Benefits of Incorporating Atipamezole in Medetomidine-ketamine Anaesthesia in Pigeons

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ABSTRACT

Smooth recovery from anaesthesia is vital in the restraint and surgery of animals, including birds. In birds, ketamine is often combined with alpha2 agonists, which are respiratory depressants to ensure safe or balanced anaesthesia. The timing of administration and the effect on recovery for specific alpha2 agonist antidotes in birds have not been widely investigated. This study was conducted to assess the reversal effects of atipamezole administered at the first sign of recovery on medetomidine-ketamine anaesthesia in pigeons. Twelve pigeons were administered with 1) medetomidine as a pre-anaesthetic at 120 µg/Kg, followed 10 min later by ketamine at 30 mg/Kg (MK group) and 2) Medetomidine and ketamine followed by atipamezole (MKA group). Medetomidine produced moderate sedation within 3.08 ± 0.21 min. Medetomidine–ketamine produced smooth and excitement free induction of anaesthesia in pigeons. Ketamine produced surgical anaesthesia within 4.58 ± 0.68 min. Duration of anaesthesia was 55.79 ± 4.51 min. Vital parameters – pulse rate, respiratory rate and cloacal temperatures decreased significantly after premedication as well as during anaesthesia in the MK group which served as the control. The MKA group significantly increased (P < 0.001) the pulse rate, respiratory rate and cloacal temperature compared to MK group that indicated a reversal effect of atipamezole. Atipamezole incorporated into medetomidine-ketamine anaesthesia produced quicker recovery (P < 0.01) in 44.17 ± 3.01 min compared to 62.5 ± 4.64 min in MK group. Atipamezole significantly (P < 0.05) decreased the duration of anaesthesia and duration of other recovery indices. Recovery was generally smooth in all birds. Atipamezole at 60 µg/Kg produced rapid recovery from anaesthesia 18 min earlier than MK group.

INTRODUCTION

Restraining of pigeons for various procedures, including physical examination, radiology, ophthalmology, and...
equipment. Injectable anaesthesia have the advantage of rapid induction, minimal equipment needed and lower cost (Athar et al., 1996; Sinclair, 2004) over inhalation anaesthesia. High metabolic rate and relatively weak hepatic glycogen storage in birds makes them prone to higher risk of fatal hypoglycaemia when recovery periods are lengthened (Atalan et al., 2002). In such cases reversal with specific antidotes offers a window for shortening recovery period even as the antidotes can be incorporated in the anaesthetic protocol.

The Medetomidine-Ketamine combination has been reported to produce safe and reliable anaesthesia and analgesia in pigeons (Lumeij and Deenik, 2003; Uzun et al., 2003; Kalpravidh, 1991). However, it significantly decreases the heart rate, respiration, and core body temperature (Lumeij and Deenik, 2003; Uzun et al., 2003; Kalpravidh, 1991). Since birds are incredibly susceptible to hypothermia because of their extensive air sac system; and low glycogen stores for energy production, they need extra care during anaesthesia to prevent them from adverse effects of hypothermia. Atipamizole is a specific medetomidine antidote available for reversal of the alpha2 agonist depressive effects on vital parameters. In various species of birds, recovery has been reported to begin from 30 seconds and completed within several min post-administration of atipamezole (Jalanka, 1991).

