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Sedative and Analgesic Effects of Intravenous Detomidine and Tramadol on Horses

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Abstract : This study was performed to evaluate the sedative and analgesic effects of intravenous (IV) administration of detomidine (D) and tramadol (T) to horses. Six warmblood horses each received D (10 μg/kg), T (2 mg/kg), and a combination of DT (10 μg/kg and 2 mg/kg). No significant differences in the heart rate, respiratory rate, rectal temperature, indirect arterial pressure, and gastrointestinal motility between D and DT were observed. The sedative effect was evident within 5 min after D and DT administration, but no significant difference between D and DT was observed. D and DT induced a similar analgesic effect up to 50 min after injection and DT maintained a longer analgesic effect than D. A significant increase in blood glucose was shown for D after the injection, but not for DT. A horse with T and DT showed an excited behavior within 5 min of the injection. This study suggests that the DT combination could be used for diagnostic procedures and simple surgeries in standing horses, with caution for excitement in the early phase after the administration.

***Key words* :** detomidine, tramadol, sedation, analgesia, horse.

Introduction

Sedatives and analgesics have been typically used for diagnostic procedures and simple surgeries in standing horses (14). Because horses can suddenly wake from sedation induced by a sole sedative when they encounter stimuli such as loud voices, severe pain and physical attacks, analgesics have been also required with the sedatives in cases of surgeries with pain. Analgesics are mainly comprised of three classes of drugs: α-2 adrenergic agonists, opioids and non-steroidal anti-inflammatory drugs (NSAIDs) (8).

The α-2 agonists, such as detomidine (D), romifidine, and xylazine (X), have potent analgesic and sedative effects and have been widely used in equine medicine (19). In particular, D has been shown to produce effective sedative and analgesic effects in horses via binding to α-2 receptors in the locus ceruleus complex of the brain stem and spinal cord (18). D also produces severe muscle relaxation, in compliance with inhibition of excitatory neurotransmitters secreted from spinal interneurons (17). These actions result in the characteristic dose-dependent head drop, ataxia, and decrease in heart rate (HR), respiratory rate (RR), and gastrointestinal (GI) motility (4,5,10).

Opioids are not widely used in horses as a sole analgesic because they can cause central nervous system (CNS) excitation, sympathetic stimulation, and can stimulate locomotion (3). However, opioids are usually used with α-2 agonists because the combination has been reported to increase clinical effects and decrease side effects, when compared to the effects of individual use of these medicines (7,9,12). In particular, most painkillers are subject to legal control, but tramadol (T) can be used without this control in Korea (22).

T is a centrally acting analgesic drug that has been clinically used for the last two decades in humans to reduce pain (6). T is also used for treatment of chronic cancer and orthopedic pain in humans and animals. In addition to minimal effects on GI motility and no significant cardiovascular or respiratory effects (21), T has the same analgesic effect on moderate pain as equipotent doses of morphine (13).

This study was performed to compare the physiological responses, sedative and analgesic effects of the combination of D as an α-2 agonist and T as an opioid for clinical use in equine practice.

Materials and Methods

Experimental animals

Six warmblood horses (five geldings and one stallion), 9 to 18 years of age (14.0 ± 3.4 years) weighing 531 to 592 kg (573 ± 25 kg), were used for this experiment. The horses were raised in individual stalls at a private stable, where they were fed with roughage and had free access to water.

Procedures

This study was performed as a blinded, randomized, three-

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way crossover design with a 7-day washout period between groups. Before each treatment, the horses were physically inspected and weighed. Food, but not water, was not provided for at least 8 hours before drug administration. During the experiment, horses were placed in calm stocks (temperature: 19.3 ± 1.6ºC, humidity: 68 ± 13%) and allowed 20 min for adaptation to their surroundings. The hair over the left jugular vein was cut, and a 16 gauge intravenous catheter was applied in an aseptic manner. An electrical stimulator (AM-3000®, TEC, Japan) was installed at least 2 m away from horses. Electrocardiogram pads for an apex-base lead, a rectal temperature (RT) probe, and an indirect arterial pressure (IAP) probe at the base of the tail for oscillometry were placed to collect HR, RT and IAP. HR, RT, and IAP were measured through a patient monitor (MEDIANA®; MEDIANA, Wonju-si, Korea). RR was estimated by counting thoracic wall motions for 1 min. The left paralumbar fossa was aseptically ready by 70% alcohol, and two 22-gauge, 1.4 inch needles were inserted 8 cm apart for analgesic effect assessment.

