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Comparative Evaluation of a No-Preload versus Ringer's Lactate, and 3.5% Polygeline as Preload; Effect on Haemodynamic and SpO_2 Variability following Spinal Anesthesia in Elective Lower-Abdominal or Lower-Limb Surgery in a Young Adult Non-Parturient Population.

First Author: Dr. Manish Sharma, DA, DNB, MBA (HCS), Assistant Professor, Department of Anaesthesiology, Dr. Y.S. Parmar Government Medical College, Nahan, Distt. Sirmaur, (H.P.), Pin Code: 173001; Mobile No.: 8219425617. Email: drmanish1966@gmail.com

Second Author: Dr. Ajay Sood, MD, Professor, Department of Anaesthesiology and Intensive Care, Indira Gandhi Medical College, Shimla, Distt. Shimla, (H.P.), Pin Code: 171001; Mobile No.: 9418028130. Email: ajay_5644@yahoo.com

Corresponding Author: Dr. Manish Sharma, Mobile No.: 8219425617. Email: drmanish1966@gmail.com

Subject: ANAESTHESIOLOGY

Full Address for Hard Copy with Name: Dr. Manish Sharma, s/o Lt. Col. Birbal Sharma (Retd.), H.No. 1731, Sector-33D, Chandigarh, Pin Code: 160020; Mobile: 8219425617. Email: drmanish1966@gmail.com

Abstract:

Background: To compare the haemodynamic and peripheral arterial oxygen saturation (SpO_2) variability among three groups, i.e. no preload, 3.5% polygeline preload and Ringer's lactate preload, after preloading and following spinal anaesthesia, in elective lower abdominal or lower limb surgery involving minimal blood loss in an adult non-parturient population.

Material and Methods: In a double-blind clinical trial, 75 ASA-I and ASA-II non-parturient adult patients undergoing elective lower abdominal or lower limb surgery were randomly allocated to three groups of 25 patients each, to receive either no preload, 3.5% polygeline solution (10 $mLkg^{-1}$), or Ringer's lactate solution (20 $mLkg^{-1}$), as a preload before spinal anaesthesia. Serial measurements of the haemodynamic parameters (such as the heart rate, systolic arterial pressure and mean arterial pressure) and the peripheral arterial oxygen saturation were compared on inter-group and intra-group basis, after preloading and following spinal anaesthesia.

Results: The SAP and MAP was significantly higher ($P \leq 0.05$) in the polygeline and Ringer's lactate groups, than in the no-preload group, after preloading, and following spinal anaesthesia (for a sustained duration in the polygeline group, and for a transitory duration in the Ringer's lactate group). The SAP and MAP in the polygeline group remained significantly higher ($P \leq 0.05$) than in the Ringer's lactate group for only intermittent short durations with no significant difference either after preloading or from 1-13 minutes following spinal anaesthesia. The difference in the incidence of spinal-induced hypotension (SIH) between the no-preload (68%), polygeline (24%) and Ringer's lactate (32%) groups was significant ($P=0.003$), with no significant difference between the polygeline and Ringer's lactate groups ($P=0.529$). The SpO_2 in the polygeline and Ringer's lactate groups remained

significantly higher than in the control group, after preloading ($P \leq 0.05$) and following spinal anaesthesia ($P=0.000$), with no significant difference between the polygeline and Ringer's lactate groups either after preloading or from 1 to 60 minutes following spinal anaesthesia.

Conclusion: The polygeline and Ringer's lactate preload compared to a no-preload maintained better haemodynamics and peripheral arterial oxygen saturation, after preloading and following spinal anesthesia, and reduced the incidence of SIH. There was no definite advantage of a polygeline preload over a Ringer's lactate preload.

Key words: Spinal-anaesthesia, SIH, no-preload, polygeline, Ringer's lactate, haemodynamic, SpO_2, variability, non-parturient, young adult, minimal blood loss.

INTRODUCTION

Hypotension during spinal anaesthesia is a common complication and can cause significant morbidity and mortality (1, 2) if not adequately and promptly treated. Most of the studies conducted in the past to ascertain the efficacy of a crystalloid or a colloid preload in the prevention of spinal-induced hypotension (SIH) have been conducted in the parturient population. The earlier studies conducted in the parturient population (3-5) established preloading as the single most important and effective method in the prevention of SIH. Various studies have propounded the superiority of a colloid preload over a crystalloid preload in the prevention of SIH and maintaining better haemodynamic stability following spinal anaesthesia in Caesarean section (6-8). One study (9) conducted in elective Caesarean section, who evaluated the effect of crystalloid and colloid preloading on uteroplacental and maternal haemodynamics found no significant statistical difference in the incidence of SIH with either a colloid or a crystalloid preload. Studies in the elderly population have yielded contradictory results (10-12).

Few studies (13) has analysed the effect of fluid preloading on cardiovascular variables after spinal anaesthesia in an adult (up to 70 yrs) non-parturient population. A number of studies have questioned the role altogether of preloading in reducing the incidence of SIH in the parturient population (14-17). The results of the previously conducted studies in the parturient and the non-parturient elderly population are contradictory and inconclusive and have failed to establish a simple, accurate and effective preloading regimen. Various confounding factors present in the earlier studies in the parturient (18-24) and the non-parturient elderly population (25, 26) known to affect the incidence of SIH are absent in the young adult (20-50 yrs) non-parturient population. Therefore, the extrapolation of these ambiguous results (obtained in the studies conducted in the parturient and the non-parturient elderly population) for the young adult non-parturient population would be extremely unreliable. The review of the literature reveals that few double-blind randomised controlled trials has been undertaken for the comparative evaluation of the effect of a no-preload, a colloid (3.5% polygeline) preload and a crystalloid (Ringer's lactate) preload, on the haemodynamic and the SpO_2 variability (both intra-group and intergroup), following spinal anaesthesia in elective lower abdominal or lower limb surgery, involving minimal blood loss in a young adult non-parturient population. We therefore compared a no-preload, 3.5% polygeline solution and Ringer's lactate solution as preload, in maintaining the haemodynamic and SpO_2 variability, after preloading, and following spinal anaesthesia in

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elective lower abdominal or lower limb surgery, involving minimal blood loss, in a young adult non-parturient population.

Material and Methods:

The study was planned in 75 ASA-I and ASA-II adult non-parturient patients, age 20–50 years, weight 50–65 kg, height 155–175 cm, undergoing elective lower abdominal or lower limb surgery, of duration less than or equal to one hour, involving minimal blood loss under spinal anaesthesia. Patients not willing for regional anaesthesia, sensitive to local anaesthetics, having previous history of postoperative nausea, vomiting and motion sickness, with abnormal coagulation profile, degenerative neurologic disease, localized infection (at puncture site), systemic infections, hypovolemia, previous history of low back pain, low cardio-respiratory reserve, haemoglobin < 10 gm% and in which spinal anaesthesia was absolutely contraindicated, were excluded from the study.

After obtaining the written informed consent from each patient and due ethical clearance from the Hospital Ethics Committee, the patients were randomly allocated to one of the three groups of 25 patients each, in a double-blind manner to receive either 1 mL min^{-1} Ringer's lactate solution to keep an intravenous cannula patent (control or no preload group, Group-I), 10 mL kg^{-1} 3.5% polygeline Haemaccel^{TM} (polygeline group, Group-II) or 20 mL kg^{-1} Ringer's lactate solution (Ringer's lactate or RL group, Group-III), intravenously, during a period of 20 minutes just before the induction of spinal anaesthesia. Randomization was performed in the preoperative/holding room using sealed envelopes (9).

