृति व्याख्यान दिया। अध्यक्षता प्रो० एम० साहू ने की।

तृतीय सत्र में डा० के०के० पान्डेय व डा० आर०के० जयसवाल आपदा प्रबन्धन (C.C.P.R.) पर डमी माडल द्वारा संकट कालीन प्राणरक्षा उपायों पर प्रदर्शन द्वारा व्याख्यान दिया। अध्यक्षता प्रो० ए०के०पाल ने की। अन्त में भारतीय संज्ञाहारक एसोशिएसन (उ०प्र०) की सभा हुई।

डा० डी०एन० पान्डे
प्रभारी संज्ञाहरण प्रभाग

डा० आर०के० जायसवाल
सचिव संयोजन समिति

GUGGULU (Commiphora mukul) IN THE MANAGEMENT OF POSTOPERATIVE PAIN

*Dr. D.N. Pande **Dr. P.R. Mishra

Abstract :

Many methods and drugs have been described for relief of pain in Ayurveda by administering orally, application on body surface in form of paste, steam bath, dietary advises etc. Guggulu is one such drug which has been used since Vedic period till today for medical purpose in some form or other, it was worshiped like God; used for fumigation; described under Sangnasthapaniya Varga in charka samhita and Eladigana of Katu varga in Sushruta samhita and Vagbhata.

To know its efficacy in acute painful condition as a sole analgesic agent was an attempt for safe ideal analgesic. For this purpose study was conducted on 40 patients of standard population undergoing primary threading by Kchar-Sutra for fistula in Ano in Shalya Shalakya department of institute of medical sciences, Banaras Hindu University, Varanasi. The patients were divided into two groups of 20 patients each. One group was given tab. Diclofenac sodium (50 mg) and other group was given capsule of purified Guggulu. (Commiphora mukul)- 1000mg for analgesia when required. There comparative efficacy as analgesic as well as effect on psycho physiological status were evaluated before premedication, after premedication and also during anesthesia and after recovery from anesthesia

The study reveals that Cap. Commiphora mukul have mild analgesic effect in post operative pain management in comparison to Tab.Diclofenac sodium.

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KEY WORD- Glycopyrrolate, Diclofenace sodium, Guggulu, sedation, apprehension and induction.

Introduction:

Guggulu- the drug under clinical study was being used since long in Ayurvedic practice for the treatment of inflammatory and painful conditions. Guggulu is one of such drug which has been used since Vedic period till today for medical purpose. Recently many works have been done on various herbal drugs for the management of pain. It inspires us to carryout a clinical study in the field of Sangyaharan to explore an Ayurvedic analgesic/ adjuvant for post operative pain management.

Aims and Object:

Now days the clinical trail of drugs has been increased in many folds. The most important support in favor of the clinical trial is to confirm the observations and claims made by previous workers in their experimental studies and also to find out any additional action and side effects which were some times not observed in experimental study but are observed in human being only. Therefore we planned to conduct a study to prove the efficacy of guggulu as an anti-inflammatory/ analgesic/ adjuvant for post operative pain management.

The aim of this study was to explore the possibility to provide a safe and effective Ayurvedic analgesic Till date many works had been done but no full proof analgesic is in hand.

Materials and Methods:

The crude drug (guggulu rhizome) was collected from Ayurvedic Pharmacy, Institute of Medical Sciences, Banaras Hindu University, Varanasi and its validity was confirmed by Dravyaguna Department. The visible impurities were remove manually. this sample was further broken down into small pieces and taken into a strong fine cloth and bundled. The cloth bundled with a strong thread all around in such a way that no guggulu particles can come out as such. About 4 liter if water was taken in a big stainless steel container and glass rod was kept horizontally above. The cloth containing guggulu was freely suspended and fully submerged in water. This container was put on electrical heater. Guggulu dissolves in water. When it become paste like it is removed from source of heat. The purified guggule was weighed, dried and keeping free from moisture. A 500 mg course powder was put into the capsules.

Determination of the Dose of Drugs

The dose for clinical study was calculated according to the dose of churn recommended for guggule in Ayurvedic literatures by various authors. For an adult weight- 40-60 kg, the dose of the drug was 500 mg.

Grouping of he patients & Inclusion criteria:

For this purpose study was conducted on 40 patients of standard population undergoing primary threading by Kchar-Sutra for fistula in Ano in Shalya Shalakya department of institute of medical sciences, Banaras Hindu University, Varanasi. The patients were divided into two groups of

20 patient each. There comparative efficacy as analgesic as well as effect

on psycho physiological status were evaluated before premedication, after premedication and also during anaesthesia and after recovery from anaesthesia

Group I: The patients of control group I received Tab. Diclofenace sodium 50 mg with an ounce of water for analgesia.

Group II: The patients of group II (trial group) received two Capsules of Commiphora mukul 500 mg with an ounce of water for analgesia.

Before giving the premedication, the B.P., P.R., R.R., Temperature, G.C., C.V.S.,R.S..,G.I.T. were checked and recorded on a proforma. 90 minutes after premedication, the effects achieved were also recorded before induction of anesthesia in calm and quite surroundings. For recording the effects, a cyclostyled Performa was employed, so that none of the planned observations could be missed. For evaluation of (1) desirable and (2) undesirable effects of the post operative analgesics, an assessment of the following signs and symptoms was made by the investigator himself.

Exclusion criteria:

The following classes of patients were excluded from the study-

1. Those that were out of 18 to 50 years of age.
2. Who were out of A.S.A. GP. 1&2.
3. Who were pregnant?
4. Patient suffering from Respiratory, cardiac, hepatic, renal, sensitive to aspirin, Diclofenace sodium, bleeding disorders and peptic ulcer.

The trial drug was clinically studied on following three stages –

1. Psychophysical effect in pre-anesthetic period before induction of anaesthesia.
2. Cardio-respiratory and other reflex responses during the course of subsequent anaesthesia.
3. Post-operative sickness in immediate post-operative period unto two hours.

Technique of Anaesthesia-

Saddle block at L4-L5 with 25 G spinal needle with Inj.Xylocaine 0.5 % 1 ml.

Observation, Result & Discussion:

The following observation was made at the label of Premedication study.

Table 1. Grouping of Patients:

Groups	Number of Patients	Premedication Drugs
Control (I)	20	1. Tab. Diclofenac soduium 50 mg night before operation. 2. Tab. Diclofenac soduium 50 mg 2 hrs before induction of anaesthesia. 3. Inj. Glycopyrrolate 0.2 mg IM 60 min.before anaesthesia.
Trial (II)	20	1. Capsule of Commiphora mukul 1000 mg night before operation. 2. Capsule of Commiphora mukul 1000 mg 2 hrs before induction of anaesthesia. 3. Inj.Glycopyrrolate 0.2 mg IM 60 min.before anaesthesia.

Table 2. Comparison of Mean age and weight:

	Group I	Group II	Statistical Comparison t	p	Remark
Mean age (years)	43.65±8.68	46.3 ±13.3	0.743	>0.05	N.S.
Mean weight (kg)	59.85 ±5.47	59.1 ± 5.37	0.438	>0.05	N.S.

Age and Weight is identical.

Table 3. Effects on Pulse Rate changes per minute

Group	Before premedication (A)	After Premedication (B)	During anaesthesia (C)	After recovery(D)
I	83.3± 7.57	86.0± 7.16	86.85±6.38	86.0±6.83
II	81.45± 7.05	82.85±6.18	83.3±6.29	82.9±6.30

Comparison of Pulse within the groups

comparison of Pulse within the groups	GROUP I			GROUP II		
	t value	p value	remark	t value	p value	remark
A vs B	1.19	>0.05	N.S	0.572	>0.05	N.S
A vs C	1.65	>0.05	N.S	0.879	>0.05	N.S
A vs D	1.21	>0.05	N.S	0.796	>0.05	N.S
B vs C	0.39	>0.05	N.S	0.22	>0.05	N.S
B vs D	0.00	>0.05	N.S	0.02	>0.05	N.S
C vs D	0.40	>0.05	N.S	0.20	>0.05	N.S

Table 4. Effects on Blood Pressure changes (M.B.P.)

Group	Before premedication (A)	After Premedication (B)	During anaesthesia (C)	After recovery(D)
I	91.1± 3.49	92.4±3.63	93.55±3.23	93.45±3.08
II	91.3± 5.14	92.4±4.79	92.95±3.61	92.2±4.0

Undesirable Effects						
Dizziness	Present	0	0	0	0	0.00
	Absent	20	100	20	100	
Vomiting	Present	1	5	2	10	0.603
	Absent	19	95	18	90	
Nausea	Present	2	10	3	15	0.479
	Absent	18	90	17	8	

Table 8. Mean induction time, Anaesthetic time and Surgical time

	GROUP I	GROUP II	T value	P value	remark
Mean induction time (mnts)	4.6±1.095	4.932.39	1.79	>0.05	N.S.
Surgical time	13.05±4.39	14.02±3.96	0.737	>0.05	N.S
duration af anaesthesia	71.5±16.69	69.15±17.38	1.95	>0.05	N.S

Table 9-ANALGESIC REQUIREMENT TIME

1st analgesic dose requirement-

Group	mean± S.D	T value	P value	remark
I	99.65± 28.48	2.041	<0.05	Significant
II	81.4± 28.03			

Comparison of temperature within the groups

comparison of temperature within the groups	GROUP I			GROUP II		
	t value	p value	remark	t value	p value	remark
A vs B	0.75	>0.05	N.S	0.79	>0.05	N.S
A vs C	2.57	<0.05	S	2.12	<0.05	S
A vs D	2.20	<0.05	S	3.26	<0.05	H.S
B vs C	1.74	>0.05	N.S	2.30	<0.05	S
B vs D	1.41	>0.05	N.S	2.68	<0.05	S
C vs D	0.47	>0.05	N.S	0.30	>0.05	N.S

Table 7. Desirable and Undesirable Effects

	Incidence	Group I		Group II		Z value
		No..	%	No.	%	
Desirable Effects Sedation	Present	1	5	2	10	0.603
	Absent	19	95	18	90	
Lack of Apprehension	Present	3	15	4	20	0.417
	Absent	17	85	16	80	
Loss of Excitement	Present	18	90	17	85	0.479
	Absent	2	10	3	15	

Comparison of R.R within the groups

comparison of R.R within the groups	GROUP I			GROUP II		
	t value	p value	remark	t value	p value	remark
A vs B	0.86	>0.05	N.S	0.19	>0.05	N.S
A vs C	1.06	>0.05	N.S	0.85	>0.05	N.S
A vs D	0.83	>0.05	N.S	0.33	>0.05	N.S
B vs C	0.05	>0.05	N.S	0.99	>0.05	N.S
B vs D	0.08	>0.05	N.S	0.53	>0.05	N.S
C vs D	0.27	>0.05	N.S	0.60	>0.05	N.S

Respiratory rate was found insignificant at all the level.