Treatments consisted of D (Domosedan®; Pfizer, NY, USA), 10 μg/kg; T (Tramadol HCl Injection®; Huons, Sungnam-si, Korea), 2 mg/kg; and a combination of D 10 μg/kg and T 2 mg/kg which were intravenously injected via a jugular vein catheter. D was given as a bolus, whereas T was slowly administered over at least 2 min. For the DT treatment, the D dose was followed by a slow T injection. HR, RR, RT, IAP, sedation, ataxia, and analgesia (using electrical stimulation and pinprick) were measured prior to drug injection and 5, 10, 20, 30, 40, 50, 60, 70, 80, and 90 min after administration. The blood chemistry and GI motility were evaluated prior to drug administration and 30, 60, and 90 min after administration.

Degree of sedation was scored using two methods. One of the methods was a 4-point criteria system in which: 1 = marked deep sedation, defined as remarkably decreased movement, lower head carriage with mouth to the carpal joint, obvious drowsiness, droopy eyelids and lip, and remarkably wide based stance; 2 = marked moderate sedation, defined as moderately declined movement, lower head carriage with mouth to the elbow joint, drowsiness, slightly droopy eyelids and lip, and moderately wide based stance; 3 = marked mild sedation, defined as slightly declined movement, lower head carriage with mouth to the shoulder, and declined sensitivity to surroundings; and 4 = marked no sedation, which was regarded as a normal behavior and appearance. The other method was measurement of lip height from the ground to the lower lip of the horse.

The degree of ataxia was scored on 4-point criteria as follows: 1 = swaying, leaning on the walls with carpi flexed and/or hind limbs crossed; 2 = swaying and leaning against the walls; 3 = stable, but mild swaying; 4 = no change from the normal non-sedated condition.

The analgesic effect was also examined using two methods: one of the methods was electrical stimulation (5.5 mV, 1 Hz, 1 sec) on the left paralumbar fossa with an electrical stimulator; and other method was by pinprick with a 22-gauge, 1.4 inch needle on the right side of the neck, right paralumbar fossa, and right hip, which were pricked in turn. The needle prick was applied to the whole length of needle (1.4 inches), but continued only once in one place. The degree of analgesia was checked by viewing the changes from baseline in appearances such as tail twitch, attention to the stimulated site, movement of the head and legs, pawing, escape from stimulus and kicking. Analgesia was indicated on a 4-point numerical scale as follows: 1 = deep analgesia, defined as remarkably different responses from baseline (5 or 6 of the observational appearances disappeared); 2 = moderate analgesia, defined as moderately different from baseline criterion (3 or 4 observational appearances disappeared); 3 = mild analgesia, defined as slightly different from baseline criterion (1 or 2 observational appearances disappeared); and 4 = no analgesia, in which no response changes were confirmed. Sedation and analgesic points of electrical stimulation were marked with a 4-point scale, but the analgesic point of pinpricks was the amount (3 to 12) of the 4-point scale of the three sites (the right neck, right paralumbar fossa, and right hip).

Venous blood samples were analyzed by I-STAT® (VetScan, CA, USA). This included hematocrit (Hct), hemoglobin (Hgb), blood urea nitrogen (BUN), glucose (Glu), chloride (Cl^-), sodium (Na^+), potassium (K^+).

GI motility was evaluated by auscultation at the 4 abdominal quadrants (superior and inferior part on the each left and right sides), with a 1 min delay between quadrant evaluations. A subjective point was designated for each quadrant in accordance with the following 5-point scale : 0 = no intestinal sounds; 1 = mild, low-pitched, audible, crepitation-like sounds at a frequency of 1 per min on both sites within a quadrant; 2 = low-pitched, crepitation-like sounds at a frequency of more than 1 per min on both sites within a quadrant; 3 = long, loud gurgling sounds audile once per min at both sites within a quadrant; 4 = long, loud gurgling sounds audible more than once per min on both sites within a quadrant. The point of the 4 quadrants was totaled, giving a cumulative range of 0 to 16.

