Multiple Case Study: Overview of Hemodynamic Changes in Controlled Hypertensive Patients Undergoing Propofol Administration with Endotracheal Tube (ETT) General Anesthesia at Wangaya Regional General Hospital

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Abstract
Background: Controlled hypertensive patients undergoing surgical procedures under general anaesthesia are at risk of hemodynamic instability. Propofol, as an induction agent, causes cardiovascular depression through vasodilation, reduced sympathetic vasoconstrictive activity, and negative inotropic effects, resulting in fluctuations in blood pressure, mean arterial pressure (MAP), heart rate, respiratory rate (RR), and oxygen saturation (SpO_2). **Objective:** To describe hemodynamic changes in controlled hypertensive patients receiving propofol induction during general anaesthesia with endotracheal tube (ETT) intubation at Wangaya Regional General Hospital. **Methods:** This study employed a qualitative descriptive design with a multiple case study approach involving five controlled hypertensive patients selected through purposive sampling. Data were collected via hemodynamic observation sheets at baseline (pre-induction) and at minutes 1, 3, 5, and 10 post-induction, semi-structured interviews, and medical record documentation. Data were analysed using single-case and cross-case analysis models. **Results:** All participants experienced consistent decreases in systolic blood pressure (SBP), diastolic blood pressure (DBP), MAP, and pulse rate from minute 1 to minute 10 following propofol induction. Participants receiving higher propofol doses (200 mg) demonstrated the greatest declines, reaching clinically significant drops ($\geq 20\%$) in SBP and DBP, as well as near-significant drops ($18\% - 19\%$) in MAP and pulse rate. RR remained stable at 14 breaths/min and SpO_2 remained optimal ($99\% - 100\%$) due to mechanical ventilation and preoxygenation. **Conclusion:** Propofol induction in controlled hypertensive patients leads to a progressive decline in blood pressure, MAP, and pulse rate over the 10-minute post-induction period, with risk factor. Vigilant hemodynamic monitoring, particularly within the first 10 minutes post-induction, is essential to maintain stability and ensure patient safety

Keywords: *controlled hypertension; endotracheal tube; general anesthesia; hemodynamics; propofol*

INTRODUCTION

General anesthesia is a drug-induced state of unconsciousness in which patients cannot be roused, even by painful stimuli (American Society of Anesthesiologists, 2019). Induction is the initial critical phase aimed at transitioning the patient into an anesthetic

state. Propofol is one of the most widely used intravenous hypnotic induction agents due to its rapid onset (30–45 seconds) and short duration of action (3–8 minutes) at a recommended adult dose of 1.5–2.5 mg/kg body weight (Rehatta et al., 2019). However, propofol administration is strongly associated with cardiovascular depression, manifesting as a significant drop in blood pressure and cardiac output. This hemodynamic instability stems from direct vascular smooth muscle relaxation, inhibition of sympathetic vasoconstrictor tone, and negative inotropic effects mediated by calcium channel blockade in the myocardium (Aggarwal et al., 2016).

Although hemodynamic changes under general anesthesia are well-documented, a critical clinical dilemma arises specifically in patients with controlled hypertension. Clinicians and nurse anesthetists often assume that patients with preoperatively controlled blood pressure are at low risk for intraoperative circulatory collapse. In reality, chronically hypertensive patients even when pharmacological control is achieved undergo long-term structural and functional cardiovascular remodelling, such as arterial stiffness and blunted baroreceptor sensitivity (Harrison et al., 2021). Furthermore, ongoing antihypertensive therapy (e.g., ACE inhibitors or calcium channel blockers) combined with propofol's vasodilatory mechanism creates a potent synergistic effect, predisposing controlled hypertensive patients to abrupt, severe hypotension and hypoperfusion of vital organs (Ismail et al., 2022; Raja & Surya, 2024). Conversely, noxious stimuli such as endotracheal tube (ETT) intubation can provoke exaggerated hyperadrenergic surges, leading to rapid blood pressure spikes (Alappat et al., 2020). Understanding this unique vulnerability provides a strong rationale for focusing specifically on controlled hypertensive individuals to optimize perioperative anaesthetic care.

Despite extensive quantitative research comparing induction agents (e.g., propofol versus etomidate) (Bhat et al., 2020; Khan et al., 2024), a distinct research gap remains. Most existing studies focus primarily on statistical mean values across large cohorts, which obscures critical individual variations, patient-specific risk profiles (such as age, smoking history, dietary habits, and stress), and dose-dependent hemodynamic trajectories over time. Quantitative aggregates fail to capture *why* two controlled hypertensive patients receiving similar propofol protocols exhibit vastly different degrees of hemodynamic depression during the crucial first 10 minutes post-induction.