Unlike other commonly used injectable anaesthetics, ketamine is a dissociative anaesthetic that is associated with rough recoveries and insufficient muscle relaxation when administered as a sole anaesthetic agent in avian species (Durrani et al., 2008; 2009; Athar et al., 1996; Hess, 2010; Mandleker, 1988). Therefore, it is recommended to use ketamine in combination with alpha2 agonists or benzodiazepines. Xylazine and medetomidine are the two most common alpha2 agonists used in conjunction with ketamine (Lierz and Korbel, 2012). The significant advantages of alpha2 agonists are stress reduction, the anxiolytic effect (Jalanka, 1991; Ko and McGrath, 1995), muscle relaxation, and availability of specific antidote such as atipamezole and yohimbine to shorten the recovery period (Lierz and Korbel, 2012; Degerens et al., 1988). However, there is paucity of literature, regarding the timing of administration of atipamezole. Manufacturer’s protocols are often vague regarding the optimal time for atipamezole administration to achieve reversal effects and little is known about how the timing of reversal may affect anaesthetic outcome. One study reported prolonged recovery times with early reversal in reindeer, which showed an elimination half-life of 76 min for medetomidine and 60 min for atipamezole (Ranheim et al., 1997). These findings suggest that longer elimination half-life of medetomidine than atipamezole might have caused ‘resedation effect’. In a survey, approximately 5% of veterinarians reported resedation effects after atipamezole administration (Kaartinen et al., 2007). An interval of 15 to 40 min between administration of medetomidine and reversal with atipamezole was recommended for use in rabbits, but the authors provided no valid justification for such recommendation (Harcourt-Brown, 2004). Another study has reported early (10 min) and late (40 min) administration after medetomidine induction in mice (Baker et al., 2011), and 30 min after induction in pigeons (Pollock et al., 2001). Atlan et al. (2002) used atipamezole at 60 min after induction to reverse the adverse effects of medetomidine-butorphanol-ketamine anaesthesia in pigeons; a delayed recovery time (248 min) was observed. In pigeons, atipamezole reversal has been reported with variations in results (Atlan et al., 2002; Lumeij and Deenik, 2003; Pollock et al., 2001). Atlan et al. (2002) used a combination of medetomidine, butorphanol and ketamine in pigeons, which was reversed by atipamezole. The anaesthesia was not quickly reversed by administration of atipamezole as suggested by a prolonged recovery period of 248 min. They indicated that this combination was not suitable for surgery as indicated by violent wing flapping in four out of 8 birds and the birds needed restraining during recovery to prevent injury from wing flapping. Since atipamezole is a specific alpha2 adrenergic antagonist an addition of butorphanol, an opioid agonist might have delayed the effects of atipamezole, consequently delaying recovery (Atlan et al., 2002). Pollock et al. (2001) evaluated medetomidine alone and in combination with ketamine using atipamezole to reverse the effects. They did not recommend medetomidine alone in pigeons at the doses mentioned, and medetomidine-ketamine combination was only for minor procedure with unpredictable results. Lumeij et al. (2003) used medetomidine and ketamine anaesthesia, followed by reversal with atipamezole. They reported an uneventful rapid recovery after atipamezole injection. In their studies Pollock et al. (2001) and Lumeij et al. (2003) compared medetomidine-ketamine group with diazepam-ketamine and midazolam-ketamine groups, respectively. Since, diazepam, midazolam and medetomidine belong to different classes of drugs; diazepam-ketamine or midazolam-ketamine does not represent an actual positive control group to compare the results and to evaluate the reversal time and effects of atipamezole. Therefore, the purpose of this study was to determine and assess the reversal time and effects of atipamezole in medetomidine-ketamine anaesthesia in pigeons.

MATERIALS AND METHODS

Birds

Twelve randomly selected healthy pigeons of
either sex weighing 250-350 g were used. Pigeons were purchased from local poultry market. All the pigeons belonged to the same flock aged between 1 to 2 years. Only those birds, which were found active and healthy based on the physical examination, were used in this study. Pigeons were kept in a quiet experimental room measuring 8 × 10 × 10 feet at the Department of Surgery and Obstetrics, Faculty of Animal Husbandry and Veterinary Sciences, Sindh Agriculture University, Tando Jam. The birds were allowed at least one-week adaptation period before the experiment. The birds were identified individually through leg rings and fed with millet and wheat grains. Water was provided ad libitum. This study was reviewed and approved by the Board of Advanced Studies and Research, Sindh Agriculture University, Tandojam, Pakistan.

