Comparison of SPO_2, hearth rate and body temperature values in abdominal compartment syndrome in a rat model with intraabdominal sepsis and intraabdominal hypertension

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Abdominal compartment syndrome (ACS) is a constellation of the physiologic sequel of increased intraabdominal pressure (IAP). Although initially thought to affect mostly trauma and surgical patients, intraabdominal hypertension (IAH) and ACS have also been identified in patients such as in ones with acute pancreatitis or with massive fluid replacement since the late 1980s and 1990s.[1] Persistent IAH may cause ACS impairing cardiac, respiratory, gastrointestinal, hepatic, renal, and neurological homeostasis. The ACS often results in the development of multiple organ failure (MOF), and it has consistently been reported to have a high mortality ranging from 25-75%.[2,3] In the presence of IAH, gut dysfunction resulting in increased gut mucosal permeability and subsequent bacterial translocation and sepsis may play an important role in the development of MOF in the critically ill patients. The purpose of this study was to investigate systemic hemodynamic changes by routinely used parameters, such as peripheral oxygen saturation (SpO_2), heart rate (HR), and body temperature in the presence of ACS caused by IAH increased to 25 mm Hg in a rat model with intraabdominal sepsis, and the effects of abdominal decompression.

Thirty adult female, specific pathogen free Sprague Dawley rats (200–220 g) purchased from Selcuk University, Experimental Research Laboratories (Konya, Turkey) were included in the study. Animals were isolated from males and were housed in standard laboratory cages and were allowed free access to food and water until 12 hours before the surgical procedure. All procedures mentioned were approved by the local ethical authority. The study protocol was designed in accordance with the 1996 revised form of The Guide for the Care and Use of Laboratory Animals published by the United States National Institutes of Health. The 30 rats were randomly assigned into one of 2 experimental groups: control group (n=15), and study group (n=15). In the study group, intraabdominal sepsis and ACS were developed, and then, abdominal decompression was performed. The rats in the study group were anesthetized with ketamine hydrochloride (100 mg/kg, intramuscular, Ketalar amp, EIP, Istanbul, Turkey). Following this, they were placed on a heating pad, the abdominal region was shaved and sterilized with povidone iodine solution. Thereafter, intraabdominal sepsis was developed by 1 cc solution containing 107 *Escheria coli* (*E. coli*) injected intraperitoneally without increasing IAP. The SpO_2, body temperature, and HR were measured at 120 and 240 minutes after injection with a portable pulse-oximeter (Nelcor Puritan, Bennet, USA). A plastic tube, 5 mm in thickness, was prepared with one end tightly secured to a serum physiologic bag, and placed intraperitoneally in a manner so as not to leak fluid in aseptic conditions at the end of the 240 minutes waiting period. A linear mercury manometer was placed between the plastic tube and serum physiologic bag, and the IAP was measured throughout the study. Serum physiologic bag was raised until IAP raised to 25 mm Hg and the bag was held at that level throughout 60 minutes. After that, the serum bag was lowered, and IAP was reduced by removal of the fluid sent into the peritoneum throughout the plastic tube. The same parameters were measured just after the fluid in peritoneum was removed and after 30 minutes. In control group, rats were anesthetized in the same manner, but *E. coli* solution was not injected, and IAP was not elevated; and same parameters were measured.

Values were classified as: control group (CG), S-120 (septic rats values at 120th minute), S-240 (septic rat values at 240th minute), SH-30 (septic + intraabdominal hypertensioned rats values at 30th minute), SH-60 (septic + intraabdominal hypertensioned rats values at 60th minute), SH-120 (septic + intraabdominal hypertensioned rats values at 120th minute), SH-240 (septic + intraabdominal hypertensioned rats values at 240th minute), SR-0 (peritoneal fluid removed rats values, reperfusion), and SR-30 (values at 30th minute after removing peritoneal fluid). Data were analyzed using SPSS (Statistical Package for the Social Science, version 10.0). Results were expressed as mean ± standard error of mean. One-way analysis of variance was used to determine the significance of any differences between the groups. Statistical comparisons between groups were performed by nonparametric Mann–Whitney U test and the difference was considered to be significant when $p<0.05$.

