Cardiopulmonary function in horses during anesthetic recovery in a hydropool

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Objective—To determine the cardiovascular and respiratory effects of water immersion in horses recovering from general anesthesia.

Animals—6 healthy adult horses.

Procedure—Horses were anesthetized 3 times with halothane and recovered from anesthesia while positioned in lateral or sternal recumbency in a padded recovery stall or while immersed in a hydropool. Cardiovascular and pulmonary functions were monitored before and during anesthesia and during recovery until horses were standing. Measurements and calculated variables included carotid and pulmonary arterial blood pressures (ABP and PAP, respectively), cardiac output, heart and respiratory rates, arterial and mixed venous blood gases, minute ventilation, end expiratory transpulmonary pressure (P_{endXes}), maximal change in transpulmonary pressure ($\Delta P_{tp}max$), total pulmonary resistance (R_L), dynamic compliance (C_{dyn}), and work of breathing ($\dot{W}$).

Results—Immersion in water during recovery from general anesthesia resulted in values of ABP, PAP, P_{endXes}, $\Delta P_{tp}max$, R_L, and $\dot{W}$ that were significantly greater and values of C_{dyn} that were significantly less, compared with values obtained during recovery in a padded stall. Mode of recovery had no significant effect on any other measured or calculated variable.

Conclusions and Clinical Relevance—Differences in pulmonary and cardiovascular function between horses during recovery from anesthesia while immersed in water and in a padded recovery stall were attributed to the increased effort needed to overcome the extrathoracic hydrostatic effects of immersion. The combined effect of increased extrathoracic pressure and PAP may contribute to an increased incidence of pulmonary edema in horses during anesthetic recovery in a hydropool. (*Am J Vet Res* 2001;62:1903–1910)

Regardless of the advancements used in equine surgical suites, few veterinary surgeons consider a procedure successful until the patient is standing in its stall following a smooth recovery. Recovery accidents may result in injuries ranging from mild abrasions and contusions to catastrophic long bone fractures or failure of orthopedic implants. Equine practitioners have developed several approaches to minimize self-trauma to horses by introducing a level of human control to the recovery process. To our knowledge, hydropool recovery (ie, immersion of anesthetized horses in water) was first used at the University of Pennsylvania's New Bolton Center in the early 1970s.[1,2] The principle benefit of hydropool recovery is that self-trauma is minimized, because the partially submerged horse struggles against water resistance until it is fully capable of standing and walking, at which point it is removed from the hydropool.

Despite this benefit, hydropool recovery has its own unique challenges. Most obvious are the risks of aspiration of water and drowning. All hydropool recovery systems have in place some form of head restraint to minimize the possibility of a horse lowering its nostrils below the water's surface. Hydropool recovery also requires more human intervention, compared with either self-recovery or rope-assisted recovery of horses in a padded stall. Some surgeons believe the decreased risk of orthopedic insult associated with hydropool recovery is worth the extra costs associated with additional personnel.[1,2]

Immersion of any animal into water results in increased extrathoracic pressure, a reflection of the displaced water and resultant hydrostatic pressure. The effect of increased hydrostatic pressure on the cardiovascular and pulmonary systems in horses is not well understood. Although there is considerable information on **head-out immersion (HOI)** in humans, there are few studies describing the effects of HOI in horses. In 1981, Smith[3] described the use of a flotation tank for long-term care of horses with orthopedic abnormalities. In that study, horses were continuously immersed or floated with the aid of a sling for 2 to 8 weeks in normal saline (0.9% NaCl) solution maintained between 35 and 36 C. Complications published with use of this sling included muscular atrophy and stiffening of the ligamentum nuchae.[3] The author noted that some horses did not survive flotation because they developed pneumonia. Moreover, he suggested that immersion above the withers may induce pathologic changes in the lungs. Hutchins et al[4] also described the use of flotation tanks for treatment of horses with orthopedic abnormalities. These authors concluded that respiratory tract complications were the most serious adverse effect of immersion, with clinical signs being apparent within 48 hours of immersion.[4] In a follow-up study, McClintock et al[5] demonstrated clinical, radiographic, and histologic pulmonary changes in horses immersed in thermoneutral (36 C) saline solution for 1 to 7 weeks. Five of 6 horses in that study developed pathologic changes that were most prominent in the dependent lung fields. The authors concluded that the

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etiopathogenesis of this immersion-induced pulmonary disease was likely multifactorial and proposed that elevation of the head, accumulation of mucopurulent exudate, and increased extrathoracic hydrostatic pressure resulted in changes in lung volumes and mechanics that led to airway obstruction and collapse, emphysema, and bronchopneumonia. McClintock et al[6] also investigated the optimal temperature of the flotation tank for long-term care of orthopedic patients and determined that water maintained at 36 C provided maximum patient comfort and resulted in the lowest incidence of respiratory tract complications.