Table 6. Effects on Body Temperature changes (0^F)

Group	Before premedication (A)	After Premedication (B)	During anaesthesia (C)	After recovery(D)
I	97.64± 0.50	97.75±0.47	98.03±0.52	97.96±0.48
II	97.61± 0.47	97.72±0.42	98.02±0.44	98.06±0.42

Comparison of M.B.P within the groups

comparison of M.B.P within the groups	GROUP I			GROUP II		
	t value	p value	remark	t value	p value	remark
A vs B	1.153	>0.05	N.S	0.70	>0.05	N.S
A vs C	2.30	<0.05	S	1.175	>0.05	N.S
A vs D	2.26	<0.05	S	0.618	>0.05	N.S
B vs C	1.05	>0.05	N.S	0.40	>0.05	N.S
B vs D	0.98	>0.05	N.S	0.14	>0.05	N.S
C vs D	0.10	>0.05	N.S	0.62	>0.05	N.S

Table 5. Effects on Respiratory Rate changes (per minute)

Group	Before premedication (A)	After Premedication (B)	During anaesthesia (C)	After recovery(D)
I	17.05± 2.37	17.65±1.98	17.75±1.98	17.6±1.75
II	17.1± 1.68	17.0±1.62	17.73±1.82	17.28±1.712

2nd analgesic dose requirement

Group	mean± S.D	T value	P value	remark
I	359.0± 69.72	9.25	<0.001	Very highly significant
II	183.5± 48.15			

Postoperative Complication: Table No. 10

Complication	Incidence	Group I		Group II		Z value	Remark
		No.	%	No.	%		
Headache	Present	3	15	3	15	0	N.S.
	Absent	17	85	17	85		
Backache	Present	3	15	5	25	0.79	N.S.
	Absent	17	85	15	75		
Convulsion	Present	0	0	0	0	0	N.S.
	Absent	20	100	20	100		
Hyper-sensitivity Reaction	Present	0	0	0	0	0	N.S.
	Absent	20	100	20	100		
Nervous system involvement diplopia; other cranial nerve involvement	Present	0	0	0	0	0	N.S.
	Absent	20	100	20	100		
Retention of Urine	Present	0	0	0	0	0	N.S.
	Absent	20	100	20	100		
Other	Present	0	0	2	0	0	N.S.
	Absent	20	100	20	100		

Conclusion

Guggulu is an effective drug in alleviating chronic inflammatory and chronic painful condition. In this study sole effect of Guggulu as analgesic agent has been observed.

First analgesic dose requirement time was significantly high in control group in comparison to trial group.

Second analgesic dose requirement time was very highly significant in control group who were given Tab.Diclofenac sodium in comparison with trial group who were given Guggulu (Commiphora mukul).

On the basis of observation it is concluded that the incidence of satisfactory pain relief was much higher in group I in comparison to group II.

The study reveals that Cap. Commiphora mukul have mild analgesic effect in post operative pain management in comparison to Tab.Diclofenac sodium.

On the basis of above observations we conclude that-

1. The trial drug guggulu has some analgesic property but the control drug tab.diclofenac sodium has more potent analgesic action.

2. In our study both drug have comparable and negligible side effects.

3. No significant changes in C.V.S were found with either drug.

4. Different method of purification, preparation of combination drug at other dose schedule have to be given and more patients involved to obtain acceptable, beneficial results.

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FLUID THERAPY- A CHALLENGE

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Introduction:-

Fluid therapy is a very vast topic to discuss about the types of fluids, electrolytes,acids and bases, its application in various pathophysiological conditions in our body.The practitioners of many disciplines of medical sciences are daily encountering fluid and electrolytes imbalances.Now it is the matter of great concern that how will we manage these fluid and electrolytes in a very simple way so that a practitioner of urban or rural area can easily practiced in the day-to-day management of the patients.I am very confident that this topic will provide detailed information about a complete fluid therapy in different medical & surgical problems.

Normal physiology

Water is the major component of our body. The percentage of total body water (TBW) to weight ranges from about 55% in the adult female, 60% in the adult male, to nearly 80% in the newborn. In the adult male of 70 kg, TBW is equal to about 42 l. There are three main compartments through which it is distributed, intracellular fluid (ICF) which contains about 27 l, interstitial fluid (ISF) 12 l and plasma volume (PV) of 3 l. The latter two (ISF and PV) constitute extra cellular fluid (ECF). PV forms the medium in which red blood cells carrying oxygen can be transported to all cells of the body and together form the circulating volume (CV) of 5 l. Tran cellular fluid (TCF) is defined as fluid in transit between various compartments, usually in body cavities such as the gut lumen where it is continuously added to by ingested fluids, secreted and re absorbed. The volume at any time is usually not large but may increase markedly in derangement of gut function such as paralytic illus, diarrhea and vomiting or is lost iatrogenic ally as by nasogastric suction. TCF is extremely difficult to quantify but should always be considered when trying to quantify fluid losses and shifts in the surgical patient. Control of volumes and constituents. In health, the volume and electrolyte distribution of these compartments of TBW are controlled by three major processes:

1.The Na+/K+ pump

An active process, relying on ATP and a Na+/K+ exchange pump, controls the electrolyte composition of the ICF and ISF. The major intracellular cation is K+ (140-160 mEq.l-1) with Na+ of 10-40 mEq.l-1 (depending on the type of cell), whilst in the ISF and PV the ratios are reversed with Na+ being the predominant cation (132-145 mEq.l-1) with a K+ of 3.5-5 mEq.l-1. Total body exchangeable Na+ and K+ are roughly equivalent to 40 mEq.kg-1 BW. Derangement to this active process in disease results in K+ leak from the cells and into the ECF with Na+ (and water) going in the opposite direction.

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2. Osmolality and tonicity

Maintenance of osmotic balance ensures that the total concentration of osmotically active particles is the same throughout all three compartments. The major osmotically active cation in ECF is Na+ whilst in the ICF it is K+. Osmolality is simply the number of osmotically active particles per Kg of solvent (in this case water). Table 1.

Table: 1- The major osmotic constituents of plasma:

Cation/Anion	Concentration in mEq.l-1
Na+	135
K+	5
Cl-	110
HCO3-	24
Glucose	5
Urea	5
TOTAL	284 (mosm.l^{-1})

Thus, a fall in plasma Na+ results in fall in PV osmolality in comparison to that in the ISF. Water will move from PV to ISF in an attempt to equalise the osmotic pressures (osmolality) in the two compartments. The resulting overall fall in ISF osmolality leads to further movement of water into the ICF. An opposite effect is seen with a rise in PV Na+. In other words a fall in plasma Na+ always results in an increase in ICF and vice versa.

Obviously, the three compartments are not in a passive state. The dynamics of the circulation and the requirement to transport oxygen and nutrients around the body necessitates the generation of a pressurized flow of blood from left heart to right atrium. The hydrostatic pressure generated by this column of blood in the capillaries would inevitably lead to a net loss of fluid by ultra filtration from the PV into the ISF and eventual depletion of the CV. This hydrostatic pressure is balanced by the presence of **colloids** in the PV.

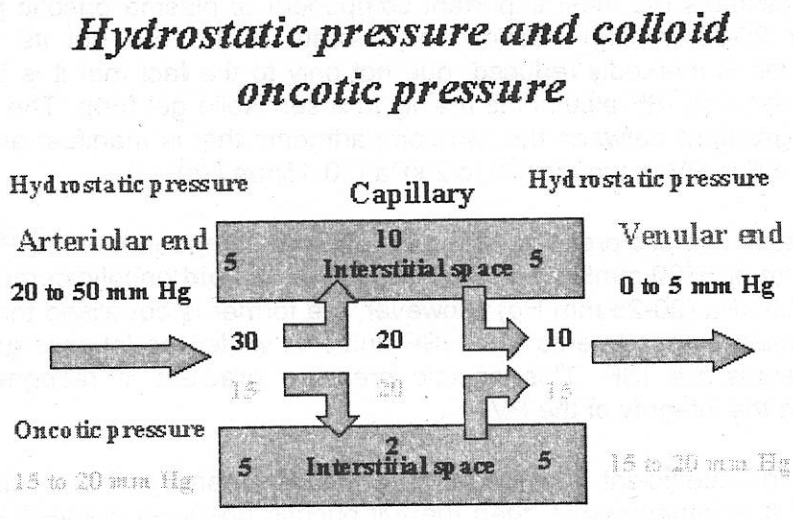
A semi-permeable membrane (the capillary endothelium) represents the barrier between the PV and the ISF. High molecular protein constituents of plasma exert '**colloid osmotic**' or '**oncotic**' pressure. Colloids are molecules capable of exerting oncotic pressure and have limited (or zero) ability to cross a semi-permeable membrane due to their molecular size. At the same time, they have the ability to attract solvent (solvent drag) from the other side of the membrane into the compartment in which they are situated (in this case PV). The oncotic

pressure of a plasma constituent is proportional to the amount (in g.l-1) divided by the molecular weight. In man, plasma albumin (40 g.l-1) with a MW of 60,000 constitutes the most important component of plasma oncotic pressure (3 kPa or 20-25 mmHg). Albumin also exists in the ISF but its effective concentration is markedly reduced, due not only to the fact that it is bound to cells, but also that ISF albumin is mainly in a semisolid gel form. The **oncotic pressure gradient** between the two compartments that is manifest as solvent drag from ISF to PV is around 1.5 to 2 kPa (10-15mm Hg).

Thus, the total osmotic pressure of the **crystalloid** component of the PV is 680 kPa (6.8 atm or 5100 mmHg, see later) whilst the **colloid** oncotic component is only 3 to 3.5 kPa (20-25 mm Hg). However, the former is equalised throughout the three main compartments (ICF, ISF and PV) whilst the latter is greater in the PV versus the ISF. This oncotic pressure gradient is responsible for maintaining the integrity of the PV.