To address this gap, this study offers clear novelty by employing a qualitative descriptive design with a multiple case study approach. Rather than generalizing data, this approach allows for an in depth, minute by minute (minutes 1, 3, 5, and 10) contextual analysis of hemodynamic changes (SBP, DBP, MAP, pulse rate, RR, and SpO_2) in relation to propofol dosage and individual patient characteristics at Wangaya Regional General Hospital. By bridging the gap between standardized clinical protocols and individualized patient nuances, this study provides actionable insights to enhance vigilance and perioperative anaesthetic nursing practice.

METHODS

This study employed a qualitative descriptive design utilizing a multiple case study approach. A qualitative descriptive approach grounded in naturalistic philosophy was selected to understand human experiences in their natural setting. The multiple case study design was specifically chosen to enable an in-depth exploration of individual hemodynamic variation (*single-case analysis*) and to perform comparative evaluation

across participants (*cross-case analysis*) who share a diagnosis of controlled hypertension but differ in individual clinical profiles, such as age, lifestyle risk factors, and antihypertensive regimens. The Central Surgical Theater (IBS) at Wangaya Regional General Hospital in Denpasar City, Bali, Indonesia, served as the study's location. The period of data collecting was December 2025–January 2026.

Participants and Research Design

Patients receiving intra-anesthesia propofol medication and endotracheal tube (ETT) implantation at Wangaya Regional General Hospital made up the study's population. Purposive sampling was the method employed. Five individuals were chosen for this study based on the following inclusion criteria: (1) patients undergoing elective surgery; (2) patients under general anesthesia using the endotracheal intubation technique and administered propofol induction; (3) patients willing to participate in the study; (4) patients with comorbid controlled hypertension; and (5) patients with ASA physical status II. Patients who suffered intraoperative cardiac arrest were excluded.

Ethical Acceptance

Ethical clearance was obtained from the Ethics Committee of the Institute of Technology and Health (ITEKES) Bali, alongside official research approval from Wangaya Regional General Hospital. Written informed consent was secured from all participants, and strict anonymity was maintained by coding participant identities as Participant I through Participant V.

Materials and Instruments

Data were collected through three primary methods: Semi-Structured Interviews: Evaluated baseline participant demographics, hypertension risk factors, history and compliance with antihypertensive medications, and subjective pre-operative complaints. Hemodynamic Observation Sheets: Standardized observation forms were used to record continuous real-time values of Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Mean Arterial Pressure (MAP), pulse rate, oxygen saturation (SpO_2), and Respiratory Rate (RR). Measurements were documented at baseline (prior to induction) and at minutes 1, 3, 5, and 10 following propofol administration. Documentation Review: Clinical profiles, lab results, and surgical details were extracted directly from patient medical records. Non-invasive hemodynamic monitoring was performed continuously using a calibrated bedside monitor equipped with Non-Invasive Blood Pressure (NIBP) uffs and pulse oximetry.

Perioperative Monitoring and Anesthetic Procedures. The study process followed three structured phases: Pre-Anesthetic Phase: Baseline hemodynamic parameters were recorded in the pre-operative holding area, and in-depth interviews were conducted alongside verification of informed consent. Intra-Anesthetic Phase: Premedication (ondansetron 8 mg, dexamethasone 10 mg, and diphenhydramine 10 mg IV) was administered in collaboration with the attending anesthesiologist. Co-induction was achieved using fentanyl 100 mcg, followed by propofol induction at tailored doses 1.8-2.3 mg/kgBB based on participant body weight and clinical status. Following ETT insertion, mechanical ventilation was initiated with a set respiratory rate of 14 breaths per minute. Hemodynamic variables were formally recorded at minutes 1, 3, 5, and 10 post-inductions. Data Analysis Phase: Observation data were synthesized and analyzed through single-case analysis and cross-case Analy.

RESULTS AND DISCUSSION

RESULTS

Participant Characteristics

Based on the results of the study conducted at Wangaya Regional General Hospital on 5 participants, the characteristic data were obtained as presented in Table 1 below.