Complications that develop following immersion for 1 to 7 weeks may not be directly extrapolated to effects that develop during 1 to 2 hours of hydropool recovery from general anesthesia. However, the hydrostatic forces that may contribute to development of bronchopneumonia are present from the moment a horse is immersed to the neck in water. At Washington State University, 10 of the first 60 horses that were recovered in a hydropool developed signs of pulmonary edema, suggesting a causal relationship between immersion and pulmonary edema and providing the incentive to better understand the effects of immersion in horses.[a] The purpose of the study reported here was to determine the cardiovascular and respiratory effects induced by thermoneutral HOI in horses recovering from general anesthesia. With improved understanding of the physiologic effects of hydropool recovery, anesthesia and recovery techniques can be modified to minimize adverse effects.

Materials and Methods

Horses—Six (5 castrated males, 1 sexually intact female) healthy horses between 4 and 10 years old with a mean (± SEM) body weight of 513 ± 18 kg were used in this study. Prior to inclusion, physical examinations were performed to rule out preexisting disease that could affect the outcome of the study. In addition, at least 4 weeks prior to inclusion in the study, the left common carotid artery was surgically relocated to a subcutaneous position to facilitate collection of arterial blood samples, and at least 5 days prior to the first anesthetic episode, blood was collected for determination of individual Evans blue dye dilution standard curves for subsequent determination of **plasma volume (PV)**.

Experimental protocol—This study was approved by the Institutional Animal Care and Use Committee at Washington State University. Each horse was anesthetized 3 times with at least 14 days between recovery episodes (lateral and sternal recumbencies and hydropool). Horses were randomly assigned to 3 groups of 2 horses each, using a randomized block design. The order in which each group underwent the 3 anesthesia recovery protocols (ie, lateral or sternal recumbency in a padded recovery stall or immersion in a hydropool) was determined by use of a 3 × 3 Latin square design. Food but not water was withheld from each horse for approximately 12 hours prior to each anesthetic episode. In addition, xylazine hydrochloride (75 mg, IV) was administered during each recovery protocol 15 minutes after horses were disconnected from the anesthetic machine.

On the morning of each anesthetic episode, the right jugular vein was cannulated, using two 8-F introducer cannulas[b] placed approximately 15 cm apart. A 7-F Swan-Ganz catheter[c] with a thermistor tip was introduced into the pulmonary artery via the distal sheath until the thermistor was approximately 5 cm distal to the pulmonic valve. To allow injection of the thermal indicator, a 14-g 170-cm fluid-filled catheter[d] was passed into the right atrium via the proximal cannula. Correct placement of all catheters was confirmed by observing the appropriate pressure waveforms on an oscilloscope. A 14-g 14-cm catheter[e] was placed in the left jugular vein for induction and maintenance of anesthesia. An 18-g 5.1-cm catheter[f] was placed percutaneously in the relocated segment of the left carotid artery for measurement of systemic **arterial blood pressures (ABP)** and collection of arterial blood samples for blood gas analysis. A polypropylene balloon catheter was passed into the midthoracic region of the esophagus via the ventral nasal meatus for measurement of transpulmonary pressure. The esophageal catheter was attached to a differential pressure transducer that had been calibrated to atmospheric pressure, using a water manometer.[g] Pulmonary function data were collected, using a 5.08-cm heated pneumotachograph[h] that was fixed to a customized facemask (preanesthesia and standing) or the oral end of the endotracheal tube (anesthesia and first 15 to 30 minutes of recovery). Prior to instrumentation, all pressure and flow signals were in phase at frequencies up to 5 Hz.