Statistical analyses

Statistical analysis of data was performed with the SPSS® 18.0 software (SPSS, NY, USA). The data for HR, RR, RT, IAP, lip height, Hct, Hgb, Na^+, Cl^-, K^+, Glu and BUN were compared by two-way repeated ANOVA. When an important difference was found among groups, the Tukey's test or paired *t*-test was used as appropriate. Sedation, ataxia, analgesic scores and GI motility were estimated by non-parametric Wilcoxon test. Statistical significance was considered at $p < 0.05$.

Results

HR and RR clearly decreased from baseline (time = 0) with D and DT, and slightly increased with T from baseline to 10 min (Table 1). The pronounced decrease in HR with DT occurred over a longer duration than for the D treatment. No

Table 1. Changes in the heart rate (HR), respiratory rate (RR) and rectal temperature (RT) after tramadol (T), detomidine (D) and detomidine plus tramadol (DT) administration

Time (min)	HR			RR			RT		
	T	D	DT	T	D	DT	T	D	DT
0	33 ± 7	36 ± 4	32 ± 3	14 ± 5	12 ± 3	13 ± 5	37.4 ± 0.3	37.5 ± 0.3	37.4 ± 0.3
5	38 ± 13	25 ± 4[†]	24 ± 3[†*]	20 ± 9	9 ± 2[†*]	10 ± 2[†]	37.4 ± 0.4	37.6 ± 0.2	37.5 ± 0.2
10	43 ± 16	26 ± 6[†]	25 ± 2[†*]	17 ± 9	8 ± 2[†*]	8 ± 1[†]	37.5 ± 0.3	37.7 ± 0.2	37.6 ± 0.3
20	38 ± 9	26 ± 6[†]	23 ± 2[†*]	18 ± 8	8 ± 3[†*]	7 ± 1[†*]	37.5 ± 0.3	37.7 ± 0.2	37.6 ± 0.2
30	35 ± 7	26 ± 6[†]	24 ± 2[†*]	16 ± 7	8 ± 3[†]	6 ± 1[†*]	37.5 ± 0.3	37.7 ± 0.2	37.7 ± 0.2
40	35 ± 6	27 ± 5	24 ± 1[†*]	15 ± 8	7 ± 1[†]	6 ± 1[†*]	37.5 ± 0.4	37.7 ± 0.2	37.7 ± 0.2
50	35 ± 8	28 ± 5	25 ± 2[†*]	12 ± 4	7 ± 1[†*]	6 ± 1[†*]	37.5 ± 0.3	37.7 ± 0.2	37.7 ± 0.3
60	34 ± 7	30 ± 4	28 ± 1[†]	12 ± 5	7 ± 1[†]	6 ± 1[†*]	37.5 ± 0.3	37.7 ± 0.2	37.6 ± 0.3
70	34 ± 5	30 ± 3	28 ± 1[*]	12 ± 4	7 ± 1[†]	6 ± 1[†*]	37.5 ± 0.3	37.7 ± 0.2	37.6 ± 0.2
80	34 ± 5	32 ± 4	31 ± 3[*]	12 ± 3	7 ± 1[†*]	6 ± 1[†*]	37.5 ± 0.3	37.6 ± 0.2	37.5 ± 0.2
90	34 ± 6	33 ± 3	31 ± 3	13 ± 6	7 ± 1[†]	7 ± 1[†]	37.5 ± 0.3	37.6 ± 0.3	37.5 ± 0.2

[†]Significantly different ($p < 0.05$) from the baseline (Time = 0)
[*]Significantly different ($p < 0.05$) between T and other groups

Table 2. Changes in systolic (SAP), diastolic (DAP) and mean arterial pressure (MAP) after tramadol (T), detomidine (D) and detomidine plus tramadol (DT) administration