The groups were comparable in physical characteristics and other demographic data (Table-1). All patients were premedicated orally with Tablet alprazolam 0.5 mg at 10 p.m. on the night before surgery and 0.25 mg orally two hours before spinal anaesthesia. All patients were fasted overnight for 6-10 hours, and no intravenous fluids were given prior to the infusion of the preloading fluid in the preoperative room (11).

The baseline measurements of heart rate (HR), systolic arterial pressure (SAP), mean arterial pressure (MAP) and the peripheral arterial oxygen saturation (SpO_2) were noted with Hewlett Packard patient monitor (No.REF M1205A B.0, address Hewlett Packard Gmbh, Boblingen, Germany) before preloading in the supine posture. An intravenous line was secured with a 16 gauge cannula. Nurses placed a brown paper bag over the plastic bottle containing the intravenous solution to conceal its identity from the Anaesthesiologist collecting data for the study (6). The patients of all the three groups received their respective infusions over a period of 20 minutes just before the induction of spinal anaesthesia. All four parameters were again recorded after the preloading was over.

In the operating room, spinal anaesthesia was administered using a midline approach, with a 24 gauge spinal needle at the L_3-L_4 intervertebral space in the right lateral decubitus position. After a free flow of cerebrospinal fluid was obtained, 3 mL of 0.5% hyperbaric bupivacaine was injected at the rate of 1 mL/5 seconds. The patient was returned to supine posture. Parameters of HR, SAP, MAP and SpO_2 were recorded at one-min interval for 15 minutes, and thereafter at 5-min intervals (and monitored visually every minute) till 60 min following spinal anaesthesia. Hypotension and/or bradycardia were noted even in between these 5-min intervals.

The maintenance fluid in the form of 5% dextrose 10 $mLkg^{-1}h^{-1}$ and oxygen 6 L min^{-1} (via ventimask) was administered during the period of 60 minutes following spinal anaesthesia. Hypotension was defined as a decrease in SAP of 25% or more from the before-preload recordings, and was treated promptly with incremental intravenous doses of 3 mg-mephentermine at 1-min intervals until the SAP returned to within 25% of baseline values. Bradycardia (heart rate < 50 bpm) was treated with intravenous atropine 0.6 mg. The patients were observed for any untoward cardiovascular side-effects such as bradycardia and arrhythmias.

The duration and type of surgery, volume of preload administered and sensory analgesia achieved at 15 min after induction of spinal anaesthesia were noted. The incidence of spinal-induced hypotension in all the three groups was recorded. The primary outcome measure in the study was the inter-group and intra-group haemodynamic and SpO_2 variability, whereas the secondary outcome measure was the incidence of SIH and cardiovascular side-effects such as bradycardia and arrhythmias.

Statistical Analysis:

The data was presented as mean (standard deviation) or frequency (percentage). The statistical analysis was performed using the IBM SPSS Statistics 26 statistical software on a standard computer. The inter-group comparison of the parametric data (the serial readings of HR, SAP, MAP and SpO_2) between the three groups was evaluated (inter-group analysis) with ANOVA one-way test. In case, a significant difference was found among the three groups, the *post-hoc* test (Tukey's HSD) was performed to find out, between which specific group(s) the significant difference lies. The intra-group (within the same group) comparison of parametric data, i.e. HR, SAP, MAP and SpO_2 (intra-group analysis) was compared with ANOVA Repeated measures (ANOVA-RM) one-way test. Non-parametric (categorical) data (Gender, ASA Grade, surgery type, incidence of SIH and cardio-vascular side effects) was evaluated with Pearson Chi-square test or Fisher's exact test (where applicable). Level of sensory analgesia among the three groups was analysed with ANOVA one-way test. The *P* values, i.e. $P \leq .05$, $P \leq .01$ and $P \leq .001$, were taken as significant, highly significant and very highly significant respectively in the study. The prospective power analysis in the study was conducted on the basis of the results, i.e. the difference in the incidence of spinal induced hypotension (SIH) between the colloid and the crystalloid group in the previous studies (6-8), wherein in order to detect a difference in the incidence of SIH between the colloid and crystalloid group of 40%, with 80% power and α (level of significance) =0.05, a group size of 21 patients would be needed. We calculated a drop-out rate of 10% and thus included 25 patients in each group.

A control or no-preload group was included in our study, so that it was not difficult to detect "regression to the mean" (random fluctuation through biological variation or measurement error, which leads to extremely high or low values) in the study, thus preventing biased selection (27). Rout and colleagues (28) observed that no volume preloading before induction of spinal anaesthesia may constitute a potentially detrimental management and therefore devised a sequential analysis design in their study, where minimum number of patients were exposed to such a treatment. Kennedy and others (29) observed almost 55 years back that acute untreated blood loss during subarachnoid block should be used with discretion. This is because the compensatory mechanism of arteriolar constriction fails during acute blood loss

in the vasodilated arteriolar vasculature (during spinal anaesthesia) thus producing a greater degree of hypotension. The above considerations were taken into account when planning our study. Consequently, adequate safeguards were adopted. Monitoring of all the cardiovascular parameters was done every minute after induction of spinal anaesthesia. On detection of hypotension, vasopressor administration was prompt till adequate restoration of blood pressure. Further, all the surgeries in our study involved minimal blood loss.

Observations and Results:-

The three groups were comparable in age, weight, height, duration of operation, type of surgery, ASA Grade, Gender Ratio and segmental level of sensory analgesia (Table-1). There was a significant difference (*P*=0.000) between the mean (SD) preload value administered to the polygeline and the Ringer's lactate group, i.e. 555 (9.76) mL vs. 1110.40 (18.69) mL.

TABLE-I: GENERAL CHARACTERISTICS OF PATIENTS

		GROUPS			
S. No.	PARAMETERS (MEAN) (S.D.)	CONTROL (*n* = 25)	POLYGELINE (*n* = 25)	RINGER'S LACTATE (*n* = 25)	*P* VALUE ON INTER-GROUP COMPARISON BETWEEN GROUP I, II AND III BY ANOVA ONE-WAY TEST
1	AGE (years)	39.52 (9.807)	38.64 (9.578)	33.88 (9.167)	*P*=0.086 (NS)
2	WEIGHT (kg)	54.88 (5.967)	55.50 (4.882)	55.52 (4.674)	*P*=0.885(NS)
3	HEIGHT (cm)	165.24 (4.861)	164.58 (6.067)	164.96 (5.295)	*P*=0.911(NS)
4	DURATION OF OPERATION (min)	50.00 (9.465)	48.40 (12.725)	49.60 (10.400)	*P*=0.145(NS)
					P VALUE ON INTER-GROUP COMPARISON BETWEEN GROUP II AND III BY STUDENT'S T -TEST
5	VOLUME OF PRELOAD INFUSION ADMINISTERED (mL)	0.00 (0.00)	555.00 (9.76)	1110.40 (18.69)	*P*=0.000***
					EVALUATION BY CHI - SQUARE TEST (df = 2)
					P VALUE χ2 VALUE
6	RATIO OF ASA I TO ASA II GRADE PATIENTS	18 : 7	21 : 4	23 : 2	0.171 (NS) 3.536
7	GENDER RATIO MALE: FEMALE	21 : 4	22 : 3	21 : 4	0.899 (NS) 0.213
8	THORACIC SENSORY LEVEL AT 15 MIN †	7.36 (2.361)	7.32 (2.231)	7.16 (2.322)	*P*=0.949 (NS) (BY ANOVA ONE-WAY TEST)

P (PROBABILITY) VALUES AT VARIOUS LEVELS OF SIGNIFICANCE (α): *P*>0.05: NOT SIGNIFICANT (NS), $P \leq 0.05$*: SIGNIFICANT, $P \leq 0.01$**: HIGHLY SIGNIFICANT AND $P \leq 0.001$***: VERY HIGHLY SIGNIFICANT S.D.: STANDARD DEVIATION, ALL VALUES ARE MEAN UNLESS OTHER-WISE SPECIFIED WITH S.D. IN PARENTHESES, n: NUMBER OF PATIENTS, GP. : GROUP; df: DEGREES OF FREEDOM.