During sepsis development. The values of SpO_2, HR and temperature of study group were measured 120 and 240 minutes after *E. coli* solution injection.

The SpO_2 level of study group minimally decreased according to the level of control group. This decrease was not statistically significant. Whereas, HR and temperature significantly increased in the S-120 and S-240 comparing the control group (p<0.05). After ACS development: The SpO_2 levels gradually decreased 60, 120, and 240 minutes after ACS development, and all of these levels were significantly different than the values of control group and than the values of study group S-120 and S-240 (p<0,05). HR values of the study group gradually increased after ACS development; and all of the values 30, 60, 120, and 240 minutes after development of ACS were significantly higher than the values of control group, and the values of S-120 and S-240 (p<0.05). Body temperature of the study group decreased 30 minutes after ACS development; but it gradually increased again 60, 120, and 240 minutes after development of sepsis and ACS, and 120 minutes after ACS development it reached to the same level with the values of S-240. After decompression: Immediately after abdominal decompression, the SpO_2 level increased and then continued to increase. It was significantly lower than the values of control group, but is was significantly higher than the values of study group at 120 and 240 minutes after ACS development (p<0.05). Thirty minutes after decompression, SpO_2 level of the study group come close to the level of control group. HR decreased immediately after decompression and continued to decrease gradually. Although, both values immediately and 30 minutes after decompression were statistically higher than the control values (p<0.05), the mean HR of SR0 was significantly lower than the mean value of S-240; and the mean HR of study group SR-30 was significantly lower than the mean values of SH-60, SH-120, and SH-240. Body temperature also decreased immediately after decompression. Although both values at immediately and 30 minutes after decompression were statistically higher than the control values (p<0,05), the level of SR-0 was significantly lower than the levels of S-120 and S-240; and the level of study group SR-30 was significantly lower than the levels of SH-60, SH-120, and SH-240, and it was at the same level with the level of study group 30 (**Table 1**).

After decompression. Immediately after abdominal decompression, the SpO_2 level increased and then continued to increase. It was significantly lower than the values of control group, but was significantly higher than the values of study group at 120 and 240 minutes after ACS development (p<0.05). Thirty minutes after decompression, SpO_2 level of the study group come close to the level of control group. The HR decreased immediately after decompression and continued to decrease gradually. Although, both values immediately and 30 minutes after decompression were

Table 1 - Peripheral oxygen saturation, hearth rate and temperature values of the groups. Statistically significant differences were shown with superscript signs.

Groups	SpO_2 (%)	Heart rate (pulse/minute)	Temperature (^{O}C)
Control	96.60 ± 0.26	104.10 ± 0.65	36.78 ± 0.10
After sepsis development			
Study group S-120	96.88 ± 0.21	112.04 ± 1.02*	38.17 ± 0.24*
Study group S-240	96.87 ± 0.22	114.04 ± 1.22$^{\neq}$	39.17 ± 0.25$^{\neq}$
After increase of intraabdominal pressure			
Study group SH-30			
Study group SH-60	95.30 ± 0.44	116.80 ± 1.10$^{\beta}$	37.98 ± 1.02$^{\beta}$
Study group SH-120	94.10 ± 0.54$^{\alpha,\mu}$	123.20 ± 1.19$^{\alpha,\mu}$	38.71 ± 0.10$^{\alpha,\mu}$
Study group SH-240	90.20 ± 0.87$^{\chi}$	131.30 ± 1.02$^{\chi}$	39.05 ± 0.16$^{\chi}$
After decompression	89.60 ± 0.95$^{\delta}$	135.10 ± 1.32$^{\delta}$	39.05 ± 0.65$^{\delta}$
Study group SR-0			
Study group SR-30	95.30 ± 0.44$^{\varepsilon,\theta,\varphi}$	116.80 ± 1.10$^{\varepsilon,\theta,\varphi}$	37.98 ± 1.02$^{\varepsilon,\theta,\varphi}$
	94.10 ± 0.54$^{\alpha,\mu}$	123.20 ± 1.19$^{\pi,\eta,\rho}$	38.71 ± 0.10$^{\alpha,\mu}$