The reflection coefficient is a measure of the permeability of the capillary to albumin. If it is impermeable, then the full oncotic pressure gradient between plasma and ISF is experienced, s will be 1. If, on the other hand, the capillary is completely permeable to albumin, then no gradient exists, and fluid leaks out entirely as expressed by the hydrostatic pressure gradient. This would result in a s of 0. In practice, depending on the capillary bed in question the range is about 0 (liver) to 0.7 (lung).
A fall in the oncotic pressure gradient, due to loss of albumin or a reduction in the reflection coefficient due to capillary endothelial damage (vide infra), causes a loss of PV and an increased propensity to the development of significant tissue and pulmonary oedema.
Overall, plasma electrolytes represent a dynamic interchange between total body stores, input and output and passive and active movements between compartments as controlled by the processes alluded to above.
Figure 2 shows the effect of the Starling Equation and the typical overall pressures in the capillaries and venules and the fluid shifts which occur. Please note that as fluid leaves the circulation at the arteriolar end, the hydrostatic pressure gradient gradually diminishes in the capillaries and the oncotic pressure gradient gradually increases. Thus, by the time we reach the venular end, the pressure gradients have been reversed and fluid reenters the circulation again.

Figure 2: Hydrostatic and colloid oncotic pressure gradients in the capillaries.



Conditions causing an increase in capillary endothelial permeability (CEP)

Three common conditions cause an increase in CEP:

- **Anaphylactic shock** (e.g. due to penicillin allergy) results in increased leakage of fluid leading to sudden hypovolaemia along with peripheral oedema and bronchoconstriction The initial treatment for a patient without venous access is i.m. adrenaline (0.5 to 1 mg i.e. 0.5 to 1 ml. of a 1:1000 solution) or in a patient with i.v. access, 1 to 2 ml. boluses of i.v. adrenaline (1mg in 10 ml. or 1:10,000). Fluids and oxygen therapy should be used in addition.

- **Burn injury** causes a systemic 'capillary leak' which is proportional to the percentage of the burned area. The amount of fluid leakage is massive and is equivalent to 4 ml. of Ringer's Lactate per kg. per % area burn in 24 hours (Parkland formula). E.g. in a 60 kg patient with a 40% burn this requires 4 * 60 * 40 = 9600 ml, 50% given in the first 8

- hours, 25% in the next two 8 hour periods) calculated from the time of the **burn injury**.

- **Septic shock** or systemic inflammatory response syndrome (SIRS) also leads to loss of CV and this fluid must be replaced in large quantities to maintain CV, despite the inevitable oedema that this causes.

Regulation of blood volume

Day to day homeostasis

The factors mentioned above are all designed to maintain the integrity of the PV and CV so that it can form the medium to allow transport of oxygen and nutrients for cellular metabolism. This ensures optimal organ function, but in a dynamic process the volumes are not static. Adequate clearance of waste products of metabolism require the kidney to excrete about 1500 ml of urine per day containing about 400-800 mmol of urea, 50 to 100 mmol K+ and 70 to 140 mmol. Na+ (depending on intake). Additional fluid losses are incurred from evaporation, respiratory tract and faeces. The total loss of fluid is about 2500 ml. water plus about 70 mmol Na+ and K+ in a 70 kg adult per day. This has to be replaced to maintain TBW. The extent of maintenance fluid requirements is more closely related to body surface area (BSA) than weight. Since a new born baby of 3.5 kg. has about 2 1/2 times the BSA/wt ratio of an adult (1/20th the weight and 1/8 th the BSA) it consequently needs 2 1/2 times the maintenance fluid per unit weight. A common formula used is shown in Table 2.

Table 2 Maintenance fluid requirements

Weight range	Fluids per 24 hours
3 - 10 kg	100 ml.kg-1
10 - 20 kg	1000 ml. for first 10 kg. + 50 ml.kg-1 for any additional wt. over 10 kg
20 kg and above	1500 ml. for first 20 kg. + 20 ml.kg-1 for wt. over 20 kg.
Thus: **a 7 kg. infant would require 700 mls. per day** **17 kg child would require 1000 + 350 = 1350 ml. per day** **a 70 kg. adult would require 1500 + 1000 = 2500 ml. per day**	

Normally this fluid requirement is regulated both by changes in volume and osmolality in the PV being detected in the hypothalamus. An increase (due to fluid deprivation) stimulates anti-diuretic hormone (ADH) release from the supra optic nucleus of the hypothalamus which causes thirst and reduces urine output by increasing reabsorption of water from the collecting ducts in the kidney. A decrease in renal perfusion (due to fluid deprivation) increases renin output from the juxtaglomerular apparatus leads to the formation of angiotensin II (AT II). This causes thirst, constricts the efferent glomerular artery to maintain glomerular arterial pressure and filtration and also causes release of aldosterone from the adrenal cortex. The latter increases renal Na+ retention in exchange for K+ in the distal tubule.

All these changes are increased by activation of the sympathetic nervous system (SNS). If fluid deprivation becomes greater or there are abnormal losses such as diarrhoea, vomiting, ileus or haemorrhage, these processes are amplified. It is important to note that volume is usually maintained at the expense of a reduction in osmolality due to hyponatraemia. This is due to the more powerful effect of ADH over aldosterone. As plasma Na+ concentration falls, proximal tubular reabsorption of both Na+ **and** water becomes intense, thus limiting the ability of the kidney to produce dilute urine and to excrete a water load. Administration of hyponatraemic solutions in the postoperative

period in the presence of a low serum Na+ further compounds the problem. 5% glucose and 4% glucose 0.18% saline should never be given if the plasma Na+ is low. 0.9% sodium chloride or Lactated Ringer's solution (Hartmann's) is more appropriate.

Acute changes in homeostasis

Acute surgical or traumatic hypovolaemia cannot be compensated for by the more chronic processes mentioned above. Additional mechanisms are brought into play. Acute reduction in CV reduces venous return and cardiac preload so cardiac output and blood pressure (BP) fall. Reduction in BP reduces the afferent activity of the carotid sinus baroreceptors to the 'pressor' area in the dorsal hypothalamus, and results in increased SNS discharge. Reduction in venous return leads to a decrease in atrial natriuretic peptide (ANP) production (thus reducing urinary sodium loss) and a fall in output from the low pressure baroreceptors in the atria and great veins to the 'depressor' centre. The resultant fall in parasympathetic nervous system (PNS) discharge augments the action of the SNS.

The effects of this are summarised as follows:

- **Direct neural effects** via alpha and beta receptors.
- **Catecholamine release from the adrenal medulla**. In different shock states, and at different stages of shock, noradrenaline or adrenaline predominates.
- **Renin release** from the juxtaglomerular apparatus of the kidney (vide infra).
- **Increased Na+ reabsorption** in the distal tubule
- **Decreased Na+ loss** due to a reduction in atrial natriuretic peptide
- **Vascular redistribution of blood in the kidney** from cortex to medulla, the latter being the major site of Na+ and water reabsorption

The ISF compartment has an important role in maintaining circulating volume. Sympathetic stimulation, particularly to the skin and splanchnic circulation, results in a reduction of flow to these nonessential areas, by alpha adrenergically mediated arteriolar vasoconstriction. This results in a reduction of flow to the capillary beds by neuro-humorally mediated increase in the pre-capillary sphincter (PCS) tone. The hydrostatic pressure in the capillary beds falls, thus allowing fluid to enter the capillary circulation distal to the PCS as a result of the change in hydrostatic/oncotic pressure gradient (see Starling's equation above).

Intravenous perioperative fluid replacement therapy

Initially, it is pertinent to consider the types of fluids that are available and their uses. Fluids can be conveniently classified into crystalloids, colloids and blood (and blood products).

Crystalloids

These are solutions containing water and electrolytes and/or glucose made up in a concentration that is usually isotonic with plasma. This means that they contain the same number of osmotically active particles as plasma, i.e. about 300 (normal plasma osmolality range 280 to 310) mosmol.l-1 of solute. Several solutions are available, 5% glucose, 4% glucose 0.18% saline, Normal (0.9%) saline and Lactated Ringer's (Hartmann's) ... see table 3).

How do we make an isotonic solution? From first principles, if 1 gram molecular weight of solute is placed in a flask containing 1 kg. of solvent (i.e. 1 litre of water) it will form a molar solution. Thus, 180g of glucose (180 = the m.w. of glucose) in a kg. of water will have 1 mol. or 1000 mmols.l-1. By definition, if one mole of glucose is dissolved in 22.4 litres of solution at NTP (i.e. 0 deg C) it will exert an osmotic pressure of one atmosphere (100 kPa or 760 mm Hg). Thus, a molar solution will exert an osmotic pressure of 22.4 atmospheres (2200 kPa or 15200 mm Hg) at NTP or 25.4 atmospheres (2540 kPa or 19340

mm Hg) at 37 deg C. This amount is referred to as 1 osmol or 1000 mosmol. One mosmol at 37 deg C thus generates an osmotic pressure of 2.5 kPa (19 mmHg), thus in total the osmotic pressure of plasma is equal to 300 * 2.5 = 750 kPa (5700 mmHg). Thus, this molar solution of 18% glucose (180g per litre or 18 g per 100 ml) is hypertonic (1000 versus 300 mosmol.l-1). Each g per litre therefore gives about 5.5 mosmol.l-1, so 50g (5% solution of glucose) will produce an isotonic solution of 50 times 5.5 or 275 mosmol.l-1.

With an electrolyte such as sodium chloride, the molar solution (58.5 g of sodium chloride) will contain nearly 2000 mosmol. l-1 as in solution it is almost completely dissociated into sodium and chloride ions. Thus, roughly 1/6th. of 58.5g.l-1 is required, i.e. 9g per litre (0.9 g.100 ml-1 or 0.9% saline) with 150 mmol. of Na+ and 150 mmol. of Cl-. In a similar manner, 4% glucose and 0.18% NaCl will give 30 mosmol. of both Na+ and Cl- per litre plus 220 mosmol.l-1 from glucose making 280 in all. It can be seen that normal (0.9%) saline is hardly *physiological* as it contains excess Na+ (150 vs 135) and excess Cl- (150 vs 105). Excess Cl- administration can result in retention of H+ and urinary loss of HCO3- and a dilutional acidosis if administered in excess. Thus, ideally, a 'balanced' salt solution (BSS) should be used. Such a solution is Lactated Ringer's (Hartmann's solution). This contains in mmol.l-1, Na+ 131, K+3.5, Cl 105, Ca++ 2, Lactate 29. The latter is a H+ acceptor and is metabolised by the liver with net formation of one molecule of HCO3- and one molecule of pyruvate which can be utilised as an energy source in the Krebs cycle. It does not cause a dilutional or lactic acidosis provided that liver perfusion is adequate.