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TABLE 2: INTER-GROUP COMPARISON OF THE SYSTOLIC ARTERIAL PRESSURE (in mmHg) CHANGES, AFTER PRELOADING, AND FOLLOWING SPINAL ANAESTHESIA, FOR A PERIOD OF 60 MINUTES.

POST HOC TEST (TUKEY'S HSD)

S.No.		CONTROL GROUP		POLYGELINE GROUP		RINGER'S LACTATE GROUP		*P* VALUE BETWEEN GP.I, II AND III *(ANOVA ONE- WAY)*	*P* VALUE BETWEEN GP.I AND II	*P* VALUE BETWEEN GP.I AND III	*P* VALUE BETWEEN GP.II AND III
		GROUP I		GROUP II		GROUP III					
		MEAN	S.D.	MEAN	S.D.	MEAN	S.D.				
1	B.P. †	122.04	10.895	121.12	11.414	121.04	9.418	0.934(NS)	0.920(NS)	0.941(NS)	1.000(NS)
2	A.P. ††	122.04	10.895	129.80	9.849	130.92	9.273	0.005**	0.021*	0.007**	0.918(NS)
3	1 MIN.	116.96	10.632	126.80	9.443	127.20	9.192	0.000***	0.002**	0.001***	0.989(NS)
4	2 MIN.	112.44	12.346	125.00	9.133	124.52	9.666	0.000***	0.000***	0.000***	0.986(NS)
5	3 MIN.	110.12	13.532	123.36	9.187	121.40	9.840	0.000***	0.000***	0.002**	0.805(NS)
6	4 MIN.	106.72	12.401	121.28	8.758	118.04	11.491	0.000***	0.000***	0.001***	0.553(NS)
7	5 MIN.	103.88	12.791	120.44	8.982	115.76	12.673	0.000***	0.000***	0.002**	0.334(NS)
8	6 MIN.	101.16	12.565	117.32	8.774	113.32	13.447	0.000***	0.000***	0.001***	0.456(NS)
9	7 MIN.	100.92	11.347	116.24	9.850	111.80	12.659	0.000***	0.000***	0.003**	0.355(NS)
10	8 MIN.	101.24	10.733	116.80	8.794	110.00	12.861	0.000***	0.000***	0.016*	0.078(NS)
11	9 MIN.	102.48	9.661	115.76	10.044	108.76	12.414	0.000***	0.000***	0.118(NS)	0.072(NS)
12	10 MIN.	101.80	8.010	114.76	11.414	108.24	12.794	0.000***	0.000***	0.100(NS)	0.095(NS)
13	11 MIN.	102.16	8.901	113.64	11.775	107.28	11.880	0.002**	0.001***	0.230(NS)	0.107(NS)
14	12 MIN.	102.20	9.725	112.56	10.928	106.96	10.830	0.004**	0.002**	0.252(NS)	0.151(NS)
15	13 MIN.	101.72	8.754	111.80	11.273	105.84	10.742	0.004**	0.003**	0.340(NS)	0.109(NS)
16	14 MIN.	103.12	10.944	113.20	10.324	104.48	10.500	0.002**	0.003**	0.893(NS)	0.013*
17	15 MIN.	101.04	9.868	112.48	10.674	103.04	10.687	0.000***	0.001***	0.777(NS)	0.006**
18	20 MIN.	100.28	9.935	111.20	10.966	102.02	9.251	0.001***	0.001***	0.625(NS)	0.013*
19	25 MIN.	100.36	8.316	111.48	11.644	104.76	8.852	0.002**	0.001***	0.252(NS)	0.101(NS)
20	30 MIN.	102.88	8.922	110.96	10.803	105.32	8.840	0.012*	0.011*	0.641(NS)	0.100(NS)
21	35 MIN.	103.28	9.745	110.28	12.085	104.96	10.346	0.063 (NS)	0.063 (NS)	0.846(NS)	0.195(NS)
22	40 MIN.	102.72	8.259	111.88	10.787	105.08	11.098	0.006**	0.006**	0.690(NS)	0.052(NS)
23	45 MIN.	101.60	7.714	111.84	11.796	104.80	9.005	0.001***	0.001***	0.474(NS)	0.032*
24	50 MIN.	100.16	9.818	111.72	12.167	104.52	6.850	0.000***	0.000***	0.268(NS)	0.031*
25	55 MIN.	99.24	9.084	112.60	7.687	104.88	8.263	0.000***	0.000***	0.051 (NS)	0.005**
26	60 MIN.	99.68	8.133	113.04	11.631	105.16	7.893	0.000***	0.000***	0.104 (NS)	0.011*

TABLE 3: INTER-GROUP COMPARISON OF THE PERIPHERAL ARTERIAL OXYGEN SATURATION (SpO_2) CHANGES, AFTER PRELOADING, AND FOLLOWING SPINAL ANAESTHESIA, FOR A PERIOD OF 60 MINUTES.

POST HOC TEST (TUKEY'S HSD)

S.No.		CONTROL GROUP		POLYGELINE GROUP		RINGER'S LACTATE GROUP		*P* VALUE BETWEEN GP.I, II AND III *(ANOVA ONE – WAY)*	*P* VALUE BETWEEN GP.I AND II	*P* VALUE BETWEEN GP.I AND III	*P* VALUE BETWEEN GP.II AND III
		GROUP I		GROUP II		GROUP III					
		MEAN	S.D.	MEAN	S.D.	MEAN	S.D.				
1	B.P. †	99.20	0.707	99.12	0.726	99.32	0.748	0.621 (NS)	0.920(NS)	0.829(NS)	0.597(NS)

