Effect of intratesticular injection of lidocaine on cardiovascular responses to castration in isoflurane-anesthetized stallions

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Objective—To evaluate the effect of intratesticular administration of lidocaine on cardiovascular responses and cremaster muscle tension during castration of isoflurane-anesthetized stallions.

Animals—28 healthy stallions (mean ± SD age, 4.2 ± 2.8 years) with no testicular abnormalities that were scheduled for castration.

Procedure—Each horse was given acemepazine (20 µg/kg, IM), romifidine (60 µg/kg, IV), and butorphanol (20 µg/kg, IV). Anesthesia was induced with ketamine (2.5 mg/kg, IV) and midazolam (50 µg/kg, IV) and maintained with isoflurane (1.7% end-tidal concentration). After 10 minutes at a stable anesthetic plane, a needle was placed in each testicle and either no fluid or 15 mL of 2% lidocaine was injected; 10 minutes after needle placement, surgery was commenced. Pulse rate and arterial blood pressures were measured invasively at intervals from 5 minutes prior to castration (baseline) until 5 minutes after the left spermatic cord was clamped. The surgeon subjectively scored the degree of cremaster muscle tension. In 2 horses, lidocaine labeled with radioactive carbon (C14) was used and testicular autoradiograms were obtained.

Results—Compared with baseline values, castration significantly increased blood pressure measurements; intratesticular injection of lidocaine decreased this blood pressure response and cremaster muscle tension. In 2 horses, autoradiography revealed diffuse distribution of lidocaine into the spermatic cord but poor distribution into the cremaster muscle.

Conclusions and Clinical Relevance—In isoflurane-anesthetized stallions, intratesticular injection of lidocaine prior to castration appeared to decrease intraoperative blood pressure responses and cremaster muscle tension and may be a beneficial supplement to isoflurane anesthesia. (Am J Vet Res 2006;67:403–408)

In horses, castration is a frequently performed procedure. Most stallions are castrated, and the procedure may be carried out under local or general anesthesia; to achieve the latter, volatile agents such as isoflurane are commonly used for maintenance of anesthesia. Prevention or reduction of nociception is considered beneficial during anesthesia.1 Most volatile agents have little or no analgesic effect, and a marked nociceptive response to castration has been identified in equids when a volatile agent is used.2,4 Cardiovascular depression is a major adverse effect of volatile agent anesthesia in horses, and castration may decrease cardiac output further as a result of increased resistance to blood flow.2 Reduction of this cardiovascular response is probably beneficial to horses undergoing anesthesia and castration. Isoflurane induces dose-dependent cardiovascular and respiratory depression.3 In horses, limiting or decreasing nociception may decrease the concentration of isoflurane required for maintenance of anesthesia, which may decrease the extent of the adverse effects associated with isoflurane anesthesia. During castration, excessive cremaster muscle tension may make the surgical procedure more difficult, and any technique that decreases this tension would be beneficial. Local anesthetic agents may decrease the cremaster muscle tone either by causing direct inhibition of motor neuron impulses to the cremaster muscle or by reducing nociceptive impulses.

Prior to castration, lidocaine may be injected SC at the incision site, infiltrated in the spermatic cord, or injected into the testicle. Intratesticular injection offers the advantage of removal of the injection site with the testicle and thereby causes little interference with the surgical procedure. Intratesticular administration of local anesthetic agents in stallions undergoing castration has previously been described4 and used in clinical practice; nevertheless, the use of intratesticular injection of lidocaine in isoflurane-anesthetized stallions undergoing castration has not been fully investigated. The purpose of the study reported here was to evaluate the effect of intratesticular administration of lidocaine on cardiovascular responses and cremaster muscle tension during castration of isoflurane-anesthetized stallions.

Materials and Methods

Horses—To be included in this study, stallions had to be at least 2 years old, weigh > 300 kg, and have testicles with grossly normal anatomic features. Prior to admission to the study, a physical examination was performed to ensure that the horse was healthy. Twenty-eight stallions were available for participation in the study; prior to inclusion of each horse, informed consent was obtained from the owner or trainer. Among the horses, there were 17 Standardbreds, 6 Norwegian Coldblooded Trotters, 2 Thoroughbreds. 1

SABP Systolic arterial blood pressure
DABP Diastolic arterial blood pressure
MABP Mean arterial blood pressure
VAS Visual analogue scale

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Quarter Horse, 1 Icelandic horse, and 1 mixed-breed horse. The mean ± SD age of the horses was 4.2 ± 2.8 years (age range, 2 to 14 years), and mean weight was 466 ± 62 kg (weight range, 322 to 620 kg).