It is worth noting that a fall in serum Na+ of 5 mmol.l-1 (accompanied by a change in Cl- of the same amount) will result in a pressure dysequilibrium between the PV, ISF and ICF of 1/30th (10/300) of total plasma osmotic pressure or about 190 mm Hg (1/30th of 5900). This large pressure difference, should it occur rapidly, results in water leaving the PV into the ISF and then into the ICF in an attempt to restore osmotic balance between the fluid compartments. If this occurs acutely, as in excess absorption of non-sodium

containing irrigation fluid during transurethral resection of prostate (TURP), cerebral oedema and raised intracranial pressure may occur. The latter occurs in the *TURP syndrome*. However, it should be noted that glycine 1.5%, the irrigation fluid traditionally used during TURP, is **hypotonic**. The molecular weight of glycine is 75, so 75 g dissolved in a kg. of solvent (i.e. a litre of water) would be a molar solution and would contain 1000 mosmol.l-1. Thus 1.5% glycine (or 15 g. l-1) has an osmolality of 200 mosmol.l-1. To aid detection of excess absorption of fluid, ethanol (1%) is usually added as a marker. If a significant amount of irrigation fluid is absorbed, the ethanol component will be detectable in the breath (cf a 'breathalyzer'). The concentration of ethanol thus reflecting the extent of absorption. The addition of ethanol 1% to the 1.5% glycine affects its osmolality. The MW of ethanol is 46, thus 10 g per litre (i.e. a 1% solution) will increase osmolality by 217 thus making the total osmolality of the irrigating solution 417 i.e. **hypertonic**! Since this combined solution is now commonly used it may account for the diminished incidence of TURP syndrome.

Thus, absorption of irrigation solution will increase **overall** osmotic **pressure** in the PV but still result in a fall in plasma Na+. In fact, the TURP syndrome is more to do with fluid overload and the possible cerebral effects of glycine than the effect of the drop in plasma Na+. In the same way, infusion of 5% glucose will cause a fall in plasma Na+ due to a dilutional effect but osmotic pressure will remain the same as the solution is isotonic. As the glucose is metabolised (the glucose concentration in 5% glucose is about 275 mmol.l-1 whilst in plasma it is 5), the osmotic pressure in the plasma will fall and water will go into the cells. The effect on cellular overhydration will depend on the rate of administration of 5% glucose. Thus, although non-sodium containing isotonic solutions will eventually result in increased cellular water they do not do so rapidly enough in most circumstances to cause problems of dysequilibrium between the body compartments.

In chronic hyponatraemia, as in patients on diuretic therapy, there is time to restore osmotic equilibrium without major pressure differences occurring between compartments. However, use of hypertonic saline may result in too rapid correction of Na+ in the plasma will cause rapid reversal of the above process and cerebral dehydration which can be equally dangerous. Thus, it is much easier to raise Na+ concentrations rapidly than to cause a fall. The commonest cause of excess hypertonic Na+ adminstration is the use of Na+HCO3- solutions (see Table 3).

Electrolyte concentrations of commonly used crystalloid solutions are shown in the Table 3.

Table 3 Content of Crystalloid solutions

Content of crystalloid solutions									
Name	**Known as:**	**Na+**	**Cl-**	**K+**	**Ca****	**Mg++**	**HCO3-**	**Lactate-**	**mosmol.l-1**
NaCl 0.9%	Normal Saline	154	154						308
Lactated Ringer's	Hartmann's	131	111	5	2			29	280
Glucose 5%	5% dextrose								252
Glucose 4% + NaCl 0.18%	Dextrose saline	30	30						286
Plasmalyte 148 + glucose	Plasmalyte	148	97	5		1	40*		552
Sodium Bicarbonate 8.4%		1000					1000		2000
amounts in mmol.l-1									
* (as gluconate/actetate)									

Crystalloid solutions containing isotonic concentrations of Na+ do not remain in the PV following i.v. administration. The Volume of Distribution (Vd) of these fluids is ECF and thus they only provide a short term expansion of the CV. Approximately three times the volume of estimated blood loss must be given to maintain CV. In severe blood loss, massive doses of crystalloid for

resuscitation (together with blood) have been implicated in the formation of pulmonary and generalised tissue oedema due to the large volumes required for resuscitation. Although this has not been substantiated in clinical trials, many anaesthetists employ colloid containing solutions for resuscitation as lower volumes are required.

Colloids

Also known as 'plasma expanders', these solutions contain high molecular weight substances such as dextrans, gelatins and starch which exert oncotic pressure and thus retain fluid in the circulating volume as stated above. In addition, naturally occurring colloid solutions such as 5% albumin and plasma protein fraction (PPF) can be used but they are expensive and have no advantages over the other synthetic colloid solutions (Table 4). **Table 4: Commonly used colloid solutions**

Content of Colloid solutions					
Name	Avge. MW	MW range	t1/2 in PV	Effect on coagulation	
PPF	69,000	69,000	20 days	none	
Dextran 70	38,000	10-250,000	12 hr.	Decreased platelet aggregation, reduced Factor VIII	
Haemaccel	24,500	5-50,000	2.5 hr.	none	
Gelofusin*	22,600	10-150,000	4 hr.	none	
Voluven 6%	130,000	10,000 to 10^6	6 hr.	Yes, but only if > than 50ml.kg-1.day	
* Contains no K+ or Ca++ N					

Intraoperative fluids

Not all patients undergoing surgery need intravenous fluid therapy. Indeed, intravenous fluid administration is not devoid of risks (air embolism, deep vein thrombosis, overloading, discomfort to the patient, infection risk and considerable cost), so it should not be undertaken lightly. There are three components to fluid therapy in the surgical patient. Firstly, as it is customary to deprive patients of fluids for at least 4 hours prior to a surgical procedure under general anaesthesia, there is an element of maintenance fluid deficit. Secondly, depending on the site and severity of the operative procedure, there will be an element of compartmental fluid shift due to tissue trauma (the so-called 'third space loss'). Thirdly, there will be the additional losses of blood and other fluids such as excessive urine output, ascites and fluid collections in the peritoneal or pleural cavities.

Maintenance fluid

The average daily requirements stated above correspond to the normal physiological losses (70 kg patient) through the urine (1.5 l), faeces (100-200 ml), perspiration (300-500 ml) and respiration (500 ml). It is not necessary to replace this loss intravenously for many minor procedures where the preoperative period of fluid deprivation has been short and it is expected that oral fluids can be commenced within hours of the procedure's termination. If maintenance fluids are required then the aim should be to replace the preoperative deficit and then provide the requirements on an hourly basis until oral fluids are tolerated. On a 24 hour basis, 4% glucose 0.18% NaCl provides a suitable solution as 2500 ml (for a 70kg patient) provides 75 mmol. Na+. On an hourly basis this equates to 100 ml. 400 ml. will compensate for the preoperative deficit and then 100 ml. thereafter (see formula above). Addition of K+ is not required for short term (less than 48 hours) therapy. However, it should be noted that the indiscriminate use of this hyponatraemic solution in paediatrics has occasionally resulted in hyponatraemia. As a result, this solution is no longer available on paediatric wards and a solution containing 5% Glucose and 0.45% Na is used instead. This avoids hyponatraemia but results in the child gettinbg excessive amounts of Na+!

Unphysiological losses

These may arise from surgical drains, nasogastric tubes or vomiting, diarrhoea, excessive body temperature and excessive urinary output due to diuretic drugs. Losses arising from nasogastric tubes, drains and urine output can be accurately measured whilst losses due to high body or ambient temperatures can only be roughly estimated.

The measurable losses must always be replaced as accurately as possible in volume, Na+ and K+ content: note that diarrhoea has a high K+ content (20-50 mmol-1) and vomit has a high chloride content (80-100 mmol-1). Patients on thiazide diuretics or frusemide may lose 50-70 mmol of K+ per litre of urine.

Elevation of body temperature is reasonably compensated for by an increase in the normal water, Na+ and K+ intake of 15% for each degree C above the normal 37.

Third space losses

This only becomes relevant during major surgery (or trauma) where there is extensive tissue damage, e.g. major abdominal and thoracic surgery. Cellular damage results in an inability to maintain the Na+/K+ pump, so Na+ and water leak into the cells which become swollen and oedematous. The major loss of fluid, however, is from the PV into a non-exchangeable compartment of the ISF (the so-called 'third space'). This is due to alterations in the oncotic/osmotic balance between the PV and ISF, again as a result of tissue damage. In extensive surgery these losses can be considerable, although estimates which have been made of 15ml.kg-1hr-1 are now thought too high. A more reasonable figure is 5 ml.kg-1hr-1 or even less. This loss of functional extracellular fluid volume (FECV), if not replaced in adequate quantities, leads to further activation of ADH and aldosterone secretion as well as inhibition of atrial natriuretic peptide (ANP). It is not surprising that there is marked water and sodium retention with oliguria in the post operative period. This fluid should be replaced with adequate quantities of a BSS such as lactated Ringer's.

Recent work has demonstrated that fluid intake during major surgery should be optimised according to the individual patient requirement by observing changes in stroke volume in response to administration of fluid boluses. To do this obviously requires the use of apparatus to measure cardiac output such as the Deltex Cardio Q oesophageal doppler or LiDCO devices. The emphasis is now on **Flow** optimisation and less emphasis on Pressure.

Blood loss

Maintenance of adequate oxygen delivery (DO2) should be the primary aim rather than simply considering blood replacement.

DO2 = Cardiac output (l.min-1) * Arterial oxygen content

(Arterial oxygen content = SaO2/100 * Hb (g.l-1) * 1.34), ignoring the small amount dissolved in the plasma. Although the latter is not numerically important, it does form the all important interface between oxygen bound to haemoglobin and cells which require it.)