*Significant difference between group S-120 and control group; $^{\neq}$Significant difference between group S-240 and control group; $^{\beta}$Significant difference between group SH-30 and control group; $^{\alpha}$Significant difference between group SH-60 and control group; $^{\mu}$Significant difference between group SH-60 and group SH-30; $^{\chi}$Significant difference between group 1-120 and group 1-60; $^{\delta}$Significant difference between group SH-240 and group SH-60; $^{\varepsilon}$Significant difference between group 2-0 and group 1-60; $^{\theta}$Significant difference between group SR-0 and group SH-120; $^{\varphi}$Significant difference between group SR-0 and group SH-240; $^{\pi}$Significant difference between group SH-30 and group SH-60; $^{\eta}$Significant difference between group SR-30 and group SH-120; $^{\rho}$Significant difference between group SR-30 and group SH-240.

statistically higher than the control values ($p<0.05$). The normal values of IAP are sub atmospheric to 0 mm Hg. The IAH is defined as IAP 10 mm Hg that persists without the characteristic pathophysiology of ACS. Recent animal data suggested that prior shock and resuscitation may actually reduce the threshold levels of IAP that cause systemic manifestations of ACS.[4] However, it is generally accepted that ACS occurs when IAP is increased above 20-30 mm Hg with multisystem involvement. Therefore, an IAP at 25 mm Hg was chosen to evaluate systemic effects of ACS in this study. Most systemic complications of ACS developing in such patients are consequences of the loss of intestinal barrier function allowing for an increased mucosal permeability, bacterial translocation, and sepsis.[1,5] Intraabdominal sepsis already negatively affects hemodynamic parameters. These various systemic detrimental effects of IAH appear gradually, starting at pressures of <10 mm Hg, before clinical manifestations of ACS become evident.[1] Devices to monitor IAP are routinely used in patients at risk in some centers. In addition, some sophisticated hemodynamic and pulmonary parameters were used such as cardiac index, pulmonary artery occlusion pressure, lactate, peak airway pressures, cardiac output, pulmonary capillary wedge pressure, static pulmonary compliance, intrathoracic blood volume, and total circulating blood volume in clinical and experimental studies.[2,3] Therefore, some routinely used parameters are necessary to follow hemodynamic and systemic changes of patients before development of systemic complications of overt ACS, as mortality and morbidity are too high after development of these complications. Decrease of SpO_2, and an increase of HR, 2 routinely used parameters in clinical practice, may be a warning in the early period for development of pulmonary and cardiovascular complications of ACS in the patients with minor abdominal trauma, or massive fluid replacement. Therefore, these parameters were chosen for this study. In addition, body temperature was evaluated as rat model with intraabdominal sepsis was used. Peripheral arterial oxygen saturation and HR were measured by pulse oximetry. Arterial hemoglobin saturation can be continuously and noninvasively evaluated by pulse oximetry.