Horses remained undisturbed for 1 to 3 hours following instrumentation prior to collection of baseline (preanesthesia) data. Horses were weighed immediately prior to the first measurement, using an electronic scale. Baseline measurements were obtained 30 minutes prior to induction of anesthesia. Beginning at the time designated as the onset of anesthesia (A0), 5 data sets were collected at 15-minute intervals (A0, A15, A30, A45, and A60) while horses were under anesthesia. Data were also collected at least twice during recovery at 15-minute intervals (R0, R15, R30). The number of data sets collected during recovery was dependent on the rate of recovery. If a horse changed body position (eg, from sternal to lateral recumbency) during the stall recovery protocols, no further samples were collected until that horse was standing. Similarly, if a horse recovering in the pool struggled sufficiently to place the animal, personnel, or equipment in danger, further sampling was discontinued until that horse was standing. Horses were also extubated at this time to decrease the risk of aspiration of pool water. Extubation precluded further measurements of breathing mechanics. **Recovery-time zero (R0)** was defined as the time at which each horse began the recovery protocol (ie, was immersed in the hydropool or positioned in lateral or sternal recumbency). For the stall recovery protocols, a final data set (standing) was collected as soon as each horse was standing and could be safely approached. For the hydropool recovery protocol, this data set was collected as soon as horses could walk from the pool and be led to an adjacent recovery stall. Each data set consisted of heart rate, respiratory rate, mucous membrane color, capillary refill time, PCV, total protein concentration, **pulmonary arterial pressure (PAP)**, systemic ABP, arterial and mixed venous gases, blood temperature, inspired and expired oxygen concentrations, end tidal carbon dioxide and halothane concentrations, **cardiac output (CO)**, plasma volume sampling, and pulmonary function testing. For each data set, samples were obtained and measurements made in the following order: blood pressure measurements, CO, blood gas sampling, and pulmonary function testing. All other sampling and measurements were performed simultaneously rather than sequentially.

For each data set, mixed venous blood samples were collected from the distal port of the Swan-Ganz catheter, and arterial samples were collected from the carotid artery catheter. Blood was collected for determination of PV, Hct, total plasma protein concentration, and blood gases. Hematocrit and total plasma protein concentration were measured in triplicate by centrifugation of heparinized

microhematocrit tubes[i] in a microcentrifuge[j] for 5 minutes at 11,700 rpm.

Anesthesia protocol—Horses were sedated with xylazine (1 mg/kg of body weight, IV) and 5 minutes later, anesthesia was induced with ketamine hydrochloride (2.2 mg/kg, IV) and diazepam (0.088 mg/kg, IV). After oral intubation with a 26-mm (inside diameter) endotracheal tube, anesthesia was maintained with halothane[k] in oxygen (6 L/min) administered via a semiclosed large-animal breathing circuit.[l] Horses were positioned in left lateral recumbency and mechanically ventilated throughout anesthesia. Lactated Ringer's solution (10 ml/kg/h, IV) was administered to all horses, and dobutamine (0.25 mg/min, IV) was administered as needed to maintain mean systemic ABP at > 70 mm Hg. End-tidal halothane concentration was maintained at 1.5% during the 60-minute anesthesia period, and the point at which each horse first achieved an end-tidal halothane concentration of 1.5% was referred to as time A0. One hour after time A0 (ie, A60), horses were disconnected from the anesthetic machine and moved to the assigned recovery location (hydropool or padded stall).

Hydropool recovery protocol—The recovery pool measured 3.66-m long × 1.22-m wide × 2.6-m deep and had a hydraulic floor that could be raised or lowered. Horses were moved to the pool and maintained in an upright position by use of a sling attached to an overhead mechanical hoist on a rail. Because of water buoyancy, the weight of each horse was partially supported, and most horses floated with the withers 4 to 6 cm under the water's surface. Two handlers helped control each horse through head ropes attached to the halter. Water temperature was maintained between 37.7 and 38.6 C. When each horse was considered sufficiently coordinated and aware of its surroundings, the sling was removed, and the hydraulic floor was raised, which lifted the horse out of the pool. In general, each horse met the following criteria before the floor was raised: return of a strong menace response, lack of nystagmus, ability to stand well on all limbs, ability to maintain head position out of the water without assistance, and voluntary tongue movement.

Stall recovery protocol—Horses were moved to a padded recovery stall (3.6 m × 3.6 m) by means of a mechanical hoist and pastern hobbles. Once in the stall, horses were positioned in lateral or sternal recumbency. Sternal recumbency was maintained with human restraint and pads. Head and tail ropes were used to assist recovery.