Thus, at a normal cardiac output of 5 lpm, oxygen saturation of 99% and Hb of 145 g.l-1:

$DO_2 = 5 * (.99 * 145 * 1.34)$

= 1000 ml.min-1

In considering when blood loss should be replaced, it is pertinent to consider that a 50% fall in Hb can be compensated by a doubling of cardiac output, provided circulating volume is maintained. In addition, although similar big swings in SaO_2 are rarely observed, a 15% reduction to 85% is not uncommon in the postoperative period and would have the same effect as the loss of 1 to 2 units blood. It is therefore important to maintain (or increase) cardiac output and oxygenation, as a priority, in patients experiencing blood loss. Only when blood loss is likely to result in a fall in Hb to below 80g.l-1, or when there are limitations on the ability of the patient to increase cardiac output should it be replaced. Although blood may not be required until 20% or more of the blood volume is lost (in a healthy patient) it is obviously necessary to replace the fluid component so that preload and thus cardiac output can be maintained.

Please also note that 1000 ml.min-1 of oxygen can only be delivered to the tissues if a similar amount is being delivered to the alveoli. This is normally achieved by an alveolar ventilation of 5 l.min-1 and an oxygen concentration of 21%. If oxygen demand is increased then more oxygen will have to be delivered to the alveoli. This can be achieved by increased ventilation, increased oxygen concentration (O_2 supplementation) or a combination of the two.

Three Special Problems

Dehydration

This is a common situation meaning depletion of water, nearly always accompanied by Na^+ depletion. Clinically, the symptoms and signs are: Thirst, dry mucosae, loss of elasticity of the skin, fall in urine output, collapsed veins, cold extremities and tachycardia. This is a common situation in the postoperative patient after major surgery. It is usually due to inadequate quantities of isotonic Na^+ containing fluids being given to compensate for continuing 'third space' losses. The important findings are:

- a low urine output of < 0.5 ml.kg-1 per hour in the adult
- low plasma Na^+ (e.g. 125 to 130 mmol.l-1)
- high urine osmolality (> 600 or at least 2:1 urine/plasma osmolality ratio)
- low urinary Na^+ (less than 20 mmol.l-1) due to Na^+ retention as a result of continuing 'third space' loss

- a low central venous pressure (CVP)
- the Hb concentration may be relatively normal due to haemoconcentration

Do not give diuretics in this situation (unless the patient is on regular diuretic therapy) as it will only exacerbate the hypovolaemia. Normal saline or Ringer's Lactate should be used for initial replacement. Begin with a 10 - 20 ml.kg-1 bolus over 5 minutes and follow this with a rate of 10 ml.kg-1 per hour monitoring the signs listed above at hourly intervals. Reduce to maintenance levels when the above signs are reversed. 4% glucose 0.18% NaCl may be used in addition if the blood Na+ concentration is higher than 140 mmol.l-1

Potassium depletion

Chronic depletion is commonly seen in the ageing hospital population who have been chronically treated with diuretics for hypertension or cardiac failure. Plasma K+ starts to fall below the normal minimum of 3.5 mmol l-1 only after 10% of total body K+ has been lost (400 mmol). A good additional indicator of depletion is a high plasma bicarbonate value (>28 mmol l-1), associated with acid urine.

Depletion of 400-600 mmol causes intracellular acidosis as hydrogen ions enter the cells to maintain ionic equilibrium. Losses should be replaced over several days with oral supplements, and the underlying cause corrected.

If the patient is unable to take oral supplements K+ must be given intravenously. It is administered as KCl, and concentrations of more than 20 mmol l-1 in Normal saline or 5% glucose cause pain and thrombosis in peripheral veins. Rapid replacement is rarely advisable. If absolutely necessary, it is given through a CVP line (preferably in the High Dependency or Intensive Care Unit, with continuous ECG monitoring and frequent plasma K+ measurements.

Water intoxication

This rarely occurs, but has dramatic consequences due to hyponatraemia. The cause is usually iatrogenic. Other causes include compulsive water drinking, transurethral resection of the prostate with excessive absorption of glycine irrigation fluid, inappropriate secretion of ADH and a few rare medical disorders.

Over prescription of intravenous 5% glucose and 4% glucose 0.18% NaCl solutions is the main iatrogenic cause. This was a not infrequent in labour wards. A fall in plasma Na+, as stated above, leads to a movement of water from the hypotonic PV into the ISF and ICF to maintain osmotic equilibrium. This expansion of ICF can lead to cerebral oedema, mental disturbances and

convulsions if plasma Na+ falls rapidly. Frusemide (0.5 mg kg-1) followed by 0.9% or 1.8% NaCl intravenously is an effective treatment of this emergency; a urinary catheter is needed. The speed of correction must be tailored to the speed on onset, chronic changes being corrected slowly.

Daily fluid requirement of our body:-

Insensible fluid input = 300ml (through oxidation)

Insensible fluid loss = 500ml (skin)

=400ml(lung)

=100ml(stool)

Daily insensible fluid loss= Fluid loss-Fluid input= 1000ml-300ml= 700ml

Normal daily fluid requirement = urine output(1600ml) + 700ml

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Assessment of Prakriti in Neonates: A New Approach

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ABSTRACT

Prakriti can be classified according to the predominance of the Dosha viz. Vata, Pitta, Kapha which are considered as the three important constituents of the livingorganism. The sample was drawn from I.P.D. of S.S. Hospital. All the newborns were delivered normal, without any history of perinatal or Post natal hypoxia, and having good Apgar score. All these neonates were exposed to the pre designed performa, based on the texual references available in ancient Ayurvedic texts. Total items were eleven.

As per data collected in the present study which was further exposed to statistical analysis the following facts emerged: Among 32 neonates of Vatal & Prakriti, 75% (24) were having blackish complexion whereas 25%(8) being pinkish and none having whitish complexion.

Introduction: Prakriti can be classified according to the predominance of the Dosha viz. Vata, Pitta, Kapha which are considered as the three important constituents of the living organism. Every individual has some specific physical and mental qualities and that is considered as the Prakriti. It can be defined as the state of the body which is used to denote the psychobiological make-ups of an individual.

Key words: Prakriti, Tridosha, Dhatu, Neonates, Dwandaj

Material and Methods: The sample was drawn from I.P.D. of S.S. Hospital. All the newborns were delivered normal, without any history of perinatal or Post natal hypoxia, and having good Apgar score. Mother was free from high risk condition. There height was more than 140 cm, weight was more than 40 kg with good nutritional status, they were free from pre eclampsia, placental previa, Rh isoimmunization, New born were free from hypoxic ischemic encephalopathy, and were free from cleft lip and cleft palate, as well as no presence of tracheo esophageal fistual or esophageal atresia. These new born were not meconium stained. All these neonates were exposed to the pre designed performa, based on the texual references available in ancient Ayurvedic texts. Total items were eleven.

As per data collected in the present study which was further exposed to statistical analysis the following facts emerged: Among 32 neonates of Vatal & Prakriti, 75% (24) were having blackish complexion whereas 25%(8) being pinkish and none having whitish complexion. Inter-group correlation however revealed no significant difference between the three complexions. Further sub-classification of these three complexions (three in each) revealed that 63.6% Vatal Prakriti infants were having black complexion and 75% presenting rosy complexion (among those having

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pinkish complexion). However, Z scores revealed highly significant (<0.001) correlation with Vatal Prakriti neonates having black complexion (Table I-a,b,c). Neonates of Pittal Prakriti (86.36%) had Pinkish complexion, where as 9.09% newborns were whitish.

However, inter-group statistical correlation revealed no significant co-relation of complexion. Rosy complexion had dominating over pink and coppery complexion in Pittal Prakriti neonates, thus indicating prevalence of rosy complexion in Pittal Prakriti subjects.These observations were further authenticated by significant p value of Z scores (Table I-c). Whitish complexion had been noticed in 54.05% of Kaphal Prakriti neonates which was further confirmed by significant values (< 0.05) in inter-group correlation. Further analysis of whitish complexion, in relation to Kaphal Prakriti revealed white complexion among neonates (42.5%) in comparison to offwhite and alba colored neonates. As regards Dwandaj Prakriti no significant correlation with complexion and Prakriti was observed in inter-group correlation except in Vata-Kaphal Prakriti neonates. However, Z score values revealed significant co-relation between Vatal, Pittal and Pitta-Kaphal Prakrities (Table I-c). These findings indicate near definite relation of blackish complexion with Vata Prakriti individuals, and rosy as well as Pinkish with Pittal Prakriti neonates. Off white and white complexions among whitish complexion neonates (on sub-classification) in Kaphal Prakriti neonates. Findings of the present study also indicated that more incidence/prevalence of association with complexion in Dwandaja Prakriti subjects in comparison to gross individual Prakriti. Although Pittal Prakriti neonates also revealed correlation of complexion, but Kaphal Prakriti infants were having highest correlation with complexion (29.83%) in the cumulative sample of neonates. Statistical analysis as based on Z scores revealed association of complexion with all types of Prakrities except Kaphal, Vata-Kaphal and Pitta-Kaphal Prakriti neonates (Table I-c).

Body Built : Thin built neonates were maximum (75%) and the rest (25%) being flabby. Medium built neonates were found not found in the study (Table II-a). Among 32 Vatal Prakriti neonates, 24 (75%) were of thin built and 25% being flabby. However, out of thin built neonates, 66.66% neonates were found moderately thin whereas among flabby neonates 75% were having flabby built (Tabe II-a,b). Pittal Prakriti neonates were of medium body built (70.80%) which was further confirmed by significant statistical values on inter-group comparison (TableII-a). Sub classification of medium body built neonates of Pittal Prakriti revealed 70.8% (34) neonates (out of 48 individuals) possessed general body built whereas all flabbies (14%) were of Kaphal Prakriti. Mild flabby (41.66%) neonates were mildly thin however 47.22% neonates were moderately thin (Table II-a,b). Among Kaphal Prakriti neonates, 68% were flabby. The incidence was further confirmed by highly significant Z score values (Table II c). Inter-group correlation revealed highly significant p values ($p<0.001$). Further sub-classification of flabby neonates revealed that 68% of them were flabby whereas 22% being mildly flabby (Table II-b). As regards Dwandaj Prakriti, neonates (both VP and VK) had dominance of this built. However, Pitta-Kaphal Prakriti neonates (71.8%) were found to be having medium body built. Z score (496 neonates) revealed statistically significant correlation with body built except Pittal Prakriti neonates (Table II-c).