Table 1. Characteristics of Controlled Hypertensive Participants at Wangaya Regional General Hospital

Participant	Age (years)	Sex	HT Stage	Occupation	HT Medication	Propofol Dose	Surgical Procedure	HT Risk Factors	BP	MAP
I	55	P	Derajat I (JNC7)	Farmer	Captopril 1x25mg	100 mg	Mini PCNL dextra	Age, fatty foods, stress, coffee	143/83	103
II	50	L	Derajat I (JNC7)	Trader	Amlodipine 1x10mg	200 mg	PCNL dextra	Salty foods, ex-smoker	134/79	97
III	35	L	Derajat I (JNC7)	Entrepreneur	Amlodipine 1x10mg	200 mg	FESS dextra/sinistra	Fatty foods, active smoker 10 cigs/day, stress	140/90	107
IV	49	P	Derajat I (JNC7)	Farmer	Amlodipine 1x10mg	150 mg	Ismolobectomy dextra	Fatty foods, stress	136/83	101
V	40	P	Derajat I (JNC7)	Employee	Amlodipine 1x5mg	150 mg	Wide eksisi	Fatty foods, passive smoker, stress	138/75	96

Note: F=Female; M=Male; HT=Hypertension; Sex=Jenis Kelamin; BP=Blood Pressure; MAP=Mean Arterial Pressure

Table 1, five adult patients (aged 35–55 years; 3 females, 2 males) diagnosed with JNC 7 Stage I controlled hypertension were evaluated. Baseline blood pressure was consistently controlled across all cases (130 until $143/75$until 90 mmHg; baseline MAP: 96 until 107 mmHg). Pharmacotherapy consisted primarily of Calcium Channel Blockers (Amlodipine, $n = 4$) and an ACE Inhibitor (Captopril, $n = 1$). Tailored propofol induction doses ranged from 100 mg to 200 mg based on patient weight and clinical assessment. Key individual variations included lifestyle risk factors such as active smoking history (Participant III) and age-related vascular changes, which contributed to varying baseline hemodynamic responses.

Overview of Intra-Anesthetic Hemodynamic Changes

Changes in the hemodynamic parameters of all participants after the administration of propofol induction are presented in Table 2.

Table 2. Overview of Intra-Anesthetic Hemodynamic Changes After Propofol Induction

Partisipan	Parameter	Baseline	1 menit (%)	3 menit (%)	5 menit (%)	10 menit (%)
I (55th)	TDS (mmHg)	143	139 (3%↓)	135 (5%↓)	130 (9%↓)	126 (12%↓)
	TDD (mmHg)	83	76 (8%↓)	75 (10%↓)	72 (13%↓)	67 (19%↓)
	MAP (mmHg)	103	97 (6%↓)	95 (8%↓)	91 (12%↓)	87 (16%↓)
	pulse(x/mnt)	82	80 (2%↓)	77 (6%↓)	75 (8%↓)	71 (13%↓)
	SpO_2 (%)	99	100 (+1%)	100 (+1%)	100 (+1%)	100 (+1%)
	RR (x/mnt)	14	14 (0%)	14 (0%)	14 (0%)	14 (0%)
II (50th)	TDS (mmHg)	134	130 (3%↓)	125 (7%↓)	115 (14%↓)	110 (17%↓)
	TDD (mmHg)	79	73 (8%↓)	68 (14%↓)	63 (20%↓)*	63 (20%↓)*
	MAP (mmHg)	97	92 (5%↓)	87 (10%↓)	80 (17%↓)	79 (18%↓)
	pulse (x/mnt)	68	67 (1%↓)	66 (3%↓)	64 (6%↓)	63 (7%↓)
	SpO_2 (%)	99	99 (0%)	99 (0%)	99 (0%)	99 (0%)
	RR (x/mnt)	14	14 (0%)	14 (0%)	14 (0%)	14 (0%)
III (35th)	TDS (mmHg)	140	136 (3%↓)	120 (14%↓)	112 (20%↓)*	112 (20%↓)*
	TDD (mmHg)	90	86 (4%↓)	84 (7%↓)	81 (10%↓)	74 (18%↓)
	MAP (mmHg)	107	103 (4%↓)	96 (10%↓)	91 (15%↓)	87 (19%↓)
	pulse (x/mnt)	84	74 (12%↓)	70 (17%↓)	70 (17%↓)	68 (19%↓)
	SpO_2 (%)	99	100 (+1%)	100 (+1%)	100 (+1%)	100 (+1%)
	RR (x/mnt)	14	14 (0%)	14 (0%)	14 (0%)	14 (0%)
IV (49th)	TDS (mmHg)	136	127 (7%↓)	124 (9%↓)	121 (11%↓)	117 (14%↓)
	TDD (mmHg)	83	81 (2%↓)	80 (4%↓)	78 (6%↓)	73 (12%↓)
	MAP (mmHg)	101	96 (5%↓)	94 (7%↓)	92 (9%↓)	88 (13%↓)
	pulse (x/mnt)	63	62 (2%↓)	62 (2%↓)	62 (2%↓)	60 (4%↓)
	SpO_2 (%)	99	99 (0%)	99 (0%)	99 (0%)	99 (0%)
	RR (x/mnt)	14	14 (0%)	14 (0%)	14 (0%)	14 (0%)
V (40th)	TDS (mmHg)	138	136 (1%↓)	127 (8%↓)	124 (10%↓)	118 (14%↓)
	TDD (mmHg)	75	74 (1%↓)	73 (3%↓)	71 (5%↓)	67 (11%↓)
	MAP (mmHg)	96	94 (2%↓)	91 (5%↓)	88 (8%↓)	84 (13%↓)
	pulse (x/mnt)	68	67 (1%↓)	65 (4%↓)	64 (6%↓)	61 (10%↓)
	SpO_2 (%)	99	99 (0%)	99 (0%)	99 (0%)	99 (0%)
	RR (x/mnt)	14	14 (0%)	14 (0%)	14 (0%)	14 (0%)