Anesthesia and instrumentation—Food was withheld from each horse for at least 12 hours prior to anesthesia, although free access to water was permitted. A catheter was placed in a jugular vein. Acepromazine (20 µg/kg, IM) was administered while each horse was in the stable; prior to the horse being walked to the induction area, sodium penicillin (10 X 10^6 units, IV) dissolved in sterile water and flunixin (1.1 mg/kg, IV) was administered. Romifidine (50 µg/kg, IV) and butorphanol (20 µg/kg, IV) were administered to each horse being walked to the induction area. Ketamine (2.5 mg/kg, IV) and midazolam (50 µg/kg, IV). After orotracheal intubation, each horse was placed in dorsal recumbency and the endotracheal tube was connected to a circle anesthesia system. Isoflurane vaporized in oxygen and air was administered; the gas mixture was continuously drawn from the rostral end of the endotracheal tube into an anesthetic monitor\(^\text{\textsuperscript{1}}\) that analyzed inspiratory and expiratory isoflurane, O\(_2\), and CO\(_2\) concentrations. Systolic, diastolic, and mean arterial blood pressures and pulse rate were measured directly through a catheter that was placed in a facial artery and connected to a pressure transducer\(^\text{\textsuperscript{2}}\) (zeroed at the level of the thoracic inlet). Intermittent positive pressure ventilation was started and adjusted to keep end-tidal CO\(_2\) concentration between 5% and 6%. Dobutamine\(^\text{\textsuperscript{3}}\) was infused with an infusion pump\(^\text{\textsuperscript{4}}\) at a rate required to maintain MABP > 60 mm Hg. A polyionic balanced electrolyte solution\(^\text{\textsuperscript{5}}\) was administered IV. End-tidal isoflurane was stabilized at 1.7% for the duration of the experimental period. The point at which isoflurane concentration became stable was designated T0 minutes. After a minimum of 10 minutes after the end-tidal isoflurane concentration was stabilized (ie, T10 minutes), the dobutamine infusion rate, fluid infusion rate, and ventilation were held constant (to avoid bias in blood pressure and pulse rate measurements) for the duration of the experimental period. Also at T10 minutes, a 0.8 X 40-mm needle was inserted in the middle of each testis; via these needles, either no injection was made (control group) or 15 ml of 2% lidocaine\(^\text{\textsuperscript{6}}\) (lidocaine group) was injected into each testicle. Both testicles in each horse received the same treatment. In 2 horses, a solution of lidocaine labeled with radioactive carbon \((\text{C}^{14})\) was used. All needle placements and injections were performed by the same anesthetist (HAH).

Surgery—All surgeries were performed by 1 of 2 designated surgeons (SL, TR). The skin over each testicle was incised, and the left incision advanced down to the common vaginal tunic. The subcutaneous tissue was stripped proximally with a gauze swab. The spermatic cord was clamped with crushing forceps before placement of a transfixing ligature proximal to the forceps. An Ochsner forceps was placed further distally on the spermatic cord to avoid leakage of blood from the testicle when it was removed; the spermatic cord was then transected with a scalpel immediately distal to the crushing forceps. The crushing forceps was kept closed for at least 5 minutes after transection of the spermatic cord. The procedure was repeated on the right testicle, after which the subcutaneous tissue and skin at each incision site were sutured with absorbable suture.

