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Research Article

Psychomotor Recovery After Dexmedetomidine-Based Total Intravenous Anaesthesia in Gastrointestinal Endoscopy Using Trieger Dot Test and Digit Symbol Substitution Test

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ABSTRACT

Background: Gastrointestinal endoscopy has progressively evolved into a cornerstone of modern diagnostic and therapeutic practice. Increasing demand for minimally invasive procedures has encouraged a shift towards day-care settings, with the potential to reduce hospital stays, costs and exposure to hospital-acquired infections while improving patient satisfaction. Advances in anaesthetic care, particularly the use of short-acting, titratable agents, have facilitated rapid clinical recovery. However, residual psychomotor impairment may persist after propofol-based total intravenous anaesthesia (TIVA). The Trieger Dot Test (TDT) and Digit Symbol Substitution Test (DSST) provide complementary assessments of functional recovery. Dexmedetomidine offers sedation and sympatholysis with relatively limited respiratory depression and may influence recovery when combined with multimodal TIVA.

Methods: This observational comparative study included 114 adults aged 18–65 years with American Society of Anesthesiologists (ASA) physical status I–II undergoing gastrointestinal endoscopy. Fifty-seven received dexmedetomidine as an adjunct to TIVA (DEX group), and 57 received the same background anaesthetic technique without dexmedetomidine (No-DEX group). TDT and DSST were administered before the procedure and at 30, 60, 90 and 120 minutes afterwards. Haemodynamic variables and peri-procedural adverse events were assessed.

Results: Mean TDT errors were fewer in the DEX group at 30 minutes (3.96 ± 1.45 vs. 5.17 ± 1.68; reported p = 0.000037), 60 minutes (0.54 ± 0.96 vs. 1.63 ± 1.67; p = 0.000022) and 90 minutes (0 vs. 0.15 ± 0.49; p = 0.0085). DSST scores were similar at 30 minutes and modestly higher in the DEX group at 60 minutes (16.56 ± 2.89 vs. 15.61 ± 2.92; reported p = 0.0425). Scores were comparable by 90–120 minutes. No sustained clinically important haemodynamic difference or major sedation-related complication was reported.

Conclusion: Dexmedetomidine as an adjunct to multimodal TIVA was associated with earlier restoration of psychomotor performance after gastrointestinal endoscopy, particularly during the first 60–90 minutes of recovery. These findings warrant further randomized evaluation in ambulatory endoscopic practice.

Keywords: dexmedetomidine; gastrointestinal endoscopy; psychomotor recovery; Trieger Dot Test; Digit Symbol Substitution Test; total intravenous anaesthesia; procedural sedation

INTRODUCTION

Gastrointestinal endoscopy encompasses common diagnostic and therapeutic procedures increasingly performed on an ambulatory or day-care basis. Sedation improves procedural tolerance, reduces anxiety and discomfort, and facilitates a technically satisfactory examination. However, the quality of recovery is particularly important in outpatient practice because apparent wakefulness does not necessarily indicate full restoration of

attention, coordination, reaction time or executive function. Riphaus et al. demonstrated that the extent and duration of post-endoscopic psychomotor impairment vary according to the sedative regimen[1].

Residual cognitive and psychomotor dysfunction is therefore an important consideration when comparing endoscopic sedation strategies. Ekmekci et al., using the Trieger Dot Test (TDT) and Digit Symbol Substitution Test (DSST), demonstrated differences in post-colonoscopy cognitive performance between sedation regimens[2]. Similarly, Allampati et al. observed that cognitive domains recover at different rates after propofol-based endoscopic sedation; cognitive flexibility may recover while psychomotor speed and reaction time remain impaired[3].

The TDT is a simple visuomotor test developed to assess recovery following anaesthesia, whereas the DSST assesses processing speed, attention, visual scanning and motor coordination. Both tests have been used in perioperative recovery research and provide complementary information on psychomotor restoration[2,4].

This distinction is particularly relevant in ambulatory practice because post-sedation instructions often restrict driving, decision-making and independent activities even when clinical discharge criteria have been met. Strategies associated with earlier restoration of psychomotor performance may improve the predictability of recovery in high-throughput endoscopy units. However, any recovery advantage must be considered alongside procedural and cardiopulmonary safety. The TDT, DSST and other neuropsychological assessments indicate that residual impairment can persist after apparent clinical awakening and that no single bedside test captures every domain of recovery[1–4].