**Experimental design**

Two treatments were administered in a crossover design with a washout period of two weeks.

**Experimental procedures**

Pigeons were weighed at the start of the experiment and withdrawn from feed 3 h before anaesthetic induction. The injection site was cleansed and disinfected with antiseptic solution (methylated spirit). All the drugs were administered by intramuscular injection into the pectoral muscle using a 1 mL disposable syringe. Treatments included 1) Medetomidine (Domitor, Orion Corporation, Orion-Farmacos Espoo, Finland) administered as a pre-anaesthetic at a dose rate of 120 µg/Kg, followed by ketamine (Ketaset, Fort Dodge, Animal Health, Iowa, U.S.A) at a dose rate of 30 mg/Kg, administered 10 min after medetomidine, for the control group (MK). 2) Medetomidine and ketamine followed by atipamezole (Antisedan, Orion Corporation, Orion-Farmacos Espoo, Finland) (MKA), administered at first sign of recovery, indicated by muscle contraction (Lumeij and Deenik, 2003).

**Physiological parameters**

Pulse rate (beats/min), respiratory rate (breaths/min) and cloacal temperature (°F) were recorded before administration of the drug (as control) and then every five min until 120 min after medetomidine administration. Pulse rate was determined by auscultation of heart sounds with a stethoscope. Respiratory rate was determined by observing thoraco-abdominal movements with each respiration, and cloacal temperature was obtained by placing digital thermometer into the cloaca of bird for at least one min.

**Sedation parameters**

The onset of sedation, optimal sedation, degree of sedation and sitting times were recorded for each bird following administration of medetomidine. The onset of sedation was defined as the appearance of first effects of drug including repeated blinking of eyes and swallowing movements.

**Anaesthesia and analgesia parameters**

Nature of induction, the onset of induction, the start of surgical plane of anaesthesia, and duration of anaesthesia were recorded in each bird after administration of ketamine. Similarly, onset of analgesia and total duration of analgesia was recorded. Analgesia was measured through reaction to a noxious stimulus feather plucking (Mostachio et al., 2008).

**Reversal parameters**

Nature and duration of recovery, the start of atipamezole effect, sitting, standing, walking, and flying times were also recorded in each bird after atipamezole administration. The total duration of recovery was defined as the period between the first sign of recovery and walking (Lumeij and Deenik, 2003).

**Other observations**

Other observations such as blinking of eyes, lifting of the head, neck movement, wing flapping, righting, anal sphincter and beak tone, salivation, defecation, arrhythmias, onset and duration of recumbency were recorded for each bird.

**Statistical analysis**

The data obtained were analysed using repeated measure ANOVA with drug combinations being the treatment factor, and differences in physiological changes described across time, with the Graph Pad Prism 5.0 (Graphpad Software, San Diego, CA, USA). Statistical significance was set at 5% (P < 0.05). Data are presented as mean ± standard error of the mean.

**RESULTS**

**Physiological parameters**

In the current study, medetomidine significantly decreased the pulse rates (P < 0.001) (Fig. 1) and respiratory rates (P < 0.001) (Fig. 2) in all pigeons at 5 and 10 min respectively post-administration. Induction with Ketamine had an insignificant effect on the pulse rate, whereas it significantly (P < 0.05) increased the respiratory rate than pre-induction values at 10 min. Ketamine significantly decreased pulse rates (P < 0.05), increased respiratory rates (P < 0.05) at 20 min and decreased body temperature (P <
Administration of atipamezole significantly increased (P < 0.001) the pulse rate, respiratory rate and cloacal temperature compared to the control group (MK). However, pulse rate, respiratory rate and cloacal temperature could not return to baseline values in all treatments at 120 min (Figs. 1, 2 and 3). The lowest pulse rate, respiratory rate and cloacal temperature were 56.33 ± 2.59 beats/min at 80 min, 26.58 ± 1.82 breaths/min at 90 min and 95.65 ± 0.56 °F at 75 min, respectively during anaesthesia.