Time (min)	SAP			DAP			MAP		
	T	D	DT	T	D	DT	T	D	DT
0	116 ± 12	113 ± 17	112 ± 18	64 ± 16	63 ± 14	50 ± 7	80 ± 9	81 ± 12	73 ± 4
5	118 ± 17	113 ± 21	122 ± 17	54 ± 13	65 ± 15	65 ± 24	74 ± 17	84 ± 15	90 ± 24
10	108 ± 22	116 ± 22	108 ± 15	64 ± 8	64 ± 25	53 ± 21	79 ± 12	80 ± 24	70 ± 19
20	107 ± 12	106 ± 23	114 ± 26	61 ± 9	60 ± 21	60 ± 15	79 ± 8	78 ± 20	84 ± 20
30	119 ± 17	99 ± 9	112 ± 16	66 ± 10	49 ± 7	56 ± 18	87 ± 17	69 ± 12	67 ± 19
40	108 ± 17	100 ± 22	112 ± 12	58 ± 11	52 ± 20	59 ± 25	76 ± 19	69 ± 21	80 ± 23
50	96 ± 22	104 ± 21	103 ± 17	48 ± 10	54 ± 19	60 ± 14	70 ± 13	81 ± 18	81 ± 14
60	103 ± 11	100 ± 19	105 ± 7	58 ± 11	59 ± 19	63 ± 17	71 ± 10	77 ± 22	80 ± 13
70	98 ± 15	101 ± 12	109 ± 21	52 ± 10	51 ± 21	59 ± 13	72 ± 10	65 ± 23	74 ± 11
80	103 ± 17	101 ± 19	109 ± 20	60 ± 9	52 ± 18	64 ± 11	76 ± 8	73 ± 21	81 ± 8
90	111 ± 13	94 ± 12	90 ± 13	54 ± 15	47 ± 18	40 ± 6	78 ± 8	66 ± 13	64 ± 10

significant changes in RT and IAP were noted in all groups (Table 1, 2).

The sedative effect was evident within 5 min and lasted 70 min in D and DT treatments (Fig 1). T produced slight sedation at about 20 min. The result of ataxia also followed a similar pattern of sedation (Fig 2).

The onset of analgesia was within 5 min with both D and DT (Fig 3). Analgesia persisted throughout the whole period of this experiment, which was confirmed by both electrical stimulation and pinprick. T also produced an analgesic effect at about 10 and 20 min based on electrical stimulation and from 5 min to 80 min based on the pinprick. DT treatment induced a clearly greater analgesic effect than D alone based on electrical stimulation at 90 min and pinprick from 70 min to 90 min.

Two marked changes were observed in the blood analyses (Table 3). One was that the blood glucose increased from baseline with D treatment, and decreased steadily from 60 min. However, no similar changes were observed with T and DT treatments. The other change was that Hct and Hgb declined consistently from baseline with D and DT treatments.

GI motility dropped sharply from baseline to 30 min, and then recovered gradually after D and DT treatments.

During the experiments, complications such as muscle tremor, excitement, salivation, urination, sweating and penile prolapse were confirmed for all treatments (Table 4). One horse was excited within 5 min after the injection of T and DT. Three horses also show signs of salivation and penile prolapse in response to D.

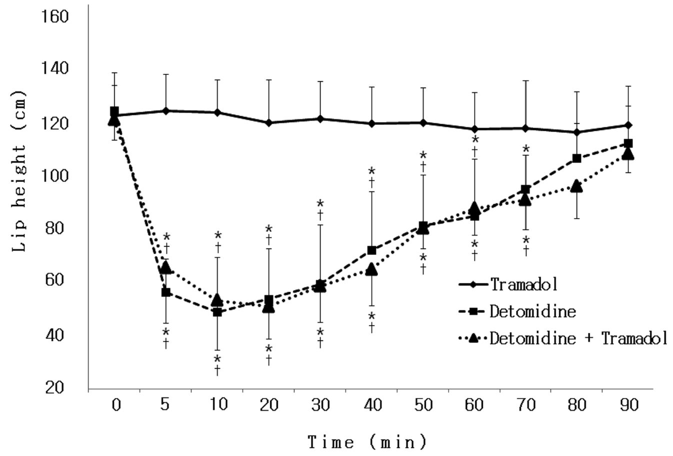

Fig 1. Lip height in six horses following intravenous administration of tramadol (T), detomidine (D) and detomidine plus tramadol (DT). †Significant differences ($p < 0.05$) from baseline (time = 0). *Significant differences ($p < 0.05$) between T and other two groups (D, DT). Significant differences between D and DT were not detected.