Dexmedetomidine, a selective α_2-adrenergic agonist, induces sedation, anxiolysis and sympatholysis through central adrenergic pathways. In human volunteer studies, dexmedetomidine produced dose-dependent sedation with relatively preserved respiratory function. Increasing concentrations were associated with reductions in heart rate and changes in blood pressure[5,6]. These pharmacological properties make it an attractive adjunct when reducing the requirements and adverse effects of other hypnotic and analgesic agents is desirable.

The application of dexmedetomidine in gastrointestinal endoscopy has also expanded. Inatomi et al. reported reduced additional sedative requirements and respiratory complications in elderly patients undergoing endoscopic retrograde cholangiopancreatography[7]. In a meta-analysis, Nishizawa et al. found dexmedetomidine to be an effective option for gastrointestinal endoscopic sedation, although satisfaction, heart rate and recovery outcomes varied according to the comparator and protocol[8]. Relatively few studies have examined serial psychomotor performance when dexmedetomidine is incorporated into multimodal TIVA for gastrointestinal endoscopy. The present study therefore evaluated whether dexmedetomidine as an adjunct to TIVA was associated with earlier psychomotor recovery, assessed using the TDT and DSST, and examined peri-procedural haemodynamic stability and adverse events.

MATERIALS AND METHODS

Study Design and Participants

An observational comparative study was conducted at a tertiary-care hospital among adults undergoing gastrointestinal endoscopic procedures. Patients aged 18–65 years with ASA physical status I or II were recruited using convenience sampling. Written informed consent was obtained before enrolment. Patients with psychiatric illness, disabling neurological disorders, alcohol dependence or substance abuse, and pregnant or lactating women were excluded. An a priori sample-size calculation reported in the original protocol yielded a requirement of 57 patients per group. Accordingly, 114 participants were included: 57 in the DEX group and 57 in the No-DEX group. The study was undertaken following approval from the institutional ethics committee.

Anaesthetic Technique

Baseline heart rate, electrocardiography, non-invasive blood pressure and peripheral oxygen saturation were monitored. Intravenous access was secured, and glycopyrrolate 0.004 mg/kg, midazolam 0.02 mg/kg and fentanyl 1 µg/kg were administered intravenously. Patients were placed in the left lateral position, and propofol 1–1.5 mg/kg and ketamine 1 mg/kg

were titrated to a Ramsay Sedation Scale score of 3–4. Patients in the DEX group received dexmedetomidine 1 µg/kg as a loading dose over 10 minutes, followed by an infusion of 0.5 µg/kg/h. Dexmedetomidine was not administered in the No-DEX group. When procedural tolerance was inadequate, propofol 0.25 mg/kg was administered as a supplementary bolus. Oxygen was delivered through nasal prongs at 4 L/min, and Ringer lactate was infused at approximately 4 mL/kg/h. All anaesthetic infusions were discontinued at the end of the procedure, defined as removal of the endoscope.

Monitoring and Safety Definitions

Respiratory depression was defined as peripheral oxygen saturation (SpO_2) <90%. Hypotension and hypertension were defined as a decrease or increase, respectively, in mean arterial pressure exceeding 20% of baseline. Tachycardia was defined as a heart rate >100 beats/min and bradycardia as a heart rate <50 beats/min. The specified management for persistent bradycardia accompanied by hypoxaemia (SpO_2 <95%) consisted of a jaw thrust and increased oxygen flow, with intravenous atropine if required. Electrocardiography, heart rate, respiratory rate, non-invasive blood pressure, SpO_2 and clinical monitoring continued during recovery.

Psychomotor Assessment

Psychomotor function was assessed using the TDT and DSST at baseline and at 30, 60, 90 and 120 minutes after the procedure. Digit configurations were varied during repeated DSST administration to mitigate practice effects. For the TDT, patients were required to connect 47 dots continuously to reproduce a given figure within 60 seconds. Performance was assessed primarily by the number of dots missed. For the DSST, patients were asked to replace randomly presented digits with corresponding symbols using the reference key. The score was the number of correct substitutions completed within 90 seconds.