In this study, intraabdominal sepsis resulted in a significant increase of HR and body temperature according to the control group. It also caused a decrease of SpO_2 level, but this difference was not statistically significant. Development of ACS in addition to sepsis caused a significant increase of HR, and a significant decrease of SpO_2 level according to both control group and the study group after sepsis development. After abdominal decompression, SpO_2 significantly increased, HR and body temperature were significantly decreased, and all of these parameters came back to the level of the study group 30 minutes after an increase of IAP, but it was still significantly higher than the level of study group before an increase of IAP. Therefore, it was thought that abdominal decompression maybe life saving in patients with sepsis in whom ACS complications develop. The PaO_2 significantly decreased 60 minutes after an increase of IAP to 25 mm Hg in the rats with sepsis, and HR significantly increased 30 minutes after increase of IAP to 25 mm Hg. These detrimental effects came back immediately after abdominal decompression was carried out 240 minutes after an increase of IAP to 25 mm Hg. These findings indicate that IAH up to 25 mm Hg may cause ACS complications in a too short time, such as 30 and 60 minutes, and abdominal decompression carried out in the early period improves these detrimental effects. Therefore, early diagnosis, and early treatment of ACS are essential to prevent morbidity and mortality.

In conclusion, SpO_2 and HR may be useful together with following the level of IAP to suspect ACS. An increase of IAP to 25 mm Hg may cause development of ACS complications in a too short time in the presence of intraabdominal sepsis. These detrimental effects may come back if abdominal decompression is carried in the early period.

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Effects of different bolus doses of remifentanil on laryngeal mask airway insertion during day-case surgery

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Many investigators have shown that the use of the laryngeal mask airway (LMA) was associated with decreased anesthetic requirements and reduced incidence of postoperative side effects compared with the tracheal tube, and could lead to a more rapid emergence from anesthesia during day-case surgery. Although propofol is the induction agent most commonly used to facilitate the placement of LMA on day-case surgery, when used alone in an unpremedicated patient, its requirements for successful LMA insertion often exceed the recommended induction dose of 2.5 $mg.kg^{-1}$, and this doses may be associated with considerable adverse effects.[1] Remifentanil, an ultra-short-acting potent opioid, can be a more appropriate choice for use as an adjunct to induction with propofol and can provide adequate depth of anesthesia to allow LMA insertion during day-case surgery. This study was therefore designed to compare the effects of 3 different bolus doses of remifentanil co-administered with propofol on LMA insertion.

After approval from our hospital ethics committee, 100 American Society of Anesthesiologists physical status I or II patients, aged 18-55 years, who were undergoing day-case surgery, where the use of LMA was indicated, consented to participate in this prospective randomized double-blinded study. After standard monitoring was applied, all patients breathed oxygen, $6l.min^{-1}$, via face mask for 3 minutes and anesthesia was induced with propofol 2.5 mg kg^{-1} IV, given over 30 seconds. Following the completion of propofol induction, all the patients were randomly allocated into 4 equal groups. While Group I received 10 ml of saline, Group II, III, and IV received remifentanil 0.5 $\mu g.kg^{-1}$, 1 $\mu g.kg^{-1}$ and 1.25 $\mu g.kg^{-1}$ diluted with saline to 10 ml, over 60 seconds through an intravenous cannula. The patient was not ventilated, and 90 seconds following the start of induction, ease of jaw opening was assessed and, if possible, LMA insertion using the standard technique was attempted. If the first attempt was unsuccessful a second and third trial were performed following 30 seconds after another dose of propofol 0.5 $mg.kg^{-1}$ bolus was given for each trial and assisted ventilation was performed by face mask between each attempt. After 3 unsuccessful attempts succinylcholine 25 mg was given to facilitate the insertion. The overall insertion conditions during LMA insertion at the first attempt and time to successful LMA insertion (the time from induction to successful LMA placement) were recorded. Following successful LMA insertion, anesthesia was maintained with 2% sevoflurane and 70% nitrous oxide in oxygen. During this time, mean arterial pressure (MAP) and heart rate (HR) were recorded every minute for 5 minutes after successful insertion of the LMA. If the patient's MAP decreased >25% from the preinduction value during the induction period, a rapid IV infusion of lactated ringer's solution was administered at a rate of 50 ml/min. If the hypotension persisted over 60 seconds, 5-10 mg ephedrine IV bolus was administered. Bradycardia (HR <50 bpm) was treated with atropine 20 mg kg^{-1} IV. Duration of apnea (the time from induction until the first spontaneous breath) was recorded. Apneic patients were manually ventilated until the return of spontaneous breathing. Surgery was only allowed to commence after the return of spontaneous respiration. The anesthetic technique was standardized for all patients. At the end of the operation, the LMAs were removed and the presence of blood on the mask was noted. Once fully awake, the patients were interviewed by a blinded observer who asked whether they had a sore throat, or hoarseness of voice. To detect a difference in the proportion of patients with successful insertion in the remifentanil groups (90%) compared with the control group (60%), 24 patients in each group were required to achieve 80% power with an alpha error of 5%. Data were analyzed by using the Chi-square test and one-way ANOVA with Tukey for post-hoc comparisons. We used SPSS version 13.0 to assess the results. The groups did not differ significantly with respect to physical characteristics, clinical history, and duration of operation. The LMA was successfully inserted without requiring any succinylcholine in all the patients. The first attempts were found successful in 64%, 76%, 96%, and 96% patients in Group I, II, III and IV. There was no significant difference in coughing, but the incidence of other adverse responses at the first attempt was significantly reduced in Group III and IV than in Group I (p=0.002, p=0.000). There was no significant difference between Group III and IV in terms of