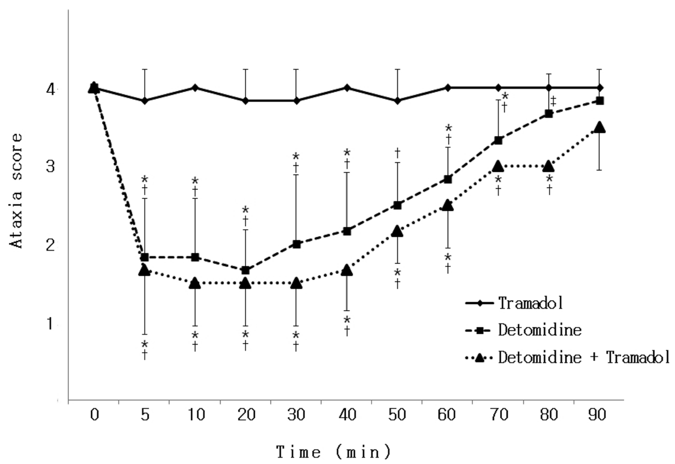

Fig 2. Ataxia score of intravenous administration of tramadol (T), detomidine (D) and detomidine plus tramadol (DT). †Significant differences ($p < 0.05$) from baseline (time = 0). *Significant differences ($p < 0.05$) between T and other two groups (D, DT). ‡Significant differences ($p < 0.05$) between D and DT.

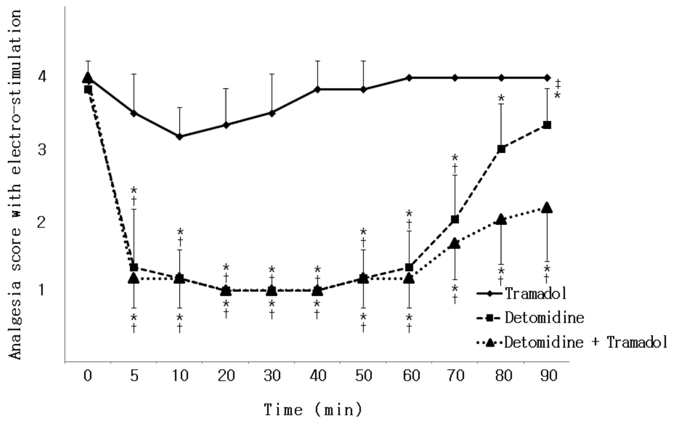

Fig 3. Analgesia score based on electro-stimulation in six horses following intravenous administration of tramadol (T), detomidine (D) and detomidine plus tramadol (DT). †Significant differences ($p < 0.05$) from baseline (time = 0). *Significant differences ($p < 0.05$) between T and other two groups (D, DT). ‡Significant differences ($p < 0.05$) between D and DT.

Table 3. Changes in glucose (Glu), hematocrit (Hct), hemoglobin (Hgb), sodium (Na^+), chloride (Cl^-), potassium (K^+), blood urea nitrogen (BUN) and gastrointestinal (GI) motility after tramadol (T), detomidine (D) and detomidine plus tramadol (DT) administration