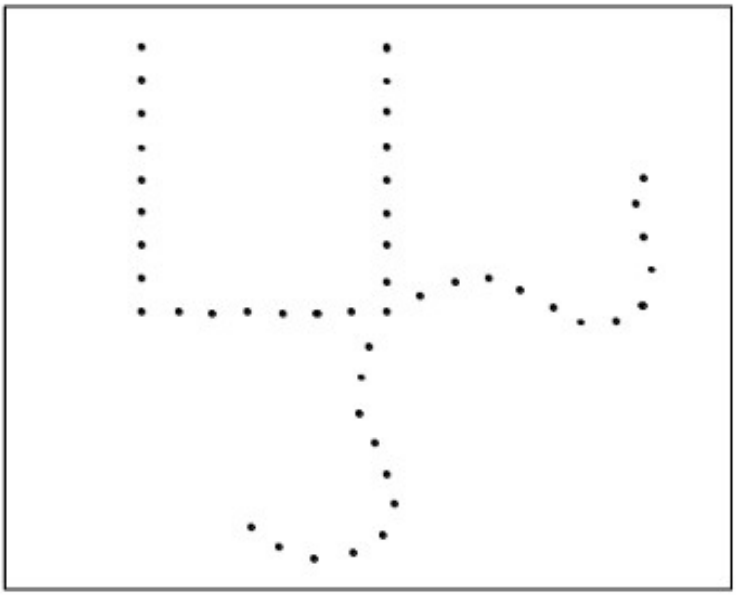


Figure 1. Trieger Dot Test Assessment Template Used for Psychomotor Evaluation

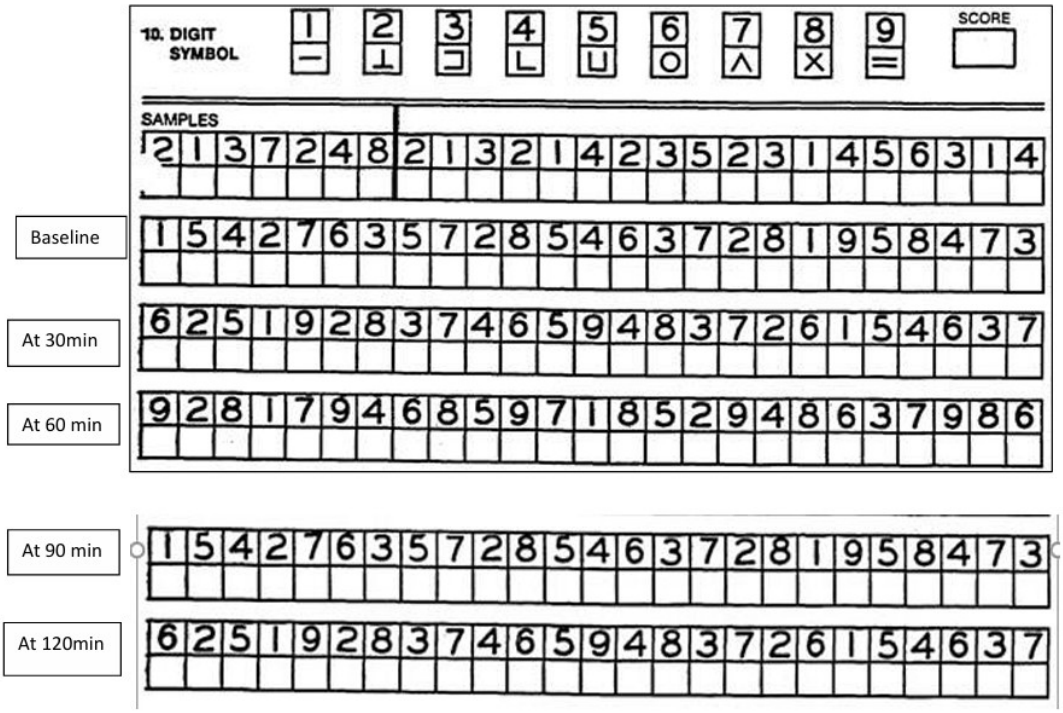


Figure 2. Digit Symbol Substitution Test Assessment Sheet Used for Serial Psychomotor Evaluation

Statistical Analysis

Data were entered into Microsoft Excel and summarized using frequencies, percentages, means, standard deviations, medians and ranges, as appropriate. Categorical variables were compared using the chi-square test. The original analysis specified two-tailed p-values and a significance threshold of $p \leq 0.05$. Between-group p-values for continuous variables were retained as reported in the source dataset; the specific tests used for these comparisons were not documented in the supplied manuscript. The reported p-values require verification against the original analysis before definitive statistical conclusions are drawn.