Recording of data—In each horse, the left testicle was always removed first; the data collected regarding blood pressures, pulse rate, and cremaster muscle tension were associated with removal of this testicle. The exact times of needle insertion, skin incision, and clamping and transection of the left spermatic cord were recorded. Values of SABP, DABP, MABP, and pulse rate were obtained from the anesthetic monitor; values were recorded 15 minutes after the end-tidal isoflurane concentration was stabilized (ie, T15 minutes) and at 30-second intervals thereafter. Baseline values were calculated as mean values of measurements made between T15 and the start of surgery. Surgery was commenced 20 minutes after the end-tidal isoflurane concentration was stabilized (ie, T20 minutes), and data were recorded until 5 minutes after the left crushing forceps was applied; the shortest data collection period extended from T20 minutes to T26.5 minutes, and the longest data collection period extended from T20 minutes to T29 minutes. Immediately after completion of surgery, the surgeon scored the degree of cremaster muscle tension encountered during removal of the left testicle by use of a 10-cm VAS; 0 cm represented no cremaster muscle tension, and 10 cm represented extreme cremaster muscle tension that prevented completion of surgery. For each horse, rectal temperature and general well-being were assessed, and surgical wounds were examined on the day after surgery and on subsequent days if the horse was not returned home. Three weeks after surgery, each client was contacted and interviewed by use of standardized questions to obtain information regarding possible postoperative complications.

Autoradiography—Radiolabeled \([\text{C}^{14}]\)lidocaine\(^\text{\textsuperscript{7}}\) (0.24 mg in 0.5 mL of ethanol; specific activity, 56 mCi/mmol) was evaporated to dryness by use of a gentle stream of N\(_2\) gas. Two milliliters of lidocaine \((20 \text{ mg/mL})\) was then added and vortexed, and the resultant solution was transferred back to the 20-mL vial of lidocaine. This resulted in a \([\text{C}^{14}]\)lidocaine solution with a radioactivity level of 1.25 µCi/mL. In each of 2 horses, radiolabeled lidocaine was injected in the left testis and the spermatic cord was clamped at 12.3 or 12.7 minutes after injection. The injected testis and spermatic cord were removed from each horse and immediately frozen in liquid nitrogen. The Ochsner forceps was kept closed to avoid loss of blood before the specimen was frozen. The testicle and spermatic cord were embedded in a 1% (wt/vol) gel of carboxymethylcellulose and frozen at –75°C in a bath of hexane and dry ice. Sagittal sections (30 µm thick) from different levels of the samples were prepared by use of a cryomicrotome\(^\text{\textsuperscript{8}}\) and collected at ~20°C on adhesive tape\(^\text{\textsuperscript{9}}\) according to the method of Ulfberg.\(^\text{\textsuperscript{9}}\) After freeze-drying at ~20°C for 24 hours, the sections were repositioned to radiographic film. Following exposure at ~20°C for 80 days, the radiographic films were developed.

Randomization and statistical analysis—The study was conducted as a prospective, randomized, partially blinded clinical study. On the basis of previous data, a power of 0.8 and a significance level of 5%, with the intention of detecting a difference of 12 mm Hg in MABP response between groups. Inclusion of at least 28 stallions was estimated to give a power > 90%. Randomization of horses to either the control or lidocaine group was done in blocks of 4 prior to the study. To have balanced group sizes, 2 horses were allocated to each treatment in each block. Just prior to the needle insertion, a numbered, sealed envelope was opened to reveal which treatment the horse was to receive; after the envelope was opened, no adjustments of infusion rates or ventilator settings were permitted. The surgeons were unaware of the treatment administered. For data analyses, a significance level of 5% and 2-sided tests were used throughout the study. Individual mean pulse rates and SABP, DABP, and MABP values for the baseline period (T15 to T20 minutes) and the intraoperative period were calculated for each horse. Differences between these individual means were calculated. Prior to further analysis, all data were tested for normality by use of a Shapiro-Wilk test. The baseline means for pulse rate, SABP,
DABP, and MABP were tested between groups by use of Student t tests. The individual differences in pulse rate, SABP, DABP, and MABP were analyzed within and between groups by use of Student t tests. The influence of surgeon on the cremaster muscle tension data was evaluated by visual inspection of the data and use of a Mann-Whitney test. To avoid surgeon-associated bias affecting the cremaster muscle tension data, the results were transformed to a common percentage scale; the results from each surgeon were corrected directly as a percentage by setting the largest observation of that surgeon to 100%. A comparison between groups was then performed by use of a Mann-Whitney test. Commercially available software was used for data handling and statistical analyses. Results are given as mean ± SD unless otherwise stated.