2	A.P. ††	99.20	0.707	99.68	0.476	99.60	0.500	0.009**	0.011*	0.041*	0.874(NS).
3	1 MIN.	98.84	0.943	99.76	0.523	99.92	0.277	0.000***	0.000***	0.000***	0.655(NS)
4	2 MIN.	98.72	0.936	99.72	0.614	99.92	0.277	0.000***	0.000***	0.000***	0.541(NS)
5	3 MIN.	98.44	1.227	99.64	0.700	99.72	0.542	0.000***	0.000***	0.000***	0.944(NS)
6	4 MIN.	98.08	1.115	99.36	0.700	99.52	0.653	0.000***	0.000***	0.000***	0.784(NS)
7	5 MIN.	97.84	1.281	99.36	0.757	99.40	0.707	0.000***	0.000***	0.000***	0.988(NS)
8	6 MIN.	97.76	1.393	99.40	0.816	99.32	0.802	0.000***	0.000***	0.000***	0.960(NS)
9	7 MIN.	97.64	1.440	99.40	0.866	99.28	0.737	0.000***	0.000***	0.000***	0.915(NS)
10	8 MIN.	97.64	1.411	99.48	0.823	99.28	0.737	0.000***	0.000***	0.000***	0.774(NS)
11	9 MIN.	97.52	1.327	99.32	0.900	99.16	0.746	0.000***	0.000***	0.000***	0.845(NS)
12	10 MIN.	97.52	1.229	99.36	0.810	99.12	0.833	0.000***	0.000***	0.000***	0.661(NS)
13	11 MIN.	97.44	1.446	99.36	0.700	99.12	0.881	0.000***	0.000***	0.000***	0.703(NS)
14	12 MIN.	97.16	1.491	99.28	0.737	99.04	1.020	0.000***	0.000***	0.000***	0.733(NS)
15	13 MIN.	97.20	1.258	99.28	0.678	99.00	0.866	0.000***	0.000***	0.000***	0.563(NS)
16	14 MIN.	97.08	1.441	99.24	0.663	98.96	0.735	0.000***	0.000***	0.000***	0.591(NS)
17	15 MIN.	97.28	1.275	99.08	1.256	99.16	0.688	0.000***	0.000***	0.000***	0.965(NS)
18	20 MIN.	97.28	1.242	99.16	0.800	99.20	0.707	0.000***	0.000***	0.000***	0.988(NS)
19	25 MIN.	97.12	1.166	99.08	0.862	99.16	0.746	0.000***	0.000***	0.000***	0.952(NS)
20	30 MIN.	96.96	1.172	99.36	0.700	99.16	0.746	0.000***	0.000***	0.000***	0.712(NS)
21	35 MIN.	97.16	1.068	99.24	0.926	99.16	0.850	0.000***	0.000***	0.000***	0.953(NS)
22	40 MIN.	97.28	1.137	99.36	0.860	99.16	0.898	0.000***	0.000***	0.000***	0.748(NS)
23	45 MIN.	97.04	1.060	99.44	0.821	99.08	0.812	0.000***	0.000***	0.000***	0.343(NS)
24	50 MIN.	97.04	0.978	99.36	0.757	99.08	0.812	0.000***	0.000***	0.000***	0.482(NS)
25	55 MIN	96.96	0.935	99.44	0.651	99.04.	0.790	0.000***	0.000***	0.000***	0.188 (NS)
26	60 MIN	96.96.	0.935.	99.48	0.653	99.04	0.790	0.000***	0.000***	0.000***	0.134 (NS)

P (PROBABILITY) VALUES AT VARIOUS LEVELS OF SIGNIFICANCE (α): DIFFERENCE IS NOT SIGNIFICANT AT *P*>0.05: (NS), SIGNIFICANT AT $P \leq 0.05$*, HIGHLY SIGNIFICANT AT $P \leq 0.01$** AND VERY HIGHLY SIGNIFICANT AT $P \leq 0.001$*** *(COMPARED WITH THE CORRESPONDING VALUES OF OTHER GROUPS; ANOVA ONE-WAY TEST)*, MIN: MINUTE, S.D.: STANDARD DEVIATION, GP.: GROUP, †B.P.: BEFORE-PRELOAD, †† A.P.: AFTER-PRELOAD.

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FIGURE-1: INTER-GROUP COMPARISON OF THE HEART RATE CHANGES AFTER PRELOADING AND FOLLOWING SPINAL ANAESTHESIA FOR A PERIOD OF 60 MINUTES.

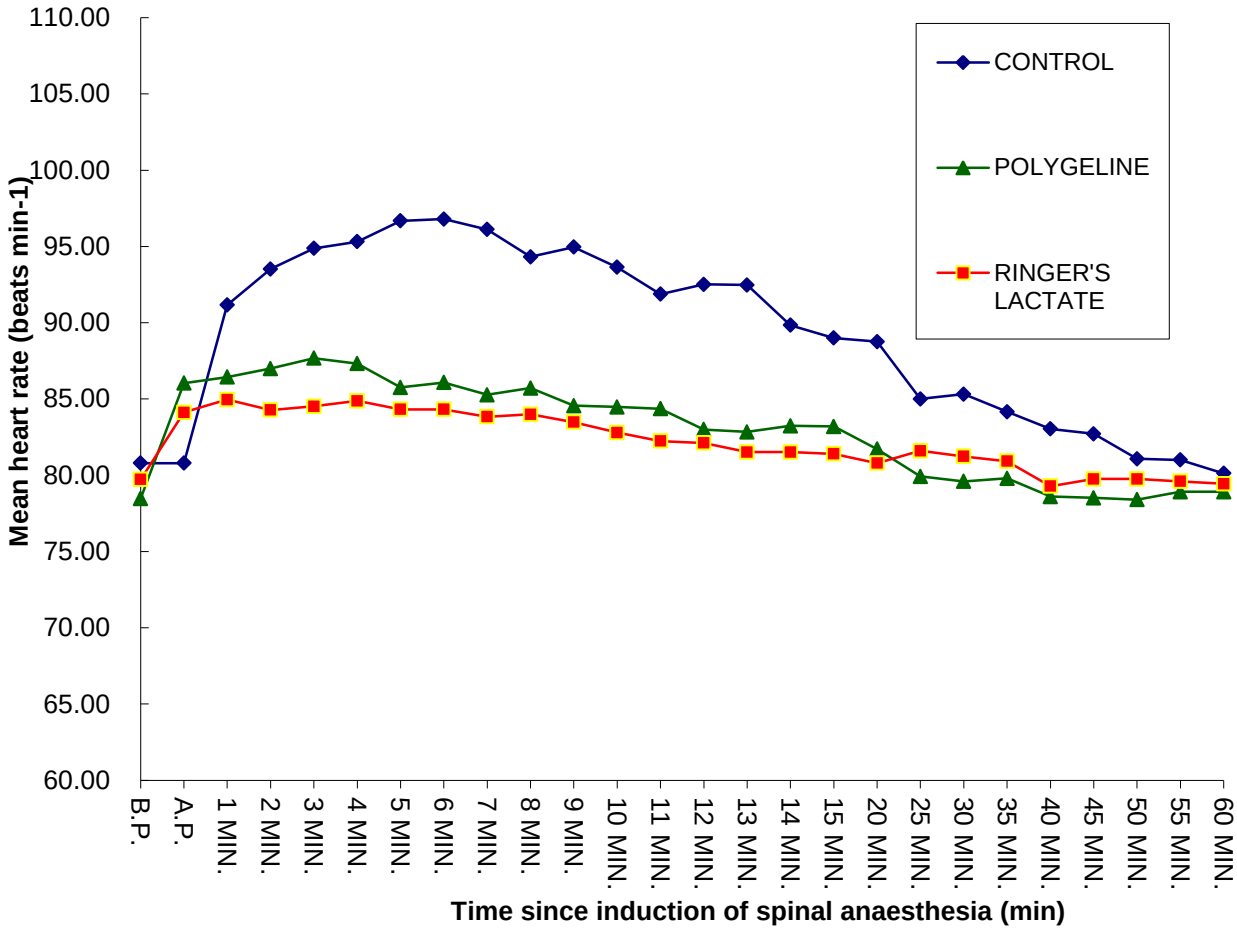

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FIGURE-2: INTER-GROUP COMPARISON OF THE SYSTOLIC ARTERIAL PRESSURE CHANGES AFTER PRELOADING AND FOLLOWING SPINAL ANAESTHESIA FOR A PERIOD OF 60 MINUTES.