these adverse responses. Time to successful insertion was significantly longer (p=0.001, p=0.000) in Group I than Group III and IV. Duration of apnea in Group I was significantly shorter (p=0.021, p=0.000) than other groups, however, there was no significant difference among remifentanil groups. Six patients out of 25 in Group I had some blood on the LMA after removal, compared with 0 patients in Group III and IV (p=0.022) (**Table 1**). However, in the postoperative period, there was no significant difference among the groups in terms of sore throat, hoarseness of voice and discharge time. Baseline hemodynamics did not differ among the 4 groups. Compared with baseline, the average decrease in MAP during the study period was 22%, 29%, 38% and 38% in Group I, II, III and IV. Patients in Group I had a slight increase in HR after the induction, whereas the HR decreased in the other 3 groups. While no differences were found with regard to hemodynamic responses between group III and IV; significant differences were found between group I and all the groups (p=0.000) (p=0.001), groups II and III (p=0.043) and groups II and IV (p=0.002). Few authors have studied the effects of single bolus dose of remifentanil co-administration on LMA insertion. Lee et al[2] showed that adding low-dose remifentanil to propofol 2.5 mg kg^{-1} at induction of anesthesia provided excellent conditions for insertion of LMA. In our study, full attenuation of laryngeal reflexes at the first attempt was found in 36% and 56% of patients in propofol alone and remifentanil 0.5 μg kg^{-1} groups (**Table 1**). In contrast to our study, they have found excellent insertion conditions in 32.5% and 85% of patients in similar propofol and remifentanil groups. Our success rate in the propofol alone group as compared with the study of Lee et al,[2] was probably due to the insertion of LMA 90 seconds after propofol injection to attain peak plasma drug concentration. The decreased success rate in our 0.5 μg kg^{-1} remifentanil group was most likely due to the timing and sequence of drug administration. In another study, Grewal and Samsoon[3] showed that administering 0.3 μg kg^{-1} remifentanil with target-controlled propofol infusion provided satisfactory

Table 1 - Quality of laryngeal mask airway (LMA) insertion, intraoperative and postoperative results.