Results

Of the 28 horses in the study, 14 were allocated to the control group and 14 were allocated to the lidocaine group. In the control group, there were 9 Standardbreds, 3 Norwegian Coldblooded Trotters, 1 Thoroughbred, and 1 mixed-bred horse; the mean age of these horses was 3.5 ± 1.7 years, and the mean weight was 460 ± 47.6 kg. Horses in the control group received dobutamine at an infusion rate of 1.1 ± 0.47 µg/kg/min. Of the 2 surgeons involved in the study, 1 surgeon castrated 9 control horses and the other castrated 5 control horses. In the lidocaine group, there were 8 Standardbreds, 3 Norwegian Coldblooded Trotters, 1 Thoroughbred, 1 Quarter Horse, and 1 Icelandic horse; the mean age of these horses was 4.9 ± 3.5 years, and the mean weight was 471 ± 75 kg. Horses in the lidocaine group received dobutamine at an infusion rate of 1.1 ± 0.53 µg/kg/min. Of the 2 surgeons involved in the study, each castrated 7 lidocaine-treated horses.

In the lidocaine group, the dose of lidocaine administered was 1.3 ± 0.13 mg/kg. Induction of anesthesia, surgery, maintenance of anesthesia, and recovery from anesthesia of all horses proceeded without complications. No horse in either group moved during surgery, and no additional bolus of anesthetics was given to any horse. In the control group, 1 horse had mild colic postoperatively and in 1 horse, dehiscence of the right suture line developed a few hours after surgery. In the lidocaine group, 3 horses had high rectal temperatures the first line developed a few hours after surgery. In the lidocaine group, there were 9 horses that had high rectal temperatures; lidocaine group) or no treatment (control group) preoperatively.

Compared with findings in the control group, intratesticular injections of lidocaine prior to castration reduced the magnitude of these surgery-induced increases in blood pressure measurements, illustrating

![Table 1](https://example.com/table1.png)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Lidocaine group (n = 14)</th>
<th>Control group (14)</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Intraoperative value</td>
<td>Intraoperative value</td>
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<tr>
<td></td>
<td>Baseline value</td>
<td>Baseline value</td>
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<tr>
<td>Pulse rate (beats/min)</td>
<td>39 ± 6.3 (14)</td>
<td>41 ± 6.1 (14)</td>
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<tr>
<td>DABP (mm Hg)</td>
<td>53 ± 8.9 (14)</td>
<td>59 ± 8.8 (14)</td>
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<td>SABP (mm Hg)</td>
<td>98 ± 5.6 (14)</td>
<td>104 ± 10.6 (14)</td>
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<tr>
<td>MABP (mm Hg)</td>
<td>68 ± 4.8 (14)</td>
<td>74 ± 8.5 (14)</td>
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<td>99 ± 6.9 (13)</td>
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<tr>
<td></td>
<td>68 ± 4.5 (14)</td>
<td>92 ± 9.5 (14)</td>
</tr>
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</table>

The number in parentheses represents the number of horses from which data were available.

*End-tidal isoflurane concentration was 1.7%. Tchange from baseline to intraoperative value was significantly (P < 0.01) different from zero within both groups. Tchange from baseline to intraoperative value was significantly (P < 0.001) different between groups.

Discussion

In the study of this report, castration induced significant increases in blood pressure measurements in stallions anesthetized with isoflurane (1.7% end-tidal isoflurane concentration), compared with baseline values, but there were no significant changes in pulse rate. Compared with findings in the control group, intratesticular injections of lidocaine prior to castration reduced the magnitude of these surgery-induced increases in blood pressure measurements, illustrating
the analgesic effect of the treatment. Cremaster muscle tension may affect the ease with which castration can be performed; after an intratesticular lidocaine injection, the surgeons involved in the present study detected significantly less cremaster muscle tension, compared with subjective findings in untreated control horses. This suggests that intratesticular administration of lidocaine may facilitate the castration procedure.

Isoflurane has little analgesic effect, as demonstrated by the increase in blood pressure measurements during castration in the horses of the present study, and analgesic supplementation is warranted. Intratesticular administration of lidocaine improved anesthetic quality by increasing muscle relaxation, providing additional analgesia, and attenuating autonomic reflexes, which together with loss of consciousness are considered to be the properties of balanced anesthesia. Volatile agents induce dose-dependent cardiovascular and respiratory depression in horses; thus, decreases in the amounts of those volatile agents required by patients undergoing various procedures are probably beneficial. Administration of other agents that provide analgesia may decrease the requirement for volatile anesthetic agents. In the study of this report, intratesticular administration of lidocaine appeared to provide a noticeable analgesic effect in stallions undergoing castration. The injection is easily performed, and because the injection site is removed during the surgery, there is little associated risk of interference with surgery or wound healing. The present study did not reveal any increased risk of complications after intratesticular injection of lidocaine in horses.