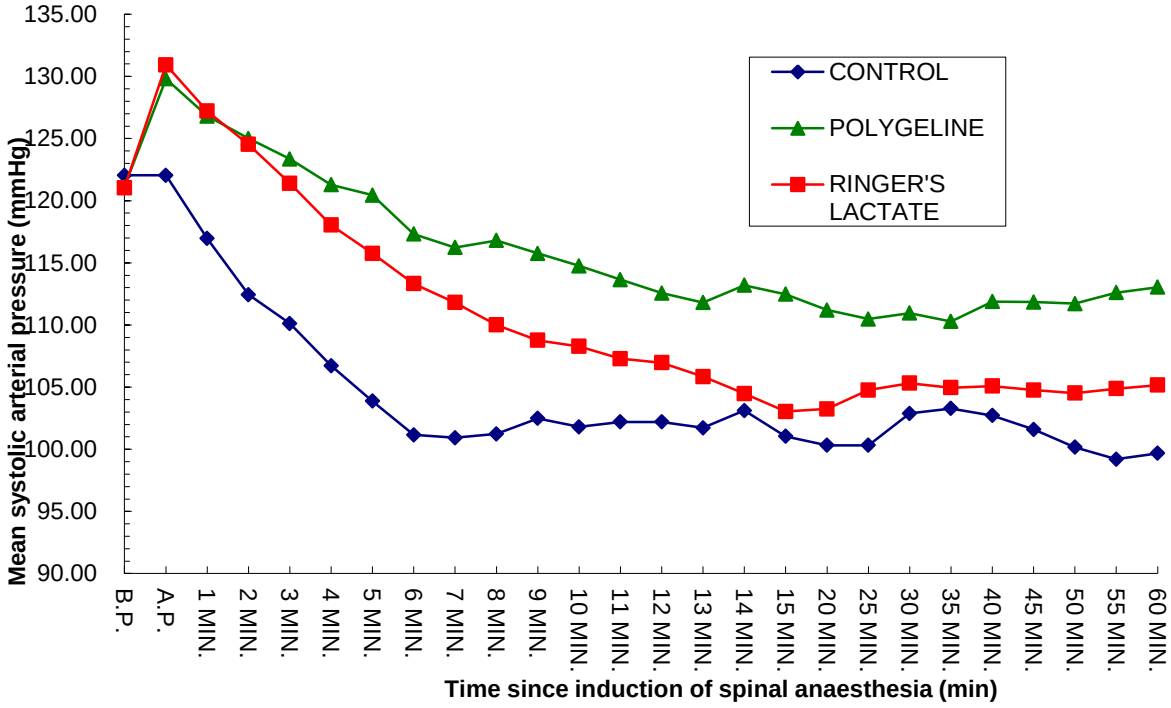

FIGURE-3: INTER-GROUP COMPARISON OF THE MEAN ARTERIAL PRESSURE CHANGES AFTER PRELOADING AND FOLLOWING SPINAL ANAESTHESIA FOR A PERIOD OF 60 MINUTES.

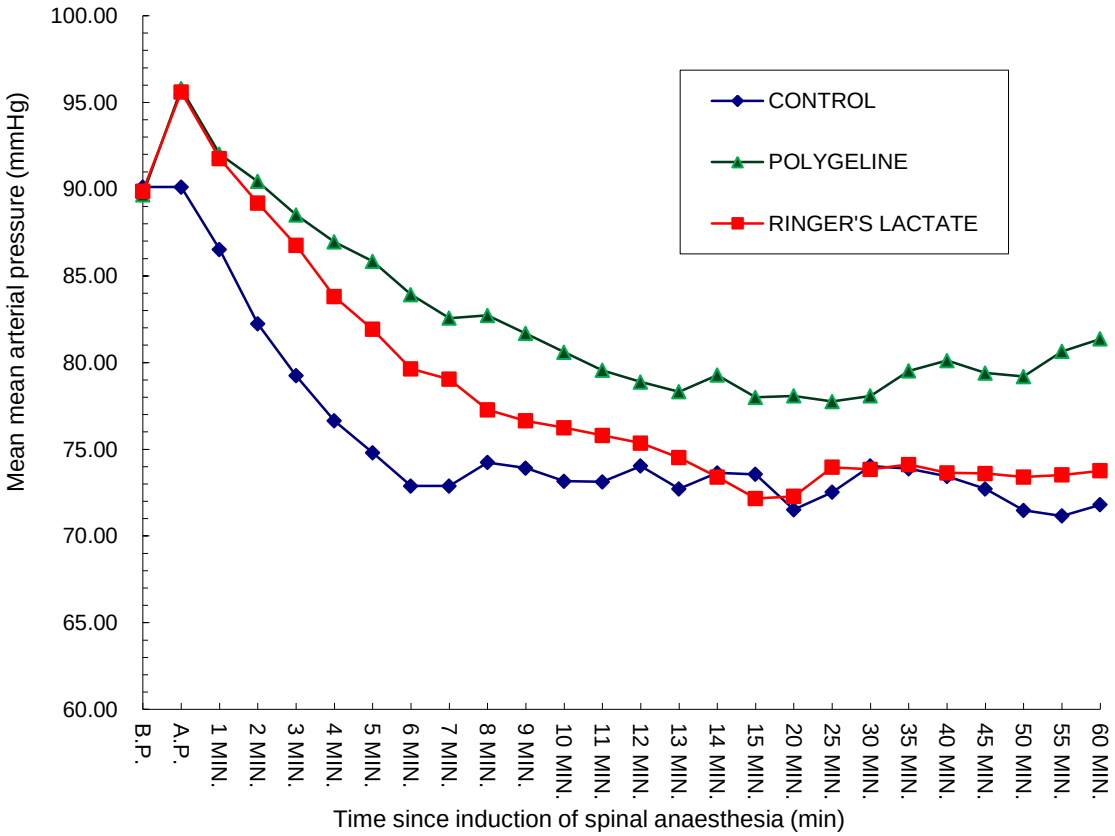

TABLE-4 : INTRA-GROUP COMPARISON OF THE SYSTOLIC ARTERIAL PRESSURE (in mmHg) CHANGES, AFTER PRELOADING, AND FOLLOWING SPINAL ANAESTHESIA, FOR A PERIOD OF 60 MINUTES.

		CONTROL GROUP			POLYGELINE GROUP			RINGER'S LACTATE GROUP		
		GROUP I			GROUP II			GROUP III		
S.No.		MEAN	S.D.	*P* VALUE	MEAN	S.D.	*P* VALUE	MEAN	S.D.	*P* VALUE
1	B.P. †	122.04	10.895	-	121.12	11.414	-	121.04	9.418	-
2	A.P. ††	122.04	10.895	-	129.80	9.849	0.000***	130.92	9.273	0.000***
3	1 MIN.	116.96	10.632	0.000***	126.80	9.443	0.000***	127.20	9.192	0.000***
4	2 MIN.	112.44	12.346	0.000***	125.00	9.133	0.006**	124.52	9.666	0.015*
5	3 MIN.	110.12	13.532	0.000***	123.36	9.187	0.120(NS)	121.40	9.840	0.804(NS)
6	4 MIN.	106.72	12.401	0.000***	121.28	8.758	0.920(NS)	118.04	11.491	0.089(NS)
7	5 MIN.	103.88	12.791	0.000***	120.44	8.982	0.697(NS)	115.76	12.673	0.013*
8	6 MIN.	101.16	12.565	0.000***	117.32	8.774	0.091(NS)	113.32	13.447	0.002**
9	7 MIN.	100.92	11.347	0.000***	116.24	9.850	0.055(NS)	111.80	12.659	0.000***
10	8 MIN.	101.24	10.733	0.000***	116.80	8.794	0.037*	110.00	12.861	0.000***
11	9 MIN.	102.48	9.661	0.000***	115.76	10.044	0.009**	108.76	12.414	0.000***
12	10 MIN.	101.80	8.010	0.000***	114.76	11.414	0.004**	108.28	12.794	0.000***
13	11 MIN.	102.20	8.901	0.000***	113.64	11.775	0.001***	107.28	11.880	0.000***
14	12 MIN.	102.20	9.725	0.000***	112.56	10.928	0.001***	106.96	10.830	0.000***
15	13 MIN.	101.72	8.754	0.000***	111.80	11.273	0.000***	105.84	10.742	0.000***
16	14 MIN.	103.12	10.944	0.000***	113.20	10.324	0.001***	104.48	10.500	0.000***
17	15 MIN.	101.04	9.868	0.000***	112.48	10.674	0.000***	103.04	10.687	0.000***
18	20 MIN.	100.32	9.935	0.000***	111.20	10.966	0.000***	103.24	9.251	0.000***
19	25 MIN.	100.32	8.316	0.000***	110.48	11.644	0.000***	104.76	8.852	0.000***
20	30 MIN.	102.88	8.922	0.000***	110.96	10.803	0.000***	105.32	8.840	0.000***
21	35 MIN.	103.28	9.745	0.000***	110.28	12.085	0.000***	104.96	10.346	0.000***
22	40 MIN.	102.72	8.259	0.000***	111.88	10.787	0.000***	105.08	11.098	0.000***
23	45 MIN.	101.60	7.714	0.000***	111.84	11.796	0.000***	104.76	9.005	0.000***
24	50 MIN.	100.16	9.818	0.000***	111.72	12.167	0.001***	104.52	6.850	0.000***
25	55 MIN.	99.20	9.084	0.000***	112.60	7.687	0.000***	104.88	8.263	0.000***
26	60 MIN.	99.68	8.133	0.000***	113.04	11.631	0.001**	105.16	7.893	0.000***