RESULTS

A total of 114 patients participated in the study. Based on the counts in Table 1, 54 were male and 60 were female. Sex and ASA physical status distributions were identical in both groups. Procedures included colonoscopy, endoscopic retrograde cholangiopancreatography (ERCP), endoscopic ultrasonography (EUS) and oesophagogastroduodenoscopy (OGD). Although the relative proportions of ERCP and colonoscopy differed numerically, the overall procedural distribution did not differ statistically ($p = 0.24$).

Psychomotor impairment was most prominent at 30 minutes after endoscopy. Patients receiving dexmedetomidine missed fewer dots on the TDT at 30, 60 and 90 minutes; both groups achieved zero missed dots at 120 minutes. DSST scores declined markedly at 30 minutes in both groups. By 60 minutes, the DEX group had a modestly higher mean DSST score than the No-DEX group. By 90 and 120 minutes, scores had approached baseline, with no apparent between-group separation in the reported comparisons.

Serial heart rate and arterial pressure measurements showed broadly parallel patterns. Baseline systolic blood pressure was lower in the DEX group (109.49 ± 4.88 vs. 111.59 ± 4.22 mmHg; reported $p = 0.0077$). Subsequent blood pressure measurements showed no consistent between-group pattern. Immediately after sedation, the reported SpO_2 difference was small (98.92 ± 0.25% vs. 98.98 ± 0.23%; reported $p = 0.021$) and clinically negligible, with no sustained oxygenation difference. No episodes of bradycardia, hypotension, desaturation, postoperative nausea and vomiting, or aspiration were reported.

Table 1. Baseline Clinical and Procedural Characteristics

Characteristic	DEX group, n (%)	No-DEX group, n (%)	p-value
Sex: Male	27 (47.37)	27 (47.37)	1.00
Sex: Female	30 (52.63)	30 (52.63)	
Procedure: Colonoscopy	8 (14.04)	16 (28.07)	0.24
Procedure: ERCP	35 (61.40)	26 (45.61)	
Procedure: EUS	2 (3.51)	3 (5.26)	
Procedure: OGD	12 (21.05)	12 (21.05)	
ASA I	37 (64.91)	37 (64.91)	1.00
ASA II	20 (35.09)	20 (35.09)	

The groups were balanced with respect to sex and ASA physical status. ERCP was numerically more frequent in the DEX group and colonoscopy in the No-DEX group, although the overall procedural distribution was not statistically different. Because treatment allocation was observational rather than randomized, differences in procedural complexity and clinician selection could still influence psychomotor outcomes.

Table 2. Trieger Dot Test Performance During Recovery

Time point	DEX group, mean ± SD	No-DEX group, mean ± SD	p-value
Pre-procedure	0 ± 0	0 ± 0	0.50
30 min	3.96 ± 1.45	5.17 ± 1.68	0.000037
60 min	0.54 ± 0.96	1.63 ± 1.67	0.000022
90 min	0 ± 0	0.15 ± 0.49	0.0085
120 min	0 ± 0	0 ± 0	0.50

TDT performance demonstrated the clearest separation between the groups during early recovery. Both groups showed impairment at 30 minutes, but patients receiving dexmedetomidine missed fewer dots. The difference persisted at 60 and 90 minutes, with complete convergence by 120 minutes. The pattern suggests an association with earlier intermediate psychomotor recovery rather than a difference in final recovery status.