In several studies of halothane-anesthetized horses, it has been found that different surgical stimuli induce an increase in MABP (compared with preoperative values) that is mediated by an increase in systemic vascular resistance; the increased resistance to blood flow results in a decreased cardiac output. This finding has also been determined in ponies when castration was applied as a surgical stimulus during anesthesia with halothane. In the present study, the intratesticular lidocaine injection decreased the castration-induced increase in MABP detected in control horses; the increase in MABP may be associated with a similar reduction in cardiac output during isoflurane anesthesia.

The testis, epididymidis, and content of the spermatic cord are innervated by visceral fibers that extend distally within the spermatic cord, whereas the scrotal skin, spermatic fascia, and cremaster muscle are innervated by somatic fibers that originate from outside the spermatic cord. During castration of unmedicated young pigs, cutting of the spermatic cord was the part of the procedure associated with most signs of pain; analgesia of the spermatic cord is thus warranted. Via an intratesticular injection, the local anesthetic is administered distal to the spermatic cord, which may seem to be an illogical treatment. In the present study, the autoradiograms of testicles treated with an intratesticular injection of radiolabeled lidocaine indicated that the drug was readily transported proximally in the spermatic cord and subsequently distributed diffusely within the spermatic cord. Distribution within the testicular parenchyma was sparse. Lidocaine did not cross the parietal layer of the tunica vaginalis into the cremaster muscle to any noticeable extent. In the horses of the lidocaine group, there was a significant blood pressure response to castration, which indicates that the local anesthetic block was not complete. Autoradiography revealed that the observed analgesic effects were most likely caused by inhibition of visceral afferent nerve fibers in the spermatic cord, whereas the somatic nociception from the skin incision and clamping of the cremaster muscle was not affected. The autoradiograms indicated that neither the cremaster muscle nor its innervation contained an appreciable amount of radiolabeled lidocaine; the reduction in cremaster muscle tension detected by the surgeons in association with intratesticular administration of lidocaine was probably caused by a decrease in visceral nociception and not because of direct paralysis of the cremaster muscle.

Extracellular fluid in the testicular parenchyma is absorbed into lymphatic capillaries, which drains into larger lymphatic vessels in the spermatic cord. In rams, the rate of lymph flow from each testis is high, compared with flow per unit weight from other parts of the body. Increased hydrostatic pressure that results from lidocaine injection into the testicular parenchyma...
probably increases lymph flow further, facilitating distribution of lidocaine into the spermatic cord. A rapid lymphatic transport of methylene blue after intratesticular injection has been identified in horses; 1.5 minutes after injection, methylene blue was detected 20 cm proximal to the testis. An autoradiographic study in young pigs in which 1% lidocaine with 5 μg of adrenalin/mL was injected into testicles revealed that there was a higher concentration of lidocaine in the spermatic cord 3 minutes after intratesticular injection, compared with the amount present in the spermatic cord 10 minutes after injection. This finding illustrates the speed of distribution of lidocaine into the spermatic cord after intratesticular administration. These results indicate that the maximum analgesic effect of lidocaine may have been attained sooner than 10 minutes after intratesticular injection in horses.

Systemically administered lidocaine may be used for analgesia during anesthesia of equids. In the present study, there was a rapid distribution of lidocaine into the spermatic cord of stallions, and a previous study revealed rapid systemic absorption of various drugs after intratesticular administration in different mammals. Systemic effects may have caused some of the analgesia detected in horses in our study. When lidocaine is administered IV for analgesia in ponies, a starting bolus dose of 2.5 to 5 mg/kg administered IV has been suggested. The mean ± SD dose of lidocaine given to the horses in our study was 1.3 ± 0.13 mg/kg. On evaluation of the autoradiograms of tissue sections from testicles removed during castration, it was evident that there was still an appreciable amount of lidocaine in the testicles at that time. We believe that the systemic analgesic effect of lidocaine in the horses of the present study was of minor importance and that the effects on blood pressure measurements were mainly a result of local analgesia.