Cry: The present study revealed husky cry in 87.5% neonates although relation not being statistically significant on inter-group correlation (Table III-a). Similarly, low pitched cry had been found in majority (80.06%) of neonates of Pittal-Prakriti but findings not being statistically significant. Heavy pitched voice (cry) was observed in 75.67% neonates of Kaphal-Prakriti (112 out of 148 neonates) being statistically significant on inter-group correlation. As regards Dwandaj Prakriti neonates, no definite correlation could be established between cry and Prakriti. Husky cry, which was present in 87.5% neonates of Vata-Pittal Prakriti. Excessively husky cry could be noticed where 80% (60) had low pitched cry, whereas 93.7% neonates were having mildly heavy cry. As regards Dwandaj Prakriti, no definite correlation emerged. However, in all the three Prakrities, the type of cry was found significant statistically on Z-scores (Table III-c).

Activity: Vatal Prakriti neonates were found active (62.5%). On inter-group correlation it was further confirmed by significant statistical values. Sub-classification of activity further revealed that among Vatal Prakriti infants, 55% were highly active and 35% being very active (Table-IV a,b). Similarly, moderate activity was found in Pittal Prakriti (54.54%) neonates and rest of the infants (45.45%) being slightly sluggish (Table IVa). As regards Kaphal Prakriti, 72.97% neonates were sluggish and very less percentage (36.19%) neonates being moderately active. However, out of 108 neonates, 72.20% neonates (78) were found slight sluggish and only 7.4 %(8) being highly sluggish (Table IV b). Inspite of being normal on all parameters of health and also having no sign of intra-uterine infection. However, Dwandaja Prakriti neonates exhibited variable picture and inter-group correlation revealed no definite association as evident from Table IV a and IV b. As regards Z scores on activity, except Vatal and Pittal Prakriti neonates, values were found highly significant (Table IV c).

Appetite: Vatal Prakriti neonates revealed low appetite (75%) in the present study (Table V a), however Pittal Prakriti neonates were having excessive appetite. In the present study 63.63% neonates emerged having Pittal Prakriti and having excessive appetite, whereas 27.27% neonates revealed moderate appetite (Va). Significant statistical values on inter-group correlation revalidated these facts (Table -Va). As per sub-classification of appetite, 8.92% (5) out of 56 subject of Pittal Prakriti had voracious appetite and only 9.09% (8) having low levels of appetite. As regard Kaphal Prakriti neonates, moderate appetite was noticed in 67.56%. Inter-group correlation between group I vs III revealed moderate appetite with significant correlation (Table V b). As regard Dwandaja Prakriti neonates, Vata-Pittal Prakriti subjects had statistically significant low appetite. Similar statistical analysis could be noticed in Pitta-Kaphal Prakriti neonates who were having moderate appetite (Va). Further sub-classification of appetite exhibited various levels of appetite in Pittal and Vata-Pittal. Prakriti neonates and very low appetite was found mostly in Vatal Prakriti neonates. The Z score values on appetite, in relation to Prakriti, revealed high correlation between Prakriti and status of appetite (Table V-c).

Stool type: Data analysis on the parameter reveled that Vatal Prakriti neonates, were constipated however 62.5% Vatal Prakriti newborns having constipated stool. No neonate of Vatal Prakriti was passing normal stool (Table VI a). Further analysis of types of stool in neonate's reveled 55% neonates (11 out of 20) passed very hard stool

and 75% of them (9 out of 12) passed occasional solid stool. As regards Pittal Prakriti neonates, 72.72% were having altered stools and only 27.27% passing normal stool. Among neonates having normal stool, 91.16% was passing occasional loose stool, whereas 70.2% Kapha Prakriti neonates passed normal stools, and also revealed incidence of occasional loose stools in 58.6% (61 out of 104) (Table VI-b). Among Dwandaja Prakriti neonates of Vata Kaphal and Pitta Kaphal Prakriti, significant correlation of stool with the Prakriti could be noticed (Table VI- a,b). The Z score values, related to type of stool, had revealed significant correlation between various Prakritis and types of stool (Table VI-c) except neonates of Vatal Prakriti neonates.

Sleep: Sleep was found disturbed among Vatal Prakriti neonates in comparison to neonates of other Prakrities. Levels of sleep was been found statistically significant when inter-group correlation was attempted (Table VII-a). Moderate sleep was observed in neonates of Pittal Prakriti, in which 68.18% neonates had experienced moderate sleep. Inter-group correlation, between I & II, groups revealed significant difference. Out of 148 neonates of Kaphal Prakriti, 59.45% (88) revealed excessive sleep whereas 27.02% (40) infants also revealed significant p values (<0.001) on inter-group correlation denoting excessive sleep in Kaphal Prakriti neonates. As regards Dwandaja Prakriti neonates, all the three constitutions revealed unstable sleep (Table VIIa andb). Further sub-classification of sleep revealed unstable sleep in Vatal Prakriti neonates usually less than 6 hour per day. Majority (68.18%) of neoantes of Pittal Prakriti had normal sleep whereas 75% of Kaphal Prakriti neonates had more than 12 hour sleep per day. As regards Dwandaja Prakriti, mixed picture emerged (Table VII-b). The Z values in terms of sleep further authenticated the relationship as values being highly significant on all Prakrities.

Hunger: Kaphal Prakriti neoantes revealed normal to severe status of hunger which was further reflected on inter-group correlation (Table VIII-a,b). However, Dwandaja Prakriti neonates revealed mixed picture on inter-group correlation. Above-average status of hunger was found in Vatal Prakriti; varied in Pittal Prakriti and below normal in Kaphal Prakriti neonates. The Z scores on all Prakrities were found significant (<0.001) except in Pittal Prakriti neonates (Table VIII-c). However, Dwandaja Prakriti neonates revealed no equivocal picture (Table VIII a,b)

Reaction to bed wetting : Pittal Prakriti neonates revealed moderate to normal reaction to bed wetting however higher values were found in moderate reaction to bed wetting in Pittal Prakriti, whereas in Kaphal Prakriti, normal reaction was noticed (Table IX a). Dwandaja Prakriti neonates did not reveal any definite correlation (Table IX a). Further sub-classification on the parameter, revealed no consistent relation because incidence being very variable (Table IX b). The Z score, on reaction to bed wetting revealed close relation of reaction to bed wetting with all types of Prakrities except Vata Prakriti neonates (Table IX c).

Weight Gain : Vatal Prakriti neonates had revealed low gains in weight; moderate in Pittal Prakriti and high in Kaphal Prakriti whereas among Dwandaja Prakriti neonates, varied picture emerged. Further sub-classification of these parameters revealed low to inconsistent weight gains in Vatal Prakriti neonates. Average to high gains in weight in Pittal Prakriti and high to moderately high in Kaphal Prakriti. Whereas inconsistent in

Vata-Kaphal Prakriti, more than average in Vata-Pittal and very low in Pitta-Kaphal Prakriti neonates was found (Table IX b). The Z scores of all the Prakrities revealed significant correlation with all the Prakrities except in neonates of Vatal and Pittal Prakrities. (Table IX-c).

Reaction to External environment: Quick reaction to external environment among Vatal Prakriti neonates, in the present study, was noticed. Association was found highly significant statistically on inter-group correlation (Table IX a). Similarly, moderate reaction was observed in Pittal Prakriti neonates where 77.27% neonates revealed moderate reaction to external environment which was further confirmed by inter-group statistical correlation (Table IX-a); whereas slow reaction was noticed in Kaphal Prakriti neonates, which was further confirmed by inter-group correlation. As regards Dwandaja Prakriti neonates, no definite picture emerged but moderate reaction was noticed in Pitta-Kaphal Prakriti neonates (Table IX a). Further sub-classification revealed quick reaction in 78.5% neonates of Vatal Prakriti; 54.4% had moderate reaction in Pittal Prakriti group however 40% neonates exhibited very slow response among Kaphal Prakriti group. As regards Dwandaja Prakriti, 57.14% neonates of Vata Pittal Prakriti showed quick response; 59.37% neonates exhibited slightly moderate response in Vata-Kaphal Prakriti and 42.04% in Pitta-Kaphal Prakriti revealed average to moderate response to external environment (Table XI b). Overall assessment revealed that Vata Prakriti neonates had the tendency to react quickly to the external environment than neonates of other Prakrities. The Z scores, on reaction to external environment, exhibited close relation with all the Prakrities except Vata-Kaphal Prakriti neoantes (Table IX c).

Table Ia : Showing inter group statistical analysis of **'Complexion' (n=496)**

Group (Prakriti)		Blackish	Pinkish	Whitish
		a Vs b	b Vs c	a Vs c
(A)	(n=32) 6.45%	n= 24 (75%)	n= 8 (n-25%)	n= 0
	M	1.50	1.38	0
V	S.D.	±0.78	±0.74	0
	t & p	0.38, >0.05	-	-
(B)	(n=88) 17.74%	n= 4 (4.54%)	n= 76 (86.36%)	n= 8 (9.09%)
	M	1.50	1.32	1.25
P	S.D.	±0.58	±0.64	±0.46
	t & p	0.55, >0.05	0.30 , >0.05	0.82 , >0.05
(C)	(n=148) 29.83%	n= 40 (27.03%)	n= 28 (18.9%)	n= 80 (54.05%)
	M	1.65	1.61	2
K	S.D.	±0.83	±0.88	±0.76
	t & p	0.19, >0.05	**2.24 , < 0.05 S**	**2.31, < 0.05 S**
(D)	(n=48) 9.67%	n= 20 (41.66%)	n= 24 (50%)	n=4 (8.33%)
	M	1.2	1.25	1.00
VP	S.D.	±0.52	±0.61	±0
	t & p	0.30, >0.05	0.81, > 0.05	0.76, >0.05
(E)	(n=80) 16.12%	n= 28 (35%)	n= 36 (45%)	n=16 (20%)
	M	1.43	1.11	1.56
VK	S.D.	±0.74	±0.40	±0.63
	t & p	**2.21, <0.05 S**	**3.12, <0.01 S**	0.59, >0.05
(F)	(n=100) 20.14%	n= 0	n=92 (92%)	n=8 (8%)
	M	0	1.22	1.25
PK	S.D.	0	±0.55	0.46
	t &p	-	0.15 , >0.05	-