Table 3. Digit Symbol Substitution Test During Recovery

Time point	DEX group, mean ± SD	No-DEX group, mean ± SD	p-value
Pre-procedure	18.64 ± 3.28	18.64 ± 3.28	0.50
30 min	10.43 ± 2.53	10.35 ± 2.46	0.42
60 min	16.56 ± 2.89	15.61 ± 2.92	0.0425
90 min	18.47 ± 3.06	18.19 ± 3.10	0.31
120 min	18.85 ± 2.99	18.63 ± 3.05	0.34

DSST scores declined in both groups during the first 30 minutes, suggesting similar early impairment of processing speed and visuomotor coordination. At 60 minutes, the DEX group showed a modestly higher mean score, with a reported p-value of 0.0425 that requires confirmation. By 90–120 minutes, mean scores in both groups approached baseline. Any recovery advantage therefore appeared transient.

Table 4. Selected Hemodynamic and Oxygenation Measurements

Variable/time	DEX group, mean ± SD	No-DEX group, mean ± SD	p-value
HR baseline, beats/min	85.94 ± 9.30	87.42 ± 9.44	0.20
HR 30 min	75.84 ± 9.84	76.17 ± 10.11	0.42
HR 60 min	77.23 ± 6.48	79.00 ± 5.89	0.35
SBP baseline, mmHg	109.49 ± 4.88	111.59 ± 4.22	0.0077
SBP post-sedation	115.73 ± 8.69	114.14 ± 7.95	0.15
SBP 30 min	108.56 ± 6.88	107.54 ± 8.06	0.23
SBP 60 min	108.58 ± 3.37	108.25 ± 6.79	0.26
DBP baseline, mmHg	66.10 ± 4.47	66.29 ± 6.84	0.41
DBP 30 min	65.64 ± 5.42	67.19 ± 5.44	0.066
DBP 60 min	69.11 ± 3.93	66.00 ± 8.48	0.17
SpO_2 post-sedation, %	98.92 ± 0.25	98.98 ± 0.23	0.021
SpO_2 30 min, %	98.92 ± 0.50	98.85 ± 0.39	0.13
SpO_2 60 min, %	98.76 ± 0.43	99.00 ± 0.81	0.23

Haemodynamic and oxygenation trends were broadly similar across the observation period. The lower baseline systolic pressure in the DEX group preceded administration of the study adjunct and could not be attributed to dexmedetomidine. The immediate post-sedation SpO_2 difference was only 0.06 percentage points and was unlikely to be clinically meaningful. No sustained cardiovascular or respiratory instability was reported in either group, although the sample was not powered to establish safety equivalence.

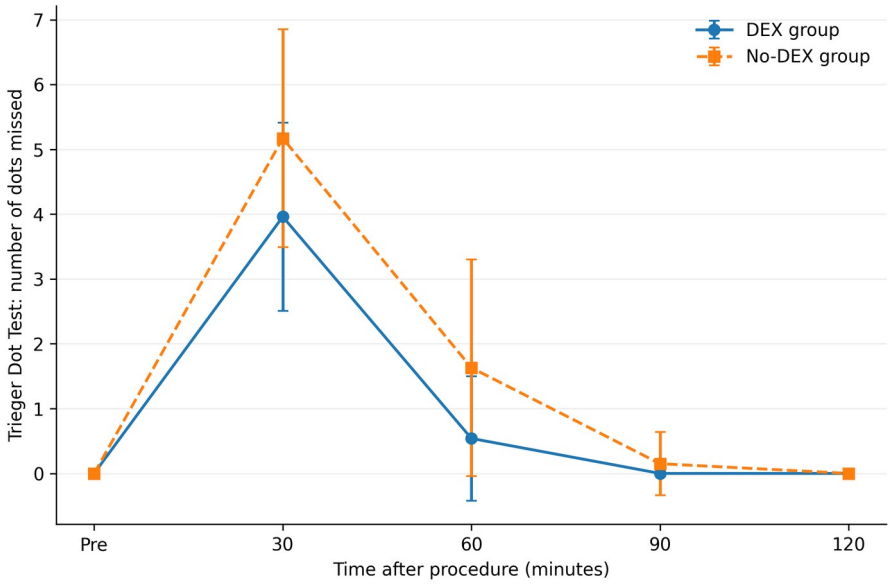


Figure 3. Serial Trieger Dot Test Recovery in the DEX and No-DEX Groups

The serial TDT profile suggests a more rapid recovery trajectory among patients receiving dexmedetomidine. The greatest impairment occurred at 30 minutes in both groups, but the mean number of missed dots was lower in the DEX group during early recovery. At 60 minutes, the DEX group was closer to baseline, whereas residual impairment was more apparent in the No-DEX group. Both trajectories converged by 120 minutes.