**Sedation**

Medetomidine produced a moderate degree of sedation in 11 out of 12 birds in both groups; One bird showed light sedation events during sedation. Medetomidine produced sitting in 10 out of 12 birds in both treatment groups before the administration of ketamine. The effect of medetomidine started with the blinking of eyes followed by swallowing, lowering of tail, head and wings, sitting and partial opening of beak.

In all the birds, onset of sedation started in 65.50 ± 3.90 seconds, optimal sedation in 3.08 ± 0.21 min, blinking of eyes in 65.0 ± 4.66 seconds, swallowing in 68.75 ± 4.94 seconds, lowering of tail in 2.5 ± 0.17 min, dropping of head in 3.92 ± 0.3 min, drooping of wings in 3.73 ± 0.25 min, sitting position in 4.57 ± 0.43 min, and partial opening of beak in 5.0 ± 0.35 min. Group results of the sedative parameters are presented in Table I.

**Anaesthesia**

All birds were in sitting position (due to sedative effects of medetomidine) before ketamine injection. Ketamine produced sternal recumbency followed by lateral recumbency, muscle relaxation, absence of tail movement,
the opening of beak, absence of toe and palpebral reflexes, closing of eyes, and relaxation of anal sphincter. Surgical plane of anaesthesia was produced in all birds, except bird 2 which had light anaesthesia.

In all birds, induction started in 72.5 ± 11.41 second, surgical anaesthesia began in 4.58 ± 0.68 min, the total duration of surgical anaesthesia was 55.79 ± 4.51 min, sternal recumbency was achieved in 1.38 ± 0.24 min, and lateral recumbency was achieved in all birds within 3.42 ± 0.64 min. Satisfactory muscle relaxation was produced in all birds in 4.44 ± 0.68 min. Group-wise results of the anaesthetic parameters are presented in Table II.

Table II. Duration (Mean ± SE) of various parameters during anaesthesia.

<table>
<thead>
<tr>
<th>Action</th>
<th>MK</th>
<th>MKA</th>
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<tbody>
<tr>
<td>Induction of anaesthesia (sec)</td>
<td>77 ± 21.7</td>
<td>67.5 ± 8.36</td>
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<tr>
<td>Start of surgical anaesthesia (min)</td>
<td>4.92 ± 1.18</td>
<td>4.25 ± 0.73</td>
</tr>
<tr>
<td>Duration of anaesthesia (min)</td>
<td>56.58 ± 7.98</td>
<td>55 ± 4.60</td>
</tr>
<tr>
<td>Sternal recumbency (min)</td>
<td>1.63 ± 0.46</td>
<td>1.13 ± 0.14</td>
</tr>
<tr>
<td>Lateral recumbency (min)</td>
<td>4.08 ± 1.24</td>
<td>2.75 ± 0.3</td>
</tr>
<tr>
<td>Satisfactory analgesia (min)</td>
<td>4.21 ± 1.16</td>
<td>3.42 ± 0.48</td>
</tr>
<tr>
<td>Total duration of analgesia (min)</td>
<td>90.3 ± 9.0</td>
<td>65.0 ± 5.8*</td>
</tr>
<tr>
<td>Muscle relaxation (min)</td>
<td>4.79 ± 1.2</td>
<td>4.08 ± 0.7</td>
</tr>
<tr>
<td>Opening of beak (min)</td>
<td>3.75 ± 0.80</td>
<td>3.37 ± 0.34</td>
</tr>
<tr>
<td>Closing of eyes (min)</td>
<td>4.06 ± 0.75</td>
<td>4.12 ± 0.71</td>
</tr>
<tr>
<td>Tail movement (min)</td>
<td>4.08 ± 1.18</td>
<td>3.46 ± 0.55</td>
</tr>
<tr>
<td>Pedal reflexes absent (min)</td>
<td>4.04 ± 1.16</td>
<td>4.58 ± 0.69</td>
</tr>
<tr>
<td>Palpebral reflexes (min)</td>
<td>4.28 ± 0.71</td>
<td>4.58 ± 0.69</td>
</tr>
<tr>
<td>Relaxation of anal sphincter (min)</td>
<td>4.70 ± 0.84</td>
<td>5.16 ± 0.68</td>
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</table>

* Significant difference (P < 0.05) between values of MK and MKA. For abbreviations, see Table I.