Table IIa : Showing inter group statistical analysis of **Body Built' (n=496)**

Prakriti		Thin	Medium	Flabby
		Group I Vs II	**Group II Vs III**	**Group I vs III**
(A)	(n=32) 6.45%	n= 24 (75%)	n=0 (0%)	n= 8 (25%)
	M	1.91	±0	1.25
V	S.D.	±0.58	-	±0.46
	t & p	-	-	**2.92, <0.01S**
(B)	(n=88) 17.74%	n= 36 (40.90%)	n= 48 (54.54%)	n= 4 (4.54%)
	M	1.69	1.95	1.0
P	S.D.	±0.66	±0.54	±0
	t & p	1.98 , >0.05	**3.49 ,< 0.01 S**	**2.07, <0.05 S**
(C)	(n=148) 29.83%	n= 28 (18.91%)	n= 20 (13.5%)	n= 100 (67.56%)
	M	1.21	1.4	1.88
K	S.D.	±0.41	±0.68	±0.55
	t & p	1.21, > 0.05	**3.42 ,< 0.001S**	**5.99 ,<0.001 HS**
(D)	(n=48) 9.67%	n= 32 (66.6%)	n= 12 (25%)	n=4 (8.33%)
	M	1.59	1.33	1
VP	S.D.	±0.75	±0.49	±0
	t & p	1.11, >0.05	1.32, > 0.05	1.55, >0.05
(E)	(n=80) 16.12%	n= 64 (80%)	n= 8 (10%)	n=8 (10%)
	M	1.32	1.12	1.5
VK	S.D.	±0.53	±0.35	±0.53
	t & p	1.08, > 0.05	1.69, > 0.05	0.87, >0.05
(F)	(n=100) 20.14%	n= 0 (0%)	n=96 (96%)	n=4 (4%)
	M	0	1.32	1.25
PK	S.D.	±0	±0.55	±0.5
	t &p	-	0.25, >0.05	-

Table IIIa : Showing inter group statistical analysis of **'Type of Cry' (n=496)**

Prakriti	**Husky**	**Pitched**	**Heavy**
	Group I Vs II	**Group II Vs III**	**Group I vs III**
(A) (n=32) 6.45%	n= 28 (87.5%)	n=4 (12.5%)	n= 0
M	2.03	1.5	
V S.D.	±0.692	±0.577	±0
t & p	1.46, >0.05	-	-
(B) (n=88) 17.74%	n= 12 (13.63%)	n= 60 (68.18%)	n= 16 (18.18%)
M	1.16	1.25	1.06
P S.D.	±0.38	±0.653	±0.25
t & p	0.25 ,>0.05	1.11, >0.05	0.84 , > 0.5
(C) (n=148) 29.83%	n= 28 (18.9%)	n= 8 (5.4%)	n= 112 (75.67%)
M	1.03	1.125	1.33
K S.D.	±0.18	±0.653	±0.494
t & p	0.70 ,> 0.05	1.36, > 0.05	**3.15 ,< 0.01 S**
(D) (n=48) 9.67%	n= 28 (58.33%)	n= 20 (41.66%)	n=0
M	1.53	1.75	0
VP S.D.	±0.576	±0.716	±0
t & p	1.18 , >0.05	-	-
(E) (n=80) 16.12%	n= 28 (35%)	n= 8 (10%)	n=44 (55%)
M	1.25	1.12	1.5
VK S.D.	±0.518	±0.35	±0.628
t & p	0.66 , > 0.05	1.66 ,> 0.05	1.76 ,>0.05
(F) (n=100) 20.14%	n= 4 (4%)	n=72 (72%)	n=24 (24%)
M	1.25	1.26	1.29
PK S.D.	±0.5	±0.503	±5.50
t &p	0.04 ,> 0.05	0.25 ,>0.05	0.14 ,> 0.05

Table IVa : Showing inter group statistical analysis of **'Activity' (n=496)**

Prakriti	Active	Moderate	Sluggish
	Group I Vs II	**Group II Vs III**	**Group I vs III**
(A) (n=32) 6.45%	n= 20 (62.5%)	n=12 (37.5%)	n= 0
M	1.55	1.08	0
V S.D.	±0.686	±0	±0
t & p	**2.27, < 0.05 S**	-	-
(B) (n=88) 17.74%	n= 0	n = 48 (54.54%)	n=40 (45.45%)
M	0	1.58	1.25
P S.D.	±0	± 0.709	±0.543
t & p	-	**2.41, <0.02**	-
(C) (n=148) 29.83%	n = 28 (18.9%)	n=12 (8.10%)	n = 108 (72.97%)
M	1.03	1.46	0
K S.D.	±0.18	±0.621	±0
t & p	1.48, >0.05	-	-
(D) (n=48) 9.67%	n = 16 (33.33%)	n=32 (66.66%)	n=0
M	1.375	1.46	0
VP S.D.	±0.619	±0.621	±0
t & p	0.45, >0.05	-	-
(E) (n=80) 16.12%	n= 12 (15%)	n=0	n=68 (85%)
M	1.25	0	1.36
VK S.D.	±0.621	±0	±0.596
t & p	-	-	0.58, >0.05
(F) (n=100) 20.14%	n=0	n=16 (16%)	n=84 (84%)
M	0	1.625	1.452
PK S.D.	±0	±0.718	±0.66
t &p	-	0.94 , >0.05	-

Table Va : Showing inter group statistical analysis of **'Appetite' (n=496)**

Prakriti	**Low**	**Excessive**	**Moderate**
	Group I Vs II	**Group II Vs III**	**Group I vs III**
(A) (n=32) 6.45% M V S.D. t & p	n= 24 (75%) 1.83 ±0.816 1.90, >0.05	n=8 (25%) 1.25 ±0.462 -	n=0 0 ±0 -
(B) (n=88) 17.74% M P S.D. t & p	n=8 (9.09%) 1.5 ±0.755 1.43, >0.05	n=56 (63.63%) 1.82 ±0.970 **6.04 , <0.001 HS**	n=24 (27.27%) 1.08 ±0.282 **2.34, <0.05S**
(C) (n=148) 29.83% M K S.D. t & p	n=28 (18.91%) 1.32 ±0.669 0.71, >0.05	n=20 (13.5%) 1.2 ±0.774 2.94, >0.05	n=100 (67.56%) 1 ±0 **3.55, <0.01 S**
(D) (n=48) 9.67% M VP S.D. t & p	n=28 (58.33%) 2.28 ±.712 **2.30, <0.05S**	n=16 (33.33%) 1.75 ±0.774 1.90, >0.05	n=4 (8.33%) 1 ±0 **2.31, <0.01 S**
(E) (n=80) 16.12% M VK S.D. t & p	n=60 (75%) 1.23 ±0.499 -	n=0 0 ±0 -	n=20 (25%) 1.5 ±0.606 1.96, >0.05
(F) (n=100) 20.14% M PK S.D. t &p	n=36 (36%) 1.13 ±0.424 0.71, >0.05	n=8 (8%) 1.25 ±0.462 0.64, >0.05	n=56 (56%) 1.41 ±0.681 **2.21, <0.05 S**

Table VIa : Showing inter group statistical analysis of **Type of Stool (n=496)**

Prakriti		Constipated	Semi-solid	Normal
		Group I Vs II	**Group II Vs III**	**Group I vs III**
(A)	(n=32) 6.45%	n=20 (62.5%)	n=12 (37.5%)	n=0
	M	1.65	1.25	0
V	S.D.	±0.812	±0.452	±0
	t & p	1.56, > 0.05	-	-
(B)	(n=88) 17.74%	n= 0	n=64 (72.72%)	n=24 (27.27%)
	M	0	1.35	1.08
P	S.D.	±0	±0.675	±0.282
	t & p	-	1.89, >0.05	-
(C)	(n=148) 29.83%	n=16 (10.81%)	n=28 (18.9%)	n=104 (70.27%)
	M	1.25	1.392	1.6
K	S.D.	±0.447	±0.628	±0.793
	t & p	0.79 ,> 0.05	1.28, > 0.05	1.72, > 0.05
(D)	(n=48) 9.67%	n=4 (8.33%)	n=40 (83.33%)	n=4 (8.33%)
	M	1.25	1.25	1
VP	S.D.	±0.5	±0.543	±0
	t & p	0, >0.05	0.91, > 0.05	1.0, > 0.05
(E)	(n=80) 16.12%	n=20 (25%)	n=4 (5%)	n=56 (70%)
	M	1.15	1	2.01
VK	S.D.	±0.366	±0	±0.88
	t & p	0.81, > 0.05	**2.28, < 0.05 S**	**4.23, < 0.001 HS**
(F)	(n=100) 20.14%	n=0	n=12 (12%)	n=88 (88%)
	M	0	1.25	2
PK	S.D.	±0	±0.452	±0.802
	t &p	-	**3.16, < 0.05 S**	-

Table VIIa : Showing inter group statistical analysis of **Sleep (n=496)**

Prakriti		Disturbed	Moderate	Excess
		Group I Vs II	**Group II Vs III**	**Group I vs III**
(A)	(n=32) 6.45%	n=24 (75%)	n=8 (25%)	n=0
	M	2.2	1.25	0
V	S.D.	±0.883	±0.462	±0
	t & p	**2.89, < 0.01 S**	-	-
(B)	(n=88) 17.74%	n=4 (4.54%)	n=60 (68.18%)	n=24 (27.27%)
	M	2	1.25	1.68
P	S.D.	±0.816	±0.43	±0.61
	t & p	0.30, > 0.05	**3.21, < 0.011 S**	**2.78, < 0.01 S**
(C)	(n=148) 29.83%	n=20 (13.5%)	n=40 (27.02%)	n=88 (59.45%)
	M	1.2	1.25	1.68
K	S.D.	±0.52	±0.43	±0.61
	t & p	0.40, > 0.50	**4.02, < 0.01 HS**	**3.26, < 0.01 S**
(D)	(n=48) 9.67%	n=28 (58.33%)	n=20 (41.66%)	n=0
	M	2.39	1.7	0
VP	S.D.	±0.737	±0.73	±0
	t & p	**3.21, < 0.01 S**	-	-
(E)	(n=80) 16.12%	n=40 (50.00%)	n=8 (10%)	n=32 (40%)
	M	2.62	1.12	1.75
VK	S.D.	±0.625	±0.35	±0.842
	t & p	**6.55, < 0.001 HS**	**2.06, < 0.05 S**	**5.03, < 0.001 HS**
(F)	(n=100) 20.14%	n=8 (8%)	n=16 (16%)	n=76 (76%)
	M	1.12	2.18	1.47
PK	S.D.	±0.35	±0.834	±0.662
	t &p	**3.42, < 0.01 S**	**3.72, <0.01 HS**	1.47, > 0.05