Time (min)		0	30	60	90
Glu (mg/dl)	T	92.5 ± 4.3	88.7 ± 4.9	90.8 ± 3.5	90.7 ± 3.7
	D	93.2 ± 3.6	112.2 ± 13.2†*	111.0 ± 13.1*	106.8 ± 12.3*
	DT	93.8 ± 6.2	95.7 ± 7.7‡	100.3 ± 6.9‡	97.3 ± 6.3‡
Hct (%)	T	34.3 ± 5.2	32.5 ± 4.9	30.7 ± 4.0	29.5 ± 4.3
	D	34.5 ± 8.2	29.0 ± 4.2*	24.5 ± 2.7†*	26.3 ± 2.9†*
	DT	34.0 ± 5.3	29.8 ± 4.3	24.5 ± 3.0†	23.8 ± 3.0†
Hgb (g/dl)	T	11.7 ± 1.8	11.1 ± 1.7	10.5 ± 1.4	10.5 ± 1.2
	D	11.5 ± 2.6	9.8 ± 1.3	8.4 ± 1.0†*	8.8 ± 1.0†
	DT	11.0 ± 1.2	9.8 ± 1.4	8.4 ± 1.1†	8.1 ± 1.2†*
Na^+ (mEq/l)	T	136.8 ± 1.5	137.8 ± 1.6	137.5 ± 1.4	137.7 ± 1.4
	D	137.3 ± 0.8	137.3 ± 0.8	137.5 ± 1.0	137.5 ± 0.5
	DT	137.0 ± 1.7	137.5 ± 1.4	137.5 ± 1.4	137.1 ± 1.2
Cl^- (mEq/l)	T	101.8 ± 1.0	100.8 ± 1.0	100.5 ± 0.5	100.8 ± 0.8
	D	101.3 ± 2.9	100.5 ± 2.0	99.2 ± 2.2	99.5 ± 2.0
	DT	100.0 ± 0.9*	99.0 ± 1.3*	98.5 ± 1.0*	98.3 ± 0.8†*
K^+ (mmol/l)	T	4.1 ± 0.2	4.1 ± 0.2	4.1 ± 0.2	4.0 ± 0.1
	D	4.2 ± 0.6	4.2 ± 0.5	4.1 ± 0.5	4.0 ± 0.5
	DT	4.1 ± 0.2	4.0 ± 0.2	4.0 ± 0.3	3.9 ± 0.2
BUN (mg/dl)	T	9.0 ± 1.1	9.2 ± 1.2	9.2 ± 0.8	9.2 ± 0.8
	D	9.8 ± 1.8	9.2 ± 1.7	9.8 ± 1.7	9.8 ± 1.5
	DT	8.7 ± 0.8	8.8 ± 1.0	8.7 ± 0.8	8.5 ± 0.5
GI motility (score)	T	13.8 ± 1.7	12.2 ± 2.5	13.3 ± 2.5	14.2 ± 1.3
	D	15.0 ± 1.1	4.2 ± 0.4†*	6.5 ± 2.6†*	12.7 ± 1.5†
	DT	13.5 ± 2.1	4.5 ± 0.8†*	5.7 ± 1.5†*	9.8 ± 2.9*

†Significantly different ($p < 0.05$) from the baseline (Time = 0)
*Significantly different ($p < 0.05$) between T and other two groups (D, DT)
‡Significant differences ($p < 0.05$) between D and DT

Table 4. Complications after intravenous administration of tramadol (T), detomidine (D) and detomidine plus tramadol (DT) administration

Complications	T	D	DT
Muscle tremor	1/6	0/6	2/6
Excitement	1/6	0/6	1/6
Yawn	0/6	0/6	0/6
Chewing	0/6	0/6	0/6
Salivation	2/6	3/6	2/6
Urination	0/6	2/6	1/6
Sweating	0/6	1/6	2/6
Penile prolapse	2/6	3/6	3/6

Data indicates the number of horses showing complications based 6 horses.

Discussion

The α-2 agonists are commonly used in equine clinical practice. The effects of these drugs are mainly mediated by α-2 adrenergic receptors located in the locus ceruleus, the pons, and the lower brain stem. In particular, D is typically used in severely painful procedures, such as flank laparotomy and castration, and has been reported to be an efficient analgesic in a laminitis model for chronic pain and in a skin thermal stimulation model (1,11,18) and produced visceral analgesia in a cecal distension model (2). D is clinically used as both a sedative for diagnostic procedures and therapeutically to alleviate abdominal pain in horses (15).

As a μ-opioid receptor agonist, T has been widely used in humans and dogs, but the analgesic effect of IV administration of T in horses is unclear. Horses may be well sedated by α-2 agonists, as indicated above. However, they can still respond to stimuli such as pain and noise (9,12), and this response is not reduced by increasing the dose of α-2 agonists. For this reason, opioids are usually used in combination with these agonists in order to reduce undesirable responses.

HR and RR both decreased throughout the entire period following injection of D and DT. This was a typical effect of α-2 agonists according to previous studies (14,22). In contrast, IV administration of T caused only slight increases in

HR and RR at 10 min. RT and IAP normally increase following injection of D (22), but they showed no significant changes in any group in the present study. A clear detection of IAP was difficult because of the thickness of the horses' tails and temperament of warmblood horses.

The D group was sedated for 70 min in this study. In a previous comparative analysis of X and T, XT treatment showed a longer sedative effect than X alone (22). However, the sedative effect of the DT group was similar administration of D alone. The sedative effect of D is normally stronger and longer than that of X (19), so it was assumed that the combined effect of DT was due to the greater sedative effect of D. A significant ataxia was also observed in the D and DT treatments, but no ataxia was detected in the T group.