Satisfactory analgesia was observed in all the birds in 4.19 ± 0.66 min. Atipamezole significantly decreased (P < 0.05) total duration of analgesia to 65.00 ± 5.77 min in MKA group in comparison with 90.33 ± 9.0 min in MK group (Table II).

Recovery effect of atipamezole

Atipamezole significantly increased pulse rate, respiratory rate, and body temperature. On the other hand, it significantly decreased the total duration of analgesia from 90.33 ± 9.0 in MK (control) to 65.00 ± 5.77 min in MKA (atipamezole) group. Atipamezole decreased duration of various recovery indices compared to MK group, such as opening of eyes (3.50 ± 0.55 versus 8.33 ± 2.37 min), lifting of head (3.66 ± 0.93 versus 5.79 ± 1.36 min), recovery of beak tone (3.92 ± 0.80 versus 16.83 ± 3.98 min), tail movement (4.58 ± 1.19 versus 12.17 ± 2.79 min), wing reflexes (5.08 ± 1.03 versus 14.25 ± 2.86 min), righting reflexes (3.75 ± 0.56 versus 9.08 ± 2.40 min), toe reflexes (13.3 ± 2.10 versus 42.8 ± 4.10 min), return of muscle tone (8.50 ± 1.17 versus 17.5 ± 2.95 min) and anal reflexes (20.42 ± 3.57 versus 37.33 ± 3.27 min), sternal position (11.92 ± 1.46 versus 20.92 ± 3.00 min), standing (37.00 ± 3.26 versus 49.83 ± 4.69 min), and walking times (44.17 ± 3.01 versus 62.50 ± 4.64 min). Slight shivering and wing flapping were also observed in some birds after administration of atipamezole.

In summary, atipamezole produced quicker recovery in all birds compared to the control (MK) group (Table III).

Table III. Duration (Mean±SE) of various parameters during recovery period.

<table>
<thead>
<tr>
<th>Action</th>
<th>MK</th>
<th>MKA</th>
</tr>
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<tbody>
<tr>
<td>Opening of eyes</td>
<td>8.33 ± 2.37</td>
<td>3.50 ± 0.55</td>
</tr>
<tr>
<td>Lifting of head</td>
<td>5.79 ± 1.36</td>
<td>3.66 ± 0.93</td>
</tr>
<tr>
<td>Beak tone</td>
<td>16.83 ± 3.98</td>
<td>3.92 ± 0.80*</td>
</tr>
<tr>
<td>Tail movement</td>
<td>12.17 ± 2.79</td>
<td>4.58 ± 1.19*</td>
</tr>
<tr>
<td>Wing movement</td>
<td>14.25 ± 2.86</td>
<td>5.08 ± 1.03*</td>
</tr>
<tr>
<td>End of muscle relaxation</td>
<td>17.5 ± 2.95</td>
<td>8.50 ± 1.17*</td>
</tr>
<tr>
<td>Closing of anal sphincter</td>
<td>37.33 ± 3.27</td>
<td>20.42 ± 3.57</td>
</tr>
<tr>
<td>Righting reflexes</td>
<td>9.08 ± 2.40</td>
<td>3.75 ± 0.56*</td>
</tr>
<tr>
<td>Return of toe reflexes</td>
<td>42.8 ± 4.10</td>
<td>13.3 ± 2.10*</td>
</tr>
<tr>
<td>Sternal position</td>
<td>20.92 ± 3.00</td>
<td>11.92 ± 1.46*</td>
</tr>
<tr>
<td>Sitting position</td>
<td>33.08 ± 4.69</td>
<td>26.33 ± 2.93</td>
</tr>
<tr>
<td>Standing position</td>
<td>49.83 ± 4.69</td>
<td>37.00 ± 3.26*</td>
</tr>
<tr>
<td>Walking</td>
<td>62.50 ± 4.64</td>
<td>44.17 ± 3.01*</td>
</tr>
<tr>
<td>Flying</td>
<td>75.08 ± 4.77</td>
<td>66.33 ± 8.21</td>
</tr>
<tr>
<td>Total duration of recovery</td>
<td>62.50 ± 4.64</td>
<td>44.17 ± 3.01*</td>
</tr>
</tbody>
</table>