P (PROBABILITY) VALUES AT VARIOUS LEVELS OF SIGNIFICANCE (α): DIFFERENCE IS NOT SIGNIFICANT AT *P*>0.05 (NS): SIGNIFICANT AT $P \leq 0.05$*, HIGHLY SIGNIFICANT AT $P \leq 0.01$** AND VERY HIGHLY SIGNIFICANT AT $P \leq 0.001$*** *(COMPARED WITH THE BEFORE-PRELOAD VALUE OF THE SAME GROUP; ANOVA REPEATED MEASURES ONE-WAY TEST)*, MIN: MINUTE, S.D.: STANDARD DEVIATION, GP.: GROUP, †B.P.: BEFORE-PRELOAD, †† A.P.: AFTER-PRELOAD.

There was no significant difference in the before-preload or baseline mean values of the four parameters, i.e. HR, SAP, MAP and SpO_2 between the three groups. The mean (SD) after-preload values of all the four parameters of the control group (patients who received only 1 ml min^{-1} of Ringer's lactate infusion in lieu of a preload), remained identical to its before-preload values.

Heart Rate (HR): There was no significant difference (*P* =0.413) in the HR between the three groups, after preloading, and during the period of 60 min after the induction of spinal anaesthesia, except for a brief period at 5 - 7 min following spinal anaesthesia. On *post-hoc* analysis, the HR in the control group was significantly higher than in the Ringer's lactate (RL) group at 5-7 min ($P \leq 0.05$) (Figure-1). Significant increase in the HR from the baseline or before-preload value, was observed in the control, polygeline and RL groups at 1 to 20

min, after-load value to 15 min, and after-load value to 9 min respectively following spinal anaesthesia.

Systolic Arterial Pressure (SAP): There was a significant difference ($P \leq 0.01$) in the SAP between the three groups, from the after-preload value to 60 min, except at 35 min (P=0.063) following spinal anaesthesia (Table-2). On *post-hoc* analysis, the SAP in the polygeline group was significantly higher than in the control group, from the after-preload value to 60 min except at 35 min (P=0.063), following spinal anaesthesia. The SAP in the RL group was significantly higher ($P \leq 0.05$) than in the control group, from the after-preload value to 8 min following spinal anaesthesia. There was no significant difference in the SAP between the polygeline and RL group, from the after-preload value to 60 min except at 14, 15, 20 and 45-60 min following spinal anaesthesia, when the SAP in the polygeline group was significantly higher. The decrease in the SAP from the before-preload value in the control, polygeline and RL groups was significant from 1 to 60 min, 8 to 60 min and 5 to 60 min respectively following spinal anaesthesia. There was a transitory significant increase in the SAP from the before-preload value, in both the polygeline group and RL groups, from the after-preload value to 2 min following spinal anaesthesia (Table-4).

Mean Arterial Pressure (MAP): There was a significant difference ($P \leq 0.05$) in the MAP between the three groups from the after-preload value to 60 min except at 12, 25 and 30 min following spinal anaesthesia (Figure-3). On *post-hoc* analysis, the MAP in the polygeline group was significantly higher than in the control group, from the after-preload value to 60 min except at 12, 15, 25 and 30 min following spinal anaesthesia. The MAP in the RL group was significantly higher than in the control group, at the after-preload value, and from 2 to 6 min following spinal anaesthesia. There was no significant difference in the MAP between the polygeline and RL group, from the after-preload value to 60 min except at 14, 15, 20 and from 35-60 min following spinal anaesthesia, when the MAP in the polygeline group was significantly higher.

The decrease in the MAP from the before-preload value in the control, polygeline and RL groups was significant from 1 to 60 min, 4 to 60 min and 5 to 60 min respectively following spinal anaesthesia. There was a transitory significant increase in the MAP from the before-preload value, in both the polygeline group and RL groups, from the after-preload value to 1 min following spinal anaesthesia (Figure-3).

Peripheral arterial oxygen saturation (SpO_2): There was a significant ($P \leq 0.01$) difference in the SpO_2 readings between the three groups from the after-preload value to 60 min following spinal anaesthesia (Table-3). On *post-hoc* analysis, the SpO_2 in the polygeline and the RL groups was significantly higher than in the control group, from the after-preload value to 60 min following spinal anaesthesia. There was no significant difference in the SpO_2 between the polygeline and RL group, from the after-preload value to 60 min following spinal anaesthesia. In the control group, the decrease in the SpO_2 from the before-preload value was significant from 1 to 60 min following spinal anaesthesia. In the polygeline group, the increase in SpO_2 from the before-preload value was significant from the after-preload value to 3 min and at 60 min following spinal anaesthesia. In the RL group, the increase in SpO_2 from the baseline or before-preload value was significant from the after-preload value to 3 min after the induction of spinal anaesthesia**.**

TABLE-5: INCIDENCE OF SPINAL-INDUCED HYPOTENSION (SIH) AND CARDIOVASCULAR SIDE-EFFECTS IN THE CONTROL, POLYGELINE AND RINGER'S LACTATE GROUPS.

S. No.	Parameter	Groups			*P* value on Inter-group Comparison by Chi-square test			
		I Control *n*=25	II Polygeline *n*=25	III Ringer's lactate *n*=25	Gp.I, II and III	Gp.I and II	Gp. I and III	Gp.II and III
1.	Incidence of Spinal Induced Hypotension	68% *n*=17/25	24% *n*=6/25	32% *n*=8/25	χ^2 Value= 11.327, df=2 *P*=0.003**	χ^2 Value= 9.742, df=1 *P*=0.002**	χ^2 Value= 6.480, df=1 *P*=0.011*	χ^2 Value= 0.397, df=1 *P*=0.529 (NS)
2.	Incidence of Bradycardia	20% *n*=5/25	12% *n*=3/25	16% *n*=4/25	χ^2 Value= 0.5952, df=2 *P*=0.743 (NS)	χ^2 Value= 0.595, df=1 *P*=0.440 (NS)	χ^2 Value= 0.136, df=1 *P*=0.713 (NS)	χ^2 Value= 0.166, df=1 *P*=0.684 (NS)
3.	Incidence of arrhythmias	0% *n*=0/25	4% *n*=1/25	0% *n*=0/25	Significance cannot be found out	Significance cannot be found out	Significance cannot be found out	Significance cannot be found out

P (PROBABILITY) VALUES AT VARIOUS LEVELS OF SIGNIFICANCE (α): DIFFERENCE IS NOT SIGNIFICANT AT *P*>0.05: (NS), SIGNIFICANT AT $P \leq 0.05$*, HIGHLY SIGNIFICANT AT $P \leq 0.01$** AND VERY HIGHLY SIGNIFICANT AT $P \leq 0.001$*** , n= number of the patients, GP.= GROUP, df=degree of freedom, χ^2 Value= Chi square value. .