The most important finding in the present study was that DT treatment produced a longer analgesic effect than D alone. A previous study also reported that XT groups showed a longer analgesic result than X alone (22). The addition of T clearly boosted the analgesic efficacy of α-2 agonists. The T group of warmblood horses in the present experiment showed significant analgesia at about 10~20 min based on electro-stimulation and slightly at 5 to 80 min based on pinprick, whereas IV administration of 2 mg/kg T was reported to produce only mild analgesia in thoroughbred horses in a previous study (22). The difference in the analgesic effect of T seems to vary according to the breed of horse because thoroughbred horses are usually more excitable than warmblood horses.

An increase in blood glucose level is the major side effect after treatment with α-2 agonists. This is mediated by a decrease in insulin release from the β cells of the spleen. In this experiment, the D group showed a sharp rise in blood glucose 30 min after injection, but no significant increase in glucose was noted in the blood from the DT group. This may mean that T blocked the increase in blood glucose after D was injected. This may be considered a positive effect of the DT combination and warrants further study. Decreases in Hct and Hgb were confirmed following injection of the D and DT combination. This may also be mediated by α-2 agonists of a peripheral vasoconstrictive hemodynamic effect and warrants further study, too (24).

GI sounds, which were assessed by auscultation, decreased in frequency and intensity following D and DT treatments. However, no significant change in GI motility was noted in the T group. This can be explained by a decrease in motility of smooth muscle in the gastrointestinal tract caused by α-2 adrenergic agonists (20). This result is mediated by activation of visceral α-2 adrenergic receptors and inhibition of acetylcholine release (23).

Complications, such as salivation and penile prolapse, which are typical side effects of α-2 agonists, and muscle tremors, which are a side effect of T administration, were detected. If T does not cause the typical opioid-induced sympathetic stimulation, increased locomotion, and CNS excitation, it has the potential to be a useful analgesic in horses (18). A side effect of T seen in the present experiment was noted in one horse that showed CNS excitation and a secondary increase in HR and RR. The safe use of T in equine practice may therefore require slow intravenous or intramuscular injection to reduce adverse effects. Epidural injection of T (1 mg/kg) has been also reported to introduce mild analgesia without any adverse effects on behavior (16).

This study showed that a combination of D and T induced a similar sedation and a significantly longer analgesia than either D or T alone by IV administration. Although a return to the baseline level of analgesia after DT administration was not measured in this study, it may occur by a continued slow return to pre-treatment levels and the total analgesic duration may continue for about 2 hours. According to the results of this study, the DT combination could be used for diagnostic procedures and simple surgeries in standing horses. However, caution is needed when using the DT combination because there is a possibility of excitement, although the rate is low after IV administration of DT.

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말에서 detomidine과 tramadol의 정맥 투여에 의한 진정 및 진통효과

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요 약 : 본 연구는 승용말에 detomidine과 tramadol을 정맥 투여한 후 진정 및 진통 효과를 평가하기 위하여 실시되었다. 여섯 마리의 승용말에 각각 detomidine (10 μg/kg), tramadol (2 mg/kg) 및 detomidine/tramadol 병용 (각 10 μg/kg과 2 mg/kg) 투여하였다. 심박수, 호흡수, 직장체온, 간접동맥혈압, 위장관 운동에서 detomidine과 detomidine/tramadol 병용은 큰 차이를 보이지 않았다. 진정효과는 detomidine, detomidine/tramadol 병용 투여 후 5분에 관찰되기 시작하였으나, detomidine 및 detomidine/tramadol 병용간 유의한 진정효과의 차이는 나타나지 않았다. Detomidine과 detomidine/tramadol 병용은 투여 후 50분까지 유사한 진통효과를 나타내었으나, detomidine/tramadol 병용이 더 긴 진통효과를 나타내었다. Detomidine의 투여 후 혈당 수치가 투여 후 60분까지 증가하였으나, detomidine/tramadol 병용 투여 후의 혈당 수치는 증가하지 않았다. Tramadol과 detomidine/tramadol 병용 투여된 한 마리가 투여 5분 이내에 흥분된 행동을 보였다. 이상의 결과로부터 detomidine/tramadol 병용 투여가 기립상태에서 말의 간단한 외과적 처치와 진단을 위하여 유용하게 이용될 수 있을 것으로 판단된다.

주요어 : detomidine, tramadol, 진정, 진통, 말