Parameters	Group I (n = 25)	Group II (n = 25)	Group III (n = 25)	Group IV (n = 25)	*P*-value				
					Groups I-II	Groups I-III	Groups I-IV	Groups II-III	Groups II-IV
Fully relaxed jaw at the first attempt	7 (28)	13 (52)	18 (72)	21 (84)		0.006	0.000		0.033
Adverse responses at the first attempt									
Coughing	5 (20)	0 (0)	0 (0)	0 (0)					
Gagging	14 (56)	9 (36)	3 (12)	1 (4)		0.002	0.000		0.01
Laryngospasm	11 (44)	4 (16)	1 (4)	0 (0)		0.002	0.000		
Head and limb movement	18 (72)	9 (36)	2 (8)	1 (4)	0.022	0.000	0.000	0.03	0.01
Full attenuation of laryngeal reflexes at the first attempt	9 (36)	14 (56)	22 (88)	24 (96)		0.000	0.000	0.025	0.002
Time to successful LMA insertion (min) mean ± SD	2.84 ± 2.07	2.08 ± 1.46	1.32 ± 0.85	1.16 ± 0.37		0.0001	0.000		
Number of attempts									
1	16 (64)	19 (76)	24 (96)	24 (96)		0.01	0.01		
2	7 (28)	5 (20)	0 (0)	1 (4)					
3	2 (8)	1 (4)	1 (4)	0 (0)					
Duration of apnea (min) mean ± SD	2.52 ± 1.29	3.64 ± 1.25	4.44 ± 1.5	4.56 ± 1.32	0.021	0.000	0.000		
Presence of blood on LMA	6 (24)	2 (8)	0 (0)	0 (0)		0.022	0.022		

All data are expressed as number (%)

LMA insertion conditions compared with placebo. However, we were unable to find any published report on the use of both 1 and 1.25 μg kg^{-1} remifentanil during the propofol induction of anesthesia to facilitate LMA insertion. Although in our study the LMA insertion conditions were generally more favorable in the remifentanil 1.25 μg kg^{-1} group than 1 μg kg^{-1} group, none reached statistical significance. Blood pressure and heart rate are usually reported to increase by 0-20% following LMA insertion.[4] Arterial pressure decreases were observed in previous studies in which propofol and remifentanil co-administration were used to provide adequate conditions for LMA insertion.[2,3] In our study, we also observed an average decrease in MAP as 29%, 38% and 38% in Group II, III and IV. The decrease, however, was transient, and only 2 patients were treated for hypotension in 1 μg kg^{-1} and 1.25 μg kg^{-1} remifentanil groups. All doses of remifentanil prevented the increase in HR related to the insertion of the LMA that was present in the propofol alone group. Bradycardia requiring escape medication occurred in one patient in group II, and in 2 patients in groups III and IV. Varying times of apnea duration after various doses of remifentanil have been reported in different studies. This may be attributed to different infusion times and different patient characteristics; the response to opioids in the public can vary immensely. Although, the use of 1 or 1.25 μg kg^{-1} remifentanil has prolonged the duration of apnea following induction, we did not find this delay to be a major clinical inconvenience. The presence of blood on LMA, which can be considered as an indication of pharyngeal mucosal trauma, was found in 24% and 8% of the laryngeal masks in Group I and II. However, insertion of the laryngeal mask with both 1 and 1.25 μg kg^{-1} remifentanil has reduced mucosal bleeding from 24% to 0%. Sore throat is one of the most common sources of morbidity after day-case surgery, and its incidence after LMA insertion ranges from 4-19%.[1] The mechanisms of sore throat after LMA insertion are probably similar to those after tracheal intubation, including trauma at insertion, cuff pressure, lubricant, temperature and humidity of anesthetic gases.[5] However, our sore throat rates were lower in remifentanil groups than the propofol alone group, although these did not reach statistical significance.

In conclusion, we found that remifentanil, 1 and 1.25 μg kg^{-1} are equally effective in facilitating insertion of the LMA following anesthetic induction with propofol, and produces similar adverse effects in terms of the level of mucosal airway damage with repeated insertion, hemodynamic and respiratory changes.

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Can incision barrier decrease the risk of surgical site infection after appendectomy?

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Appendicitis is one of the most frequent reasons for surgical intervention of acute abdominal pain. Despite low mortality, frequent surgical site infection (SSI) rates revealed, as 5-33% in the literature are still bothersome. Surgical site infection makes patient care more expensive by prolonging antibiotic usage and hospital stay. Protection of surgical wounds from contamination is one of the most recommended methods described in the literature,[1-4] in order to reduce SSI. Our aim in this study was to evaluate the effect of surgical site protection from contamination on SSI by using a hand-made incision barrier.