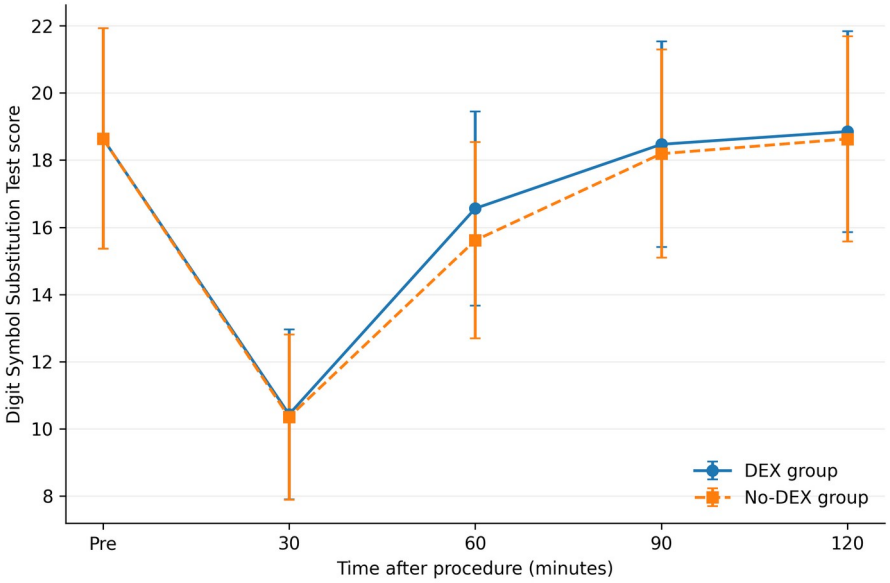


Figure 4. Serial Digit Symbol Substitution Test Recovery in the DEX and No-DEX Groups

DSST performance declined at 30 minutes after endoscopy irrespective of dexmedetomidine exposure, indicating early impairment of attention, processing speed and visuomotor integration. Recovery was modestly greater in the DEX group at 60 minutes. By 90 minutes, scores in both groups approached baseline. The observed difference was therefore confined to the intermediate recovery phase, when functional assessment may be particularly relevant to discharge planning.

DISCUSSION

The principal finding of this observational study was that dexmedetomidine as an adjunct to multimodal TIVA was associated with earlier psychomotor recovery during the first 90 minutes after gastrointestinal endoscopy. Differences in TDT performance were observed at 30, 60 and 90 minutes, while the DSST showed a smaller numerical advantage at 60 minutes. These observations are consistent with the randomized study by Mishra et al., which reported earlier TDT recovery and improved early DSST performance following dexmedetomidine administration as part of balanced anaesthesia[9]. Takayama et al. demonstrated that the TDT could detect differences in intermediate psychomotor recovery between TIVA regimens[10]. Perika et al. observed earlier recovery of some motor tasks following dexmedetomidine rather than propofol sedation during spinal anaesthesia, although the benefit was not uniform across psychomotor domains[11].

The findings are also relevant to ambulatory gastrointestinal practice. Ekmekci et al. demonstrated the sensitivity of the TDT and DSST to cognitive differences following colonoscopy sedation[2], while Riphaus et al. reported better psychomotor recovery with propofol than with midazolam–pethidine sedation[1]. Allampati et al. subsequently demonstrated that psychomotor speed may remain impaired even after some cognitive functions have recovered following propofol-based endoscopy[3]. Collectively, these observations suggest that conventional awakening criteria may underestimate residual functional impairment. In the present study, mean DSST scores at 30 minutes remained below baseline in both groups despite clinical recovery.

Several endoscopy-specific studies have evaluated dexmedetomidine-containing combinations. Akarsu Ayazoglu et al. reported faster recovery of protective reflexes and motor function with dexmedetomidine- or sufentanil-containing propofol regimens during colonoscopy compared with meperidine- or midazolam-based combinations[12]. Cheung et al. reported reduced propofol and alfentanil requirements with dexmedetomidine during upper gastrointestinal endoscopy[13]. Wu et al. described effective sedation with dexmedetomidine during oesophagogastroduodenoscopy and relatively preserved respiratory function, although propofol produced deeper sedation and greater patient satisfaction[14].