Incidence of spinal induced hypotension and cardiovascular side-effects: The incidence of SIH in the control, polygeline and Ringer's lactate groups was 68% (*n* = 17/25), 24% (*n* = 6/25) and 32% (*n* = 8/25) respectively. There was a significant difference in the incidence of SIH between the control, polygeline and Ringer's lactate groups (*P* = 0.003). On further inter-group comparison, the difference in the incidence of SIH between the control and RL group (*P* = 0.002) and the control and Ringer's lactate groups (*P*=0.011) was significant. There was no significant difference in the incidence of SIH between the polygeline and RL group (*P* = 0.529). Bradycardia occurred in 20% (*n* = 5/25), 12% (*n* = 3/25) and 16% (*n* = 4/25) of the patients in the control, polygeline and RL groups respectively. There was no significant difference in incidence of bradycardia between the control, polygeline and Ringer's lactate groups (*P* = 0.743). Only one patient in the polygeline group developed arrhythmias i.e. ventricular ectopics. The significance difference if any in respect of the incidence of arrhythmias between the three groups cannot be found, because of the number of patients developing arrhythmias being very less.

Discussion:

Preloading before spinal anaesthesia retains the status of the most commonly followed, effective and safe method for the prevention of spinal anaesthesia induced hypotension. However, an increasing number of studies have begun to dispute its efficacy in the parturient (15-17, 28, 30) and the non-parturient elderly (11, 12) population. Venn and colleagues (13) analysed the effect of crystalloid preloading versus no preloading on cardiovascular variables after spinal anaesthesia in an adult (up to 70 yrs) non-parturient population, even though this study failed to include a colloid group for comparative evaluation. In the said study (13) conducted in patients undergoing elective lower abdominal or lower limb surgery

involving minimal blood loss, it was concluded that a crystalloid preload may be of value in reducing the maximum decrease of both systolic and diastolic arterial pressure, but only in patients with blocks extending above the T_6 dermatome.

Studies (16, 17) have seriously questioned the efficacy of a crystalloid preload and concluded that it could no longer be perceived as a magic bullet, with little or no demonstrable effect on the incidence of SIH. Other authors (7, 31) disagreed with the concept that speed and volume of the preload were not important in reducing the incidence of SIH. Ueyama and others (7) showed a significant correlation between the per cent change in blood volume and cardiac output and suggested that the augmentation of blood volume with volume preload must be great enough to result in significant increase in cardiac output. Hahn and Svensen (31) studied plasma dilution and the rate of infusion of Ringer's solution and demonstrated that the infusion rate had to be at least 50 mL min^{-1} in the young man to yield a plasma dilution of 20%, which corresponds to an increase in blood volume of approximately 10%. Basing their calculations on nomograms, the authors (31) suggested that a fast infusion was more effective than a slow one. Ewaldsson and Hahn (32) showed that the best method to prevent a drop in arterial pressure during the period of time between the injection of bupivacaine and the expected onset of hypotension 5-10 min later was to use a very high rate just after the anaesthetic solution had been injected. It was demonstrated by Mojica, Melendez and Bautista (33) that crystalloid administration at the time of spinal block led to a significant reduction in the risk of cardiovascular side-effects as compared with placebo or with crystalloids administered before spinal block.

The difference in the study protocols in the various studies which compared the efficacy of a colloid preload with a crystalloid preload in the prevention of SIH accounted for the heterogeneity of results making an effective assessment very difficult (34). Most studies concluded that a colloid was superior to a crystalloid in maintaining better haemodynamic stability following spinal anaesthesia.

1) Heart Rate (HR):

There was no significant difference (P =0.413) in the HR after preloading among the three groups in our study. The HR recorded a significant increase from the before-preload value following spinal anaesthesia in all the three groups in our study. There was no significant difference ($P > 0.05$) in the HR among the three groups, during the duration of 1-60 min following spinal anaesthesia, except at 5 to 7 min after the induction of spinal anaesthesia. Our results (except for a brief duration, i.e. from 5 to 7 min after the induction of spinal anaesthesia) partially support the findings of Rout and colleagues (28) who found no difference in the heart rate between the un-preloaded and preloaded groups at any time. At least two studies (9, 10) have showed a decrease in the HR after induction of spinal anaesthesia in both their crystalloid and colloid groups, however there was no significant difference ($P > 0.05$) in the serial values either between the groups or within the group (from the baseline).Various other studies showed an increase in the HR after preload and after the induction of spinal anaesthesia. (25, 28, 35, 36) and support the findings in our study which also showed an increase in HR, both after preload and the induction of spinal anaesthesia.

Critchley and others (25) observed that the increase in heart rate was related to the severity of hypotension and returned to the baseline on correcting the venous filling pressure with

colloid solution. Further, it was found by the same authors that heart rate increased by 8-13% in elderly patients when SAP decreased by 25%.

On intra-group comparison in our study, the HR was significantly higher than the baseline recordings in the control, polygeline and RL groups at 1 to 20 min, after-preload value to 15 min, and after-preload value to 9 min respectively following spinal anaesthesia. These findings were consistent with the results of Rout and coworkers (28), who conducted a study for Caesarean section under spinal anaesthesia and found that HR was significantly greater ($P < 0.0025$) than baseline at 1, 2 and 4-17 min after spinal anaesthesia in preloaded patients and 5-8, 10, 11, 14 and 15 min after spinal anaesthesia in un-preloaded patients.

Our observations support the results of Hahn and Resby (36), who studied the volume kinetics of Ringer's solution and dextran 3% during induction of spinal anaesthesia for Caesarean section and demonstrated that the HR remained higher ($P < 0.001$) during the induction and course of spinal anaesthesia (from baseline values) in both the groups.

2) Systolic Arterial Pressure (SAP):

In our study, on the intergroup comparison, the SAP was significantly higher in the polygeline group than in control group for a sustained period thereby suggesting that as compared to patients who received no preload, the colloid preload was more effective in maintaining intravascular volume (37). The SAP in the RL group was significantly higher than in the control group from the after-preload value to only till 8 min after the induction of spinal anaesthesia. This finding was consistent with the results of earlier studies, which showed the crystalloids to possess a very short intravascular half-life (7, 37). The SAP in the polygeline group in our study was significantly higher than in the RL group at 14, 15, 20 and 45 - 60 min following spinal anaesthesia, and is indicative of the shorter intravascular life of Ringer's lactate.

Our data support the observations of Ueyama and others (7), who demonstrated that only 0.43 ± 0.20 L (28%) of the Ringer's lactate solution remained in the intravascular space after the administration of 1.5 L RL solution over 30 min. Even the volume sustaining effect of this remaining small percentage in the intravascular space would decrease considerably, when induction of spinal anaesthesia would result in vasodilatation (both arteriodilatation and venodilatation), thus fall in the blood pressure may not be prevented. The results of two studies under spinal anaesthesia, one conducted by Riley and others (6) (in Caesarean section) and the other by Sharma, Gajraj and Sidawi (38) (in a non-parturient population undergoing post-partum ligation) partially support our observations and showed that although the SAP remained higher in the colloid group (in comparison to crystalloid group), there was no significant difference between the two. Riley and others (6) attributed this trend to the fact that patients receiving additional ephedrine were excluded from further analysis in their study. The SAP in the polygeline group in our study underwent significant decrease ($P \leq 0.05$) from the baseline, at a later stage, compared to the control and RL groups, and confirmed observations in earlier studies (5-8, 10) that the colloid produces better intravascular expansion, in the face of consequent vasodilatation after spinal anaesthesia.