* For abbreviations and statistical details, see Table II.

Other observations

Other observations include defecation during sedation and anaesthesia in all birds, and slight shivering and wing flapping in few birds after the administration of atipamezole in the MKA group.

DISCUSSION

In this study, medetomidine produced satisfactory sedation followed by adequate anaesthesia and analgesia after administration of ketamine. Atipamezole produced quick and smooth recovery, increased the pulse rate, respiration and body temperature; however, it shortened the time of analgesia.

Alpha2 agonists significantly affect cardiovascular
function through stimulating central and peripheral adreno-receptors, resulting in decrease sympathetic tone and reduced norepinephrine outflow in the CNS. This decrease in norepinephrine dampens central sympathetic tone resulting in sedation and bradycardia. Moreover, CNS depression and reduced muscle activity lead to hypothermia and respiratory depression (Sinclair, 2004).

In this study, medetomidine significantly decreased the pulse rate, respiratory rate and cloacal temperature in all pigeons. Similar effects of medetomidine have also been reported in buzzards (Kilic and Paşa, 2009), pigeons (Atalan et al., 2002; Lumeij and Deenik, 2003; Uzun et al., 2003; Sandmeier, 2000; Ostrowski and Ancrenaz, 1995), mallard ducks (Machin and Caulket, 1998a, 1998b), cassowaries (Westcott and Reid, 2002), and in rock partridges (Alectoris graeca) (Ostrowski and Ancrenaz, 1995a).

In the present study, the time for onset of sedation was 65 ± 5 seconds in all the birds, and complete sedation occurred within 3 min of medetomidine administration. Sandmeier (2000) reported onset of sedation in pigeons within 2 min after medetomidine administration. Satisfactory sedation after medetomidine administration has also been reported in pigeons (Atalan et al., 2002; Uzun et al., 2003; Sandmeier, 2000; Langan et al., 2000) in zebra doves (Cherdchanpipat et al., 1989), in ostriches (Langan et al., 2000; Ostrowski and Ancrenaz, 1995), and in amazon parrots (Sandmeier, 2000).

The time of induction in all the birds averaged 72.5 ± 11.41 seconds (77 ± 21.7 versus 67.5 ± 8.36 in MK and MKA, respectively). Similar induction time of 1.6 ± 0.48 min has been reported in pigeon with detomidine and ketamine (Durrani et al., 2008). Induction with ketamine transiently increased the pulse rate (insignificant) and respiratory rate (significant) than pre-induction values. Increase in pulse and respiratory rates after ketamine injection has been reported in cockerel chickens (Mahmud et al., 2014) and in pigeons (Durrani et al., 2008). Furthermore, Pulse rate, and cloacal temperature were depressed during ketamine anaesthesia in this study. Similar results have been reported in various species of animals (Atalan et al., 2002; Lumeij and Deenik, 2003), in pigeons (Uzun et al., 2003), in mallard ducks (Machin and Caulket, 1998b), in peafowl (Athar et al., 1996), in chickens (Christensen et al., 1987; Mohammad et al., 1993; Valverde et al., 1993; Varner et al., 2004), in red-tailed hawks (Degernes et al., 1988), and ostriches (Ostrowski and Ancrenaz, 1995).