Inatomi et al. also reported lower additional sedative requirements and fewer respiratory complications in elderly patients receiving dexmedetomidine during ERCP[7].

Recovery findings have varied across studies and anaesthetic regimens. Tekeli et al. compared ketamine–propofol and dexmedetomidine–propofol during upper gastrointestinal endoscopy and reported differences in recovery and sedation profiles[15]. Ohtani et al. found that adding dexmedetomidine to general anaesthesia could delay certain conventional recovery endpoints following abdominal surgery[16]. Such variability may reflect differences in loading doses, maintenance infusions, concomitant anaesthetic agents, procedural duration and the endpoints used to define recovery. Clinical awakening and restoration of psychomotor coordination are distinct outcomes; recovery cannot therefore be adequately characterized using a single measure.

The broader evidence supports this nuanced interpretation. Nishizawa et al. found no consistent overall difference in pooled recovery time between dexmedetomidine and propofol in gastrointestinal endoscopy[8]. A larger meta-analysis by Tang et al., including 40 trials and 2,955 patients, found that dexmedetomidine reduced hypoxia and additional sedative requirements but increased the risk of bradycardia[17]. Liu et al. reported earlier attainment of post-anaesthesia discharge criteria and fewer respiratory adverse events with dexmedetomidine–propofol during upper gastrointestinal endoscopic ultrasonography, although hypotension and bradycardia were more frequent[18]. These findings highlight that dexmedetomidine may offer advantages for selected recovery or respiratory outcomes without being superior across all aspects of procedural sedation.

The safety observations in the present sample must be interpreted in the context of dexmedetomidine's pharmacodynamic profile. The absence of sustained differences in heart rate, blood pressure or oxygen saturation suggests that the regimen was tolerated in the enrolled ASA I–II population, but the sample size was insufficient to exclude uncommon adverse events. Controlled studies indicate that relative preservation of respiration can be accompanied by bradycardia or hypotension, particularly with higher loading doses or rapid administration[5,6,17,18]. Careful titration and appropriate monitoring remain essential.

Several mechanisms may explain the observed psychomotor recovery profile. Dexmedetomidine produces α_2-mediated sedation and sympatholysis with relatively preserved ventilation and may reduce requirements for propofol and opioids, potentially limiting residual effects of other anaesthetic drugs[5–7]. Its neurophysiological sedative profile differs from that of GABAergic hypnotics. However, total doses of supplementary sedatives were not fully documented in this study, so a drug-sparing mechanism remains hypothetical. The absence of major haemodynamic events in the present sample is reassuring but does not exclude their occurrence in other populations.

The study had important limitations. Its observational, non-randomized design and convenience sampling introduced the possibility of treatment selection bias and unmeasured confounding. Endoscopic procedures were heterogeneous, and the study was not powered to detect rare cardiovascular or respiratory adverse events. The TDT and DSST are paper-and-pencil assessments susceptible to learning effects even when digit configurations are varied. Total sedative doses and procedural durations were incompletely reported, limiting assessment of a potential drug-sparing effect. The continuous-variable statistical tests and reported p-values require verification, and multiple serial comparisons may increase the risk of false-positive findings. Larger randomized, blinded studies with standardized discharge criteria, multidomain cognitive assessments and detailed sedative-dose evaluation are needed.

CONCLUSION

Dexmedetomidine as an adjunct to multimodal total intravenous anaesthesia was associated with earlier psychomotor recovery following gastrointestinal endoscopy. The observed advantage was most apparent within the first 60–90 minutes, with fewer TDT errors and modestly higher DSST scores at 60 minutes; both groups achieved comparable recovery by 120 minutes. No sustained clinically

important haemodynamic or oxygenation disturbance was reported. These observations suggest a potential role for dexmedetomidine-based TIVA in early functional recovery during ambulatory endoscopy. However, the observational design, limited safety sample and need to verify the reported statistical comparisons warrant cautious interpretation and confirmation in adequately powered randomized trials.

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