*Decrease of ≥ 20% from baseline (clinically significant). SBP=Systolic Blood Pressure; DBP=Diastolic Blood Pressure; MAP=Mean Arterial Pressure; ↓=decrease

Table 2 Following propofol induction, all five participants exhibited progressive decreases in Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Mean Arterial Pressure (MAP), and pulse rate from Minute 1 through Minute 10. Clinically significant drops (≥ 20%) were recorded in Participant II (DBP decreased by 20% at Minutes 5 and 10) and Participant III (SBP decreased by 20% at Minutes 5 and 10). Similarly, MAP reductions peaked at Minute 10 in Participant III (19% decline) and Participant II (18% decline). Pulse rate demonstrated varying degrees of reduction, with the greatest decrease observed in Participant III (19% drop). In contrast, Respiratory Rate (RR) remained unchanged at 14 breaths/min across all time points due to mechanical ventilation, and oxygen saturation (SpO_2) was stably maintained at 99–100% throughout the 10-minute intra-anesthetic period

DISCUSSION

Demographic and Baseline Characteristics

Five participants (3 females: Participants I, IV, V; 2 males: Participants II, III) aged 35–55 years were included in this study. In female participants aged 40–55 years, the perimenopausal to menopausal transition plays a pivotal role in cardiovascular vulnerability. Estrogenic decline during this phase leads to loss of nitric oxide–mediated vasodilatory effects, increased arterial stiffness, and sympathetic hyperactivation, thereby doubling the risk of hypertension (Iorga et al., 2024). Baseline blood pressure across all subjects fell within stage I hypertension (130–143 / 75–90 mmHg, JNC 7 criteria). All participants were receiving maintenance antihypertensive therapy: Participants II, III, and IV were on amlodipine (CCB) 10 mg; Participant V was on amlodipine 5 mg; and Participant I was treated with captopril (ACE-I) 25 mg. Notably, according to the 2024 ACC/AHA guidelines, withholding ACE inhibitors 24 hours preoperatively reduces the incidence of perioperative hypotension in well-controlled patients. All subjects were classified as ASA physical status II owing to their controlled hypertension.

Hemodynamic Dynamics: Systolic and Diastolic Blood Pressure Changes

Following induction with propofol, a progressive decline in both systolic blood pressure (SBP) and diastolic blood pressure (DBP) was observed across all participants from minute 1 to minute 10. Clinically significant hypotensive responses (>20% baseline reduction) were prominently observed in Participant III (SBP drop from 140 to 112 mmHg between minutes 5 and 10) and Participant II (DBP drop from 79 to 63 mmHg during the same interval).

Rather than merely representing transient blood pressure fluctuations, these reductions reflect the profound pharmacodynamic impact of propofol on an underlying compromised vascular architecture. Propofol induces systemic hypotension via a triad of physiological mechanisms: direct inhibition of sympathetic outflow, peripheral vasodilation (via enhanced GABA-A receptor activation and vascular smooth muscle relaxation), and direct negative inotropism mediated by calcium channel inhibition in the myocardium (Aggarwal et al., 2016; Nagelhout & Elisha, 2018). Notably, both participants exhibiting >20% hemodynamic drops received the highest induction dose of propofol (200 mg), demonstrating a clear dose-dependent cardiovascular depressant response.