A total of 122 patients diagnosed with acute appendicitis and operated on between March 2001 and March 2003 were enrolled in the study. Patients were divided into 2 groups. Patients who were given an incision barrier were recruited in Group-1 and those who were not were put into Group-2.

Figure 1 - Hand-made incision barrier.

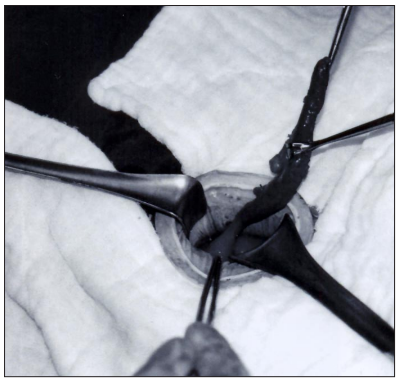

Figure 2 - Application of the hand-made incision barrier.

Randomization was achieved by enrolling every other patient assigning each group on an alternating basis. All operations were performed by the same surgeon in order to achieve standardization. Incision barrier was created by modifying Alexis Wound Retractor™ (Applied Medical Rancho Santa Margarita California USA). It was formed by 2 flexible multifilament one mm-thick steel wire rings (bicycle brake wire) and cylindrical wrist part of the latex surgical glove that was 6 cm long. Two different -sized apparatus (6 and 8 cm) were created for the study in order to use different size incisions. The steel wire rings were placed at both edges of the wrist part of the latex surgical glove. Seven millimeter space was left between the end of the wrist part of the glove and steel wire ring on both sides in order to bond the free edge over itself around the ring with a silicon based elastic glue. The length of the apparatus was approximately 4.6 cm. [Total length (4.6 cm) = Total wrist length (6 cm) - 2 x bonding space (0.7 cm)]. The apparatus was sterilized with ethylene oxide or sterilizing liquids (**Figure 1**). All patients were received one gram of intravenous cefazolin sodium 5 minutes before the skin incision. Operations were performed using the standard appendectomy technique described before.[1,2] The barrier was applied just after the peritoneum was opened and then abdominal exploration was performed. Application of the 8 cm diameter incision barriers is shown in **Figure 1**. We used flexible specification of the incision barrier on various sizes of incision (mean 5 cm, range 3-8 cm). After the appendectomy the incision barrier was pulled out of the abdomen and the surgical team changed their gloves. The contaminated surgical instruments were replaced by sterile ones and the abdomen was closed. The same procedure was performed on Group 2 except for the incision barrier application. Patients with perforated appendicitis, abdominal spoilage of intestinal material and intensive pus in the abdominal cavity received placement of a rubber drain, which is pulled out on postoperative second or third day when cessation occurred. Surgical side infections were defined as existence of one or more findings of hyperemia, swelling, pain or pus drainage from the surgical wound, increased white blood cell and significantly increased body temperature was observed. When a SSI was noted, an adequate amount of cutaneous and subcutaneous sutures was removed, abscesses were drained and the subcutaneous area was irrigated with sterile 0.9% NaCl. An adequate amount of samples of infected materials were collected for culturation just after the drainage. Subcutaneous irrigation was applied for 3 consecutive days with sterile 0.9% NaCl solution. When the wound was judged as completely healed, secondary suturation was performed. Pathologic classification of appendix was established according to the classification system introduced by Shubing and Litian.[3] Student t test and Fischer exact test was used for statistical analysis. Because our hospital serves military people, all patients in this study were male. The mean age in Group-1 was 24.9 ± 5.3 (range 21-44) and 24.9 ± 5.5 in Group-2 (range 20-41). In Group-1, 8 patients (12.50%) had acute simple, 20 (31.25%) had suppurative, 24 (37.50%) had gangrenous, 8 (12.50%) had perforated and 4 (6.25%) had normal appendicitis. In Group-2, 10 (17.25%) patients had acute simple, 21 (36.22%) had suppurative, 18 (31%) had gangrenous, 6 (10.35%) had perforated and 3 (5.2%) had normal appendicitis. In Group-1 there were not infectious complications on the surgical wound. All patients were discharged on the third day and the remaining except for 7 patients with perforated appendicitis who were discharged on the 6th postoperative day. Patients were revisited on seventh postoperative day and sutures were removed at that time. In one case with perforation, intestinal obstruction had developed. Despite medical treatment, no improvement was achieved. The patient was re-operated and an adhesion between distal ileum to abdominal wall was released. Postoperative