3) Mean Arterial Pressure (MAP): Few studies have evaluated the mean arterial pressure as a parameter of hypotension during spinal anaesthesia. Deflaque (39) compared the effects of

spinal and extradural anaesthesia upon the blood pressure, and observed that while some authors believe that spinal sympathectomy affects chiefly systolic tension, others record mean pressure and claim diastolic changes occur before systolic changes. However, the observations of the author favoured the former hypothesis.

The significant decrease in the MAP in the RL group of our study from the before-preload value is consistent with a similar reduction in the D5RL group in the study conducted by Mathru and colleagues (5). The greater reduction in the MAP at 3 min (after the induction of spinal anaesthesia) in the crystalloid group in Mathru's study could be because of the presence of various confounding factors (18-24) in the parturient population which makes it more prone to develop spinal-hypotension. The 5% albumin group in Mathru's study, however, did not show any decrease in the MAP as compared to the polygeline group in our study, and may be because of the higher intravascular half life of 5% albumin solution compared to 3.5% polygeline solution used in our study. The intravascular half-life of 3.5% polygeline has been variously quoted as 2-3 hours (40) to 3-6 h (41), whereas the intravascular half-life of 5% albumin is 9.4±4.4 hrs (42). Our data supports the results of Hahn and Resby (36), in whose study the fall in the MAP (in both the Ringer's solution and dextran 3% groups) after the induction and during spinal anaesthesia was significantly decreased from the baseline values ($P < 0.001$).

4) Peripheral Arterial Oxygen Saturation of Blood (S_PO_2).

There was a significant ($P \leq 0.01$) difference in the SpO_2 readings between the three groups from the after-preload value to 60 min following spinal anaesthesia (Table-3). On *post-hoc* analysis, the SpO_2 in the polygeline and the RL groups was significantly higher than in the control group, from the after-preload value to 60 min following spinal anaesthesia. There was no significant difference in the SpO_2 between the polygeline and RL group, from the after-preload value to 60 min following spinal anaesthesia. In the control group, the decrease in the SpO_2 from the before-preload value was significant from 1 to 60 min following spinal anaesthesia. In the polygeline group, the increase in SpO_2 from the before-preload value was significant from the after-preload value to 3 min and at 60 min following spinal anaesthesia. In the RL group, the increase in SpO_2 from the baseline or before-preload value was significant from the after-preload value to 3 min after the induction of spinal anaesthesia. Our results are consistent with the results of Lang and others (43), who compared 6% 130/0.4 HES and RL solution with relation to tissue oxygen tension in patients undergoing major abdominal surgery, and found out that although 6% HES improved tissue oxygenation during and after major surgical procedures compared with a crystalloid based volume replacement strategy, there was no significant difference in the systemic haemodynamics (MAP, HR and CVP) and oxygenation (PaO_2, $PaCO_2$) between the two volume groups throughout the study. Our results are also consistent with the results of Reiter and others (44) who found that goal-directed colloid administration did not increase subcutaneous oxygen tension ($PsqO_2$) compared with goal-directed crystalloid administration in patients undergoing open abdominal surgery. In that study there was no significant difference in the haemodynamic variables (MAP, HR, SV, CO) and oxygenation (PaO_2, $PaCO_2$) between the colloid and the crystalloid groups.

5) Incidence of Spinal-Induced Hypotension (SIH) and cardiovascular side-effects.

The incidence of SIH in the control, polygeline and Ringer's lactate groups in our study was 68% (*n* = 17/25), 24% (*n* = 6/25) and 32% (*n* = 8/25) respectively. A prominent finding of our study was that there was a significant difference in the incidence of SIH between the control, polygeline and Ringer's lactate groups (*P* = 0.003). On further inter-group comparison, the difference in the incidence of SIH between the control and RL group (*P* = 0.002) and the control and Ringer's lactate groups (*P*=0.011) was significant. There was no significant difference in the incidence of SIH between the polygeline and RL group (*P* = 0.529). This finding in our study suggests that both a polygeline and a Ringer's lactate preload were effective in reducing the incidence of SIH, although not fully successful in eliminating it. Our results however differ from the observations of Rout and colleagues (28), who questioned the role of a crystalloid preload in the prevention of hypotension associated with spinal anaesthesia for elective Caesarean section, despite finding statistical significance in the incidence of spinal-induced hypotension in the "preload" and the "unpreload" groups in their study. There was no significant difference in the incidence of SIH between the polygeline and RL group (*P* = 0.529) in our study, which is consistent with the results of other studies (9, 11). Two studies (45, 46) comparing Ringer's lactate and polygeline solution as preload fluids prior to the administration of spinal anaesthesia in patients undergoing caesarean section have found no significant difference in the incidence of SIH between the crystalloid and colloid (polygeline) group, and support our findings.

Recent research article (47) which reviewed a total of 49 studies (4317 patients) has held that the trial sequential analysis (TSA) reveals that there were insufficient data for a definite conclusion that colloid preload is more effective than crystalloid preload in preventing hypotension, and is consistent with the observations in our study.

Bradycardia occurred in 20% (*n* = 5/25), 12% (*n* = 3/25) and 16% (*n* = 4/25) of the patients in the control, polygeline and RL groups respectively in our study. There was no significant difference (*P* =0.743) in the incidence of bradycardia between the three groups in our study. Our results compare favourably with the results of a large prospective study conducted by Carpentar and others (48) in 952 patients undergoing spinal anaesthesia, in which the incidence of bradycardia was 13.1%. Only one patient in the polygeline group in our study developed arrhythmias i.e. ventricular ectopics. The significance difference if any in respect of the incidence of arrhythmias between the three groups cannot be ruled out, because of the number of patients developing this side effect being very less in our study.

Conclusion: Both the polygeline and Ringer's lactate preload were effective in maintaining better haemodynamics and peripheral arterial oxygen saturation, after preloading, and following spinal anesthesia, and significantly reduced the incidence of SIH, when compared to the no-preload (control) group, in elective lower abdominal or lower limb surgery involving minimal blood loss in an adult young non-parturient population. However, the polygeline preload offered no clear and definite advantage *vis-à-vis* the Ringer's lactate preload, as the SAP and MAP in the polygeline group remained significantly higher than in the Ringer's lactate group only for intermittent short periods following spinal anaesthesia, with no significant difference between the two groups, either after preloading or during the first 13 minutes immediately after the induction of spinal anaesthesia. There was no significant difference in the incidence of SIH between the polygeline and Ringer's lactate

groups. There was also no significant difference in the HR and the SpO_2 after preloading and following spinal anaesthesia between the polygeline and Ringer's lactate group.

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Abbreviations:

ANOVA: Analysis of variance; ASA: American Society of Anaesthesiology; bpm: beats per minute; CO: Cardiac output; CVP: Central venous pressure; ECG: Electrocardiogram; HR: Heart Rate; HSD: Honest significance test; IBM: International Business Machines Corporation; L: Lumbar; MAP: Mean arterial pressure; min: minute; NIBP: Non-invasive blood pressure; *P*: Probability; PaO_2: Partial pressure of arterial oxygen; $PaCO_2$: Partial pressure of arterial carbon dioxide; $PsqO_2$: Partial pressure of subcutaneous oxygen; RL: Ringer's lactate; SAP: Systolic arterial pressure; SD: Standard deviation; SIH:

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Spinal- induced hypotension; SpO_2: Peripheral arterial oxygen saturation; SPSS: Statistical Package for Social Sciences; SV: Stroke volume; TSA: Trial sequential analysis.

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