Ketamine produces dissociative anaesthesia with no muscle relaxation resulting in excitation during recovery in birds for surgical procedure. Thus, it is not suitable as a sole anaesthetic drug and has been recommended with a benzodiazepine or alpha2 agonists. Alpha2 agonists produce enough muscle relaxation and provide smooth recovery (Lierz and Korbel., 2012). In this study, ketamine and medetomidine produced smooth induction of anaesthesia with proper muscle relaxation and analgesia within 5 min of intramuscular administration. Surgical anaesthesia was produced within 5 min in 11 out of 12 birds, and one bird had light anaesthesia. Ketamine produced anaesthesia of 55.79 ± 4.51 min duration. Satisfactory duration of anaesthesia after ketamine administration have been reported in pigeons (Atalan et al., 2002; Lumeij and Deenik, 2003; Uzun et al., 2003), zebra doves (Kalpravidh, 1991), chickens (Mohammad et al., 1993), owls, psittacines, geese (Jalanka, 1991), and budgerigars (Heaton and Brauth, 1992).

Although cardio-pulmonary depression is one of the significant disadvantages of using alpha2 agonists, the advantage, on the other hand, is the availability of specific antagonists (e.g., atipamezole) to shorten the period of recovery (Durrani et al., 2008). Administration of atipamezole significantly increased the pulse rate, respiratory rate and cloacal temperature compared to control group. Similar effects of atipamezole have been reported in pigeons (Atalan et al., 2002; Lumeij and Deenik, 2003; Pollock et al., 2001), in amazon parrots (Sandmeier, 2000), in ostriches (Langan et al., 2000; Ostrowski and Ancrenaz, 1995), in owls, psittacines, geese (Jalanka, 1991), and cassowaries (Westcott and Reid, 2002). Findings of the current study showed that total duration of recovery in control (MK) group was 62.50 ± 4.64 min. Similar recovery time of 61.3 ± 17.26 min has been reported in pigeon with detomidine and ketamine (Durrani et al., 2008). On the other hand, longer recovery time of 96.2 ± 19.06 min with xylazine-ketamine was reported in pigeons (Durrani et al., 2009). Total recovery after atipamezole injection has been reported within 10 min in dogs (Pypendop et al., 1996) and 15 min in lambs (Ko and McGrath, 1995). Similarly, Forbes (1998) reported that atipamezole was given at the same dose as medetomidine rapidly reversed medetomidine-induced anaesthesia in other avian species. Although atipamezole at 250 µg per pigeon, a dose five times that of medetomidine, used in pigeons produced recovery from total anaesthesia within 248 ± 28.9 min. The prolonged recovery might be due to residual effects of butorphanol and ketamine in pigeons (Atalan et al., 2002). In this study the total recovery time was 44.17 ± 3.01 min after atipamezole compared with 62.50 ± 4.64 min in the MK (control) group.

In the current study, recovery was smooth in all the birds and was faster when atipamezole was used compared to MK (control) group (18 min difference). Although, the speedier recovery has been reported by other studies.
(Lumeij and Deenik, 2003; Kilic and Paşa, 2009); however, those studies used different class of drugs like diazepam or midazolam with ketamine, therefore, were not accurate. Positive controls to compare and determine the actual time difference and outcome effects.

CONCLUSION

The current study revealed that medetomidine ketamine combination produced satisfactory anaesthesia and analgesia. However, this combination depressed the cardio-respiratory system and produced severe hypothermia during anaesthesia. Therefore, it is necessary to keep the pigeons warm during anaesthesia. Atipamezole satisfactorily reversed anaesthesia and significantly shortened the recovery period by 18 min compared to the MK group; however, it also reversed the analgesic effects of the drugs.

Statement of conflict of interest

The author(s) declare(s) that there is no conflict of interests regarding the publication of this article.

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