The exaggerated response in individual subjects underscores the interplay between patient-specific lifestyle factors and vascular compliance: Participant III (35-year-old active smoker, high-fat diet): Chronic exposure to tobacco smoke induces severe endothelial dysfunction through carbon monoxide induced tissue hypoxia and nicotine-mediated catecholamine surges (Rahmantika, 2021). Over time, atherosclerotic plaque buildup reduces arterial compliance. When propofol induces rapid blunting of sympathetic tone, these rigid, non-compliant blood vessels are incapable of compensatory vasoconstriction to sustain perfusion pressure, resulting in a precipitous drop in SBP. Participant II (50-year-old male, high-sodium diet, past smoking history): Chronic high sodium intake induces fluid retention and structural vascular remodelling, leading to sustained elevation of baseline systemic vascular resistance (SVR) (Purwono et al., 2020). Combined with age-related vascular stiffness (Harrison et al., 2021), propofol-induced loss of basal vascular tone causes a collapse in SVR, manifested predominantly as a sharp decline in diastolic pressure (DBP). These observations align with findings by Bhat et al. (2020), who reported that propofol (2 mg/kg) induces significantly greater reductions in SBP and DBP in hypertensive patients compared to alternative induction agents such as etomidate.

Clinical Implications

The observed hemodynamic changes highlight critical considerations for intra-anaesthetic management in patients with underlying hypertension. Even in controlled stage I hypertensive patients, propofol induction can precipitate significant hypotension due to altered vascular autoregulation and diminished compensatory mechanisms. Anaesthesiologists should consider dose titration, slower administration rates, or pre-induction fluid optimization, particularly in patients with additional risk factors such as smoking, high sodium intake, or advanced age. Furthermore, careful consideration should be given to alternative induction agents with more stable hemodynamic profiles (e.g., etomidate or ketamine) in high-risk patients.

Study Limitations

First, invasive arterial pressure monitoring was not utilized. Second, non-invasive blood pressure measurement at discrete time intervals may miss transient hemodynamic troughs. Third, pre-an aesthetic baseline vascular stiffness and cardiac output were not directly measured using objective modalities (such as pulse wave velocity or echocardiography), relying instead on patient history and risk profiles to infer underlying pathophysiological mechanisms. Future studies with larger cohorts and continuous hemodynamic monitoring are warranted.

CONCLUSION

In controlled hypertensive patients undergoing general anaesthesia with endotracheal tube (ETT) intubation, propofol induction produces dynamic hemodynamic fluctuations characterized by progressive decreases in systolic blood pressure, diastolic blood pressure, mean arterial pressure, and pulse rate during the first ten minutes post-induction. The magnitude of cardiovascular depression may be associated with the propofol induction dose and may be influenced by advanced age, and lifestyle risk factors such as chronic smoking and high sodium intake, whereas respiratory parameters remain stable due to controlled mechanical ventilation. Practically, these findings imply that nurse anaesthetists and anaesthesiologists must exercise dose titration, avoid rapid bolus administration, and perform rigorous pre-anaesthetic risk assessments that evaluate not

only baseline blood pressure but also specific vascular risk factors. Furthermore, vigilant monitoring at minutes 1, 3, 5, and 10 post-induction using early pulse rate drops as an indicator is critical to guide timely fluid optimization or vasopressor support before severe hypoperfusion occurs. To build upon these findings, future research should utilize larger sample sizes, continuous invasive arterial pressure monitoring to capture transient troughs, comparative cohorts evaluating propofol against etomidate, and extended monitoring through the post-anaesthesia care unit (PACU) recovery phase.

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CONFLICT OF INTEREST
The author solemnly declares that this study is free from any conflicts of interest with any party. This research was conducted solely for scientific purposes and the advancement of knowledge in the field of anesthesiology nursing, without any personal.

AUTHOR'S CONTRIBUTION
Rani Valentina Angelina Djami: Research concept and design, literature review, participant recruitment, data collection, data analysis, manuscript drafting and revision.
Yustina Ni Putu Yusniawati, S.Kep., M.Kep: Research supervision, methodological guidance, critical review, and final approval.
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