Table VIIIa : Showing inter group statistical analysis of **Reaction to Hunger (n=496)**

Prakriti		Mild	Severe	Normal
		Group I Vs II	**Group II Vs III**	**Group I vs III**
(A)	(n=32) 6.45%	n=28 (87.5%)	n=0	n= 4 (12.5%)
	M	2.67	0	1.25
V	S.D.	±0.547	±0	±0.5
	t & p	-	-	**4.89, < 0.001 HS**
(B)	(n=88) 17.74%	n=12 (13.63%)	n=52 (59.09%)	n=24 (27.27%)
	M	1.08	1.35	2.489
P	S.D.	±0.28	±0.558	±0.687
	t & p	**7.68, < 0.001 HS**	**9.43, < 0.001 HS**	**0.90, < 0.001 HS**
(C)	(n=148) 29.83%	n=28 (18.91%)	n=28 (18.9%)	n=92 (62.16%)
	M	1.21	1.35	2.489
K	S.D.	±0.49	±0.558	±0.687
	t & p	0.99, > 0.05	**8.0, < 0.001 HS**	**9.15, < 0.001 HS**
(D)	(n=48) 9.67%	n=16 (33.33%)	n=24 (50%)	n=8 (16.66%)
	M	2.75	2.58	1.125
VP	S.D.	±0.447	±0.653	±0.353
	t & p	**0.91, < 0.02 S**	**5.97, < 0.001 HS**	**8.95, <.001 HS**
(E)	(n=80) 16.12%	n=40 (50%)	n=12 (15%)	n=28 (35%)
	M	1.625	1.08	1.64
VK	S.D.	±0.740	±0.28	±0.621
	t & p	**2.48, < 0.02 S**	**2.98, <0.01 S**	0.09, > 0.05
(F)	(n=100) 20.14%	n=8 (8%)	n=12 (12%	n=80 (80%)
	M	1.375	2.16	1.85
PK	S.D.	±0.744	±0.71	±0.85
	t &p	**2.38, < 0.05S**	1.20, >0.05	1.52, > 0.05

Table IXa : Showing inter group statistical analysis of **Reaction to Bed Wetting (n=496)**

Prakriti		Severe	Moderate	Normal
		Group I Vs II	**Group II Vs III**	**Group I vs III**
(A)	(n=32) 6.45%	n=16 (50%)	n=8 (25%)	n=8 (25%)
	M	2.124	1.25	0
V	S.D.	±0.718	±0.462	±0
	t & p	**3.12, < 0.01 S**	**7.65, < 0.001 HS**	**8.28, < 0.001 HS**
(B)	(n=88) 17.74%	n=12 (13.63%)	n=60 (68.18%)	n=16 (18.18%)
	M	1.66	1.91	1.125
P	S.D.	±0.778	±0.76	±0.341
	t & p	1.04, >0.05	**4.01, < 0.001 HS**	**2.48, < 0.05 S**
(C)	(n=148) 29.83%	n=28 (18.91%)	n=28 (18.91%)	n=92 (62.16%)
	M	1.39	1.214	1.94
K	S.D.	±0.628	±0.568	±0.73%
	t & p	1.10, > 0.05	**4.83, < 0.001 HS**	**3.59, < 0.001 HS**
(D)	(n=48) 9.67%	n=16 (33.33%)	n=24 (50%)	n=8 (16.66%)
	M	2.0	1.66	1.125
VP	S.D.	±0.894	±0.761	±0.353
	t & p	1.29, > 0.05	1.91, > 0.05	**2.64, < 0.02 S**
(E)	(n=80) 16.12%	n=28 (35%)	n=4 (5%)	n=48 (60%)
	M	2.07	1.25	1.85
VK	S.D.	±0.71	±0.5	±0.798
	t & p	**2.22, <0.05 S**	1.47, > 0.05	1.21, > 0.05
(F)	(n=100) 20.14%	n=0	n=0	n=100 (100%)
	M	0	0	1.79
PK	S.D.	±0	±0	±0.80
	t &p	-	-	-

Table Xa : Showing inter group statistical analysis of **Weight Gain (n=496)**

Prakriti		Low	Moderate	High
		Group I Vs II	**Group II Vs III**	**Group I vs III**
(A)	(n=32) 6.45%	n=20 (62.5%)	n=8 (25%)	n=4 (12.5%)
	M	2.2	1.625	1.5
V	S.D.	±0.767	±0.744	±0.577
	t & p	1.81, > 0.05	0.29, > 0.05	1.72, > 0.05
(B)	(n=88) 17.74%	n=20 (22.72%)	n=36 (40.90%)	n=32 (36.36%)
	M	1.3	2.25	1.14
P	S.D.	±0.571	±-0.731	±0.368
	t & p	**5.02, < 0.001 HS**	**7.69, < 0.001 HS**	1.15, > 0.05
(C)	(n=148) 29.83%	n=28 (18.91%)	n=20 (13.5%)	n=100 (67.56%)
	M	1.21	1.5	1.95
K	S.D.	± 0.568	±0.76	±0.796
	t & p	1.51, > 0.05	**2.32, < 0.05 S**	**4.60, < 0.001 HS**
(D)	(n=48) 9.67%	n=16 (33.33%)	n=28 (58.33%)	n=4 (8.33%)
	M	2.25	2.21	1.25
VP	S.D.	±0.774	0.875	±0.5
	t & p	0.15, > 0.05	**2.13, < 0.05 S**	**2.43, < 0.05 S**
(E)	(n=80) 16.12%	n=44 (55%)	n=16 (20%)	n=20 (25%)
	M	2.545	1.56	2.35
VK	S.D.	±0.761	±0.727	±0.67
	t & p	**4.48, < 0.001 HS**	**3.39, < 0.01 HS**	0.98, > 0.05
(F)	(n=100) 20.14%	n=28 (28%)	n=44 (44%)	n=28 (28%)
	M	1.28	2.25	2.14
PK	S.D.	±0.534	±0.781	±0.705
	t &p	**5.76, < 0.001 HS**	0.60, > 0.05	**5.15, < 0.001 HS**

Table XIa : Showing inter group statistical analysis of **Reaction to Ext. Environment (n=496)**

Prakriti		Quick	Moderate	Slow
		Group I Vs II	**Group II Vs III**	**Group I vs III**
(A)	(n=32) 6.45%	n=28 (87.5%)	n=0	n=4 (12.5%)
	M	2.71	0	1.25
V	S.D.	±0.59	±0	±0.5
	t & p	-	-	**4.63, < 0.001 HS**
(B)	(n=88) 17.74%	n=16 (18.18%)	n=68 (77.27%)	n=4 (4.54%)
	M	2.25	2.44	1.0
P	S.D.	±0.77	±0.47	±0
	t & p	1.26, > 0.05	**5.99, < 0.001 HS**	**3.16, < 0.01 HS**
(C)	(n=148) 29.83%	n=24 (16.2%)	n=4 (2.7%)	n=120 (81.08%)
	M	1.416	1.25	2.11
K	S.D.	±0.77	±0.5	±0.821
	t & p	0.41, > 0.05	**2.08, < 0.05 S**	**3.81, < 0.01 S**
(D)	(n=48) 9.67%	n=28 (58.33%)	n=16 (33.33%)	n=4 (8.33%)
	M	2.28	2.5	1.25
VP	S.D.	±0.89	±0.63	±0.5
	t & p	0.85, > 0.05	**3.65, < 0.01 S**	**2.24, <0.05 S**
(E)	(n=80) 16.12%	n=20 (25%)	n=32 (40%)	n=28 (35%)
	M	2.3	1.53	2.10
VK	S.D.	±0.80	±0.717	±0.87
	t & p	**3.60 , < 0.001 HS**	**2.78, < 0.01 S**	0.81, > 0.05
(F)	(n=100) 20.14%	n=4 (4%)	n=88 (88%)	n=8 (8%)
	M	1.5	2.05	2.12
PK	S.D.	±0.57	±0.763	±0.64
	t &p	1.42, > 0.05	0.25, > 0.05	1.63, > 0.05

Findings of the present study could not be matched with any other study because no such study specially related to neonates has been attempted documented. Whatever studies are available these pertain to adult group. However, these findings are very close to texual references available in Ayurvedic texts, however in relation to all age groups. Further attempts, to co-relate relation of different Prakrities in neonates with the parameter adopted in the present study are needed. The relation between the Prakriti and complexion had been found highly significant on the basis of parameters evaluated in each case.

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21st – 22nd Nov. 2009	New Delh INDIA	1st Annual Conference of Indian College of Anaesthesiologists	Tel: (O) +91-11-42252513 Fax: +91-11-25861002 E-mail: anaesthesiasgrh@gmail.com Web: www.sgrh.com
25th – 29th Dec. 2009	Chennai INDIA	57th Annual National Conference of ISA ISACON 2009	E-mail: isacon2009@hotmail.com Web: www.isacon2009chennai.com
9th – 13th Jan. 2010	Miami Beach USA	39th Critical Care congress of society of critical care medicine	Web: **www.sccm.org**
5th – 7th Feb. 2010	Udaipur INDIA	25th Annual National Conference of Indian society for study of pain	Tel: 0294-2560495, 09414160495 E-mail: bajajpramila@hotmail.com
10th – 14th Feb. 2010	Hyderabad INDIA	Criticare 2010 16th Annual conference of Indian society of critical care medicine	E-mail: **criticare2010@gmail.com** Web: www.ccriticare2010.org
12th 14th Feb. 2010	Tamil Nadu	IAPC Conference 2010 Trich	**www.palcantrichi.com** Mohan.sudharsana@gmail.com
26th – 28th Feb. 2010	Kolkata INDIA	IACTA Kolkata 2010	Tel: 09831256910 E-mail: iactakolkata@gmail.com Web: www.iactakolkata.com
22nd – 23rd April 2010	Manchester UK	7th annual critical care symposium	E-mail: **ct.veerappan@gmail.com** Veerappan.chithambaram@pat.nhs.uk Web: www.critcaresymposium.co.uk
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