In our investigation, broad inclusion criteria were used to achieve a representative sample of the hospital equid population; therefore, breed, age, and weight varied among the horses included in the study. This variation among horses could potentially have biased the data collected in our study; however, because the breeds, age, and weights of horses and the dobutamine infusion rates did not differ much between the lidocaine and control groups, a possible bias was considered unlikely. Ideally, 1 surgeon should have performed all surgeries, but this was not possible in our situation. The shortcoming of having 2 surgeons perform the castrations was reduced because both surgeons contributed almost equally in both groups. Cremaster muscle tension was scored subjectively; as a consequence, the influence of the surgeon on the results was investigated further and a difference between surgeons was identified. To overcome this, each surgeon’s VAS scores were corrected directly as a percentage of the greatest observation of that surgeon, thereby reducing variability between surgeons.

A needle insertion without subsequent injection was chosen as the control treatment because pilot investigations revealed that bleeding after the needle insertion revealed whether an injection had been made or not. Injection of saline solution was not used in our study because we intended to compare the use of lidocaine injection with a clinical alternative, namely the injection of no fluid. Masking of treatments to the anesthetist who administered them was not considered necessary because when treatment allocation for each horse was revealed, no adjustments that could influence cardiovascular variables were permitted. A crossover study design in which each horse would receive an injection of lidocaine in one testicle and not the other was considered for our study but was not implemented because of concerns regarding carryover effects between removals of the 2 testicles.

In the present study, cardiovascular responses were considered to be indicators of nociception; such responses have traditionally been used as indicators of nociception during anesthesia. In a previous study in isoluflurane-anesthetized horses, cardiovascular responses were compared with electroencephalographic responses, and cardiovascular responses were found to be more sensitive indicators of nociception. Experimental work performed in rodents revealed that blood pressure measurements and pulse rate may increase or decrease in response to nociception. During ocular or genital surgery, a vasovagal response may also decrease blood pressure and pulse rate in other species. In the present study, castration induced an increase in blood pressure measurements in all horses in both groups. A possible reason is that all horses were anesthetized at the same end-tidal isoluflurane concentration and the intention was to attain similar physiologic conditions in all horses prior to castration. No significant difference in pulse rate response to castration was detected within or between groups. However, results of a previous study indicated that pulse rate decreased from baseline values during castration of isoluflurane-anesthetized horses. A possible explanation for the difference between these 2 studies may be that in that previous study, an end-tidal isoluflurane of 1.4% was used; therefore, the CNS of those horses was less depressed and thus may have been more responsive to nociception that could facilitate a vasovagal response. Another difference was that glycopyrrolate was administered to the horses in the previous study, which may have confounded the results. In the present study, we believe it is fair to assume that the smaller increase in blood pressure measurements in stallions during castration indicated comparatively less intense nociception.

Our data suggest that following intratesticular administration, lidocaine distributes into the spermatic cord of horses in concentrations that are sufficient to decrease nociception through visceral nerves, whereas nociception from the cremaster muscle, scrotal skin, and spermatic fascia that is conducted through somatic afferent nerve fibers is probably unaffected. An intratesticular injection of lidocaine in isoluflurane-anesthetized horses prior to castration appears to decrease cardiovascular responses to the surgical stimulus and cause less cremaster muscle tension during surgery.

a. Datex-Engstrom AS/3, Helsinki, Finland.
c. Dobutamine, Abbott Laboratories, North Chicago, Ill.
d. Asena GW, Alaris Medical Systems, Basingstoke, UK.
e. Ringer acetat, Fresenius Kabi, Oslo, Norway.
f. Xylocain, Astra-Zeneca, Oslo, Norway.
g. American Radiolabelled Chemicals Inc, St Louis, Mo.
h. PMV 450 MP, Palmstierna Mekaniska Verkstad, Stockholm, Sweden.
i. 3M Scotch Brand tape, No. 821, 100 mm × 66 m, Saint Paul, Minn.
j. Agfa Structurix, D7 DW ETE, Agfa-Gevaert NV, Belgium.
l. Jump, version 5.01a, SAS Institute Inc, Cary, NC.

References