course was uneventful and he was discharged and completely relieved from all his symptoms on the15th postoperative day. In Group-2, 4 patients (6.9%) (3 patients with perforated and one patient with gangrenous appendicitis) developed SSI. All infections were treated and had complete wound healing and patients were discharged from the hospital with total recovery. Their hospital stay was prolonged approximately 3 days (range 2-5 days) compared with others. Microbiological study of all cultured materials revealed that *Escherichia coli* was the unique microbiological agent that was responsible for those 4 SSI. Statistical analysis also revealed that if compared with Group-1, surgical site infection rates were significantly higher in Group-2 (p=0.048). Surgical site infection is one of the most common postoperative complications of the appendectomy. The rate of SSI after appendectomy has been concluded to be between 5% and 33% in the literature.[1-4]. It was stressed in the literature that the most common factor which causes the post-appendectomy SSI is the direct contamination of the incision with the infected materials.[1,4,5] Tissue edema allows the intra-appendicular flora spread out to the periappendicular area without perforation. This insidious invasion of infected materials, even when there is any suppurative material at the operation field, can lead to SSI. As a rule, the more infection and inflammation in the appendix, the more that contamination might occur. Furthermore, in case of gangrenous and perforated appendicitis, the intraluminal flora and the infectious agents may contaminate the abdomen and incision site via direct contamination during the operation. In the literature, it is pointed out gram (-) aerobic and anaerobic bacteria are mainly responsible for post-appendectomy SSI.[1-5] Our findings supported this conclusion. In order to prevent SSI, many authors suggested different methods: delayed saturation of surgical wound, coverage of the wound with sterile gauze during the operation, wound irrigation with topical antibiotics and antiseptics (one of the most effective methods). Additionally, prophylactic systemic antibiotic usage is one of the most frequently used methods to prevent SSI. In this study, we observed the effect of surgical site prevention on SSI. For this purpose an incision barrier modified from a commercial one has been applied to wound edges in order to prevent contamination of infected materials (**Figure 2**). Commercial apparatuses can be found in medical markets but at a relatively high price if compared with our device. If we could considered its cost, the usage in 5 patients by re-sterilization could actually become negligible. We believe that this becomes important when supply of the original commercial products is limited for reasons such as out of stock of the material in the hospital stocks when operations are performed or especially in times of economic shortness. Our results in this study showed that patients to whom this hand-made device was applied had lower SSI when compared with those patients in whom the device was not used. This also indicates low morbidity rates. It is clear that not only its low price but also minimization of hospital stay and antibiotic usage and low morbidity rates makes this apparatus more economic and reliable to use. The elastic structure of the steel wires and the latex gloves allowed us to apply our apparatus even for smaller incisions. Furthermore, it can also can be applied to all patients with different size of abdominal walls (thick or thin) and can be used for all sorts of incisions by creating wire rings in various diameters and using various sizes of latex gloves.

In conclusion, prevention of surgical site from contamination by using incision barriers offers less morbidity for appendectomy and hand-made incision barriers are practical to use for this purpose, cheap and decreases operation costs by lowering morbidity. Simple and cheap hand-made incision barriers are as effective as commercial ones in case of shortness of supply. Further studies evaluating the usage and benefits of this device in different incision types of the abdomen will be beneficial.

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