Measurements and calculations—Pressure and airflow signals were recorded graphically on a physiograph,[m] and analog signals from the transducers were digitized and stored on disk for subsequent analysis. Minute ventilation ($\dot{V}_E$), tidal volume (V_T), dynamic compliance (C_{dyn}), pulmonary resistance (R_L), work of breathing ($\dot{W}$), end expiratory transpulmonary pressure (P_{endXes}), respiratory exchange ratio ($R;\dot{V}_{CO_2}/\dot{V}_{O_2}$), and maximal change in transpulmonary pressure ($\Delta P_{tp}max$) were calculated, using a customized software program for physiologic assessment of horses. End expiratory transpulmonary pressure was taken to be the transpulmonary pressure at the end of expiration when airflow equaled zero. Inspired and expired percentages of oxygen and carbon dioxide were determined and recorded, and the alveolar arterial oxygen gradient was calculated, using the ideal gas equation.

Temperature-corrected blood gas and pH data were determined, using a blood gas analyzer.[n] Resting heart and respiratory rates, pulmonary arterial blood temperature, and oral mucous membrane color and capillary refill time were recorded. Cardiac output was determined by **thermodilution (TD)**, using ice-cold (0 C) 5% dextrose solution as the thermal indicator. Briefly, using a high-speed injector, 40 ml of ice-cold dextrose was injected rapidly through the delivery catheter into the right atrium. The resultant TD curves were digitized[o] and analyzed, using the modified Stuart-Hamilton equation to calculate CO:

$$CO\ (L/min) = cc(T_b - T_i)/1.22(\Sigma DT_b)$$

where cc is the computational constant determined for the delivery catheter, T_b is the temperature of blood, T_i is the temperature of the dextrose solution, and ΣDT_b is the area under the TD time versus temperature curve. At least 3 measurements of CO were made at each sampling time, and the mean of these 3 readings was used as the CO data point for that data set. Stroke volume was calculated as the quotient of CO and heart rate. **Cardiac index (CI)** was calculated, using the following formula:

$$CI\ (ml/kg/min) = CO/body\ weight\ (kg)$$

End-tidal halothane concentration and carbon dioxide and oxygen tensions were measured, using an anesthetic gas analyzer.[p] A 45-cm 5-F polypropylene catheter was maintained within the lumen of the endotracheal tube to allow constant real-time sampling by the gas analyzer.

Approximately 15 minutes prior to anesthesia, 14 ml of 1% Evans blue dye (weighed to 4 decimals for accuracy) was injected through the distal cannula into the right jugular vein. This cannula was flushed with heparinized saline solution and was not used to collect blood for the remainder of the experiment. Blood samples for determination of PV were collected from the Swan-Ganz catheter and transferred to tubes containing lithium heparin.[q] Plasma was separated by centrifugation, transferred to polyethylene tubes, and refrigerated overnight. The concentration (absorbance) of dye in each plasma sample was measured on a spectrophotometer[r] at a wavelength of 620 nm, and PV was calculated, using individual standard curves as described.[7] **Blood volume (BV)** was calculated as follows:

$$BV = PV/1 - Hct$$

Diastolic, systolic, and mean carotid ABP and PAP were measured, using calibrated pressure transducers connected to an oscilloscope.[s] In standing and sternally recumbent horses, the transducer zero reference was the point of the shoulder joint, whereas in laterally recumbent horses, the zero reference was the sternal midline. To facilitate pressure measurements while horses were immersed in the hydropool, the pressure transducer[t] was placed on the decking adjacent to the pool at a vertical distance of 76 to 96 cm from the shoulder joint. After immersion, distances were measured from the shoulder joint to the water surface and from the water surface to the transducer level. The resulting pressure measurements were corrected for the vertical distance from the shoulder joint to the transducer according to methods described for adjustments in pressure measurements for dogs undergoing HOI.[8] For standing horses, exact measurements linking the relationship between vertical distance of the transducer from the shoulder joint and PAP and ABP were determined prior to each anesthetic episode at the time of instrumentation. Data from these measurements were then applied to measurements made during hydropool recoveries to correct pressure values.

Statistical analyses—Data were analyzed by use of 2-way ANOVA with repeated measures and posthoc Bonferroni tests where applicable. Data collected during anesthesia (A15, A30, A45, A60) were analyzed separately to rule out effects of repeated anesthetic episodes. Data sets collected at A60 and during recovery were analyzed as a separate subset, because the hypothesis being tested related only

to the recovery period. All analyses were performed, using a commercial software package.[u] Significance was set at $P < 0.05$, and data were expressed as the mean ± SEM.

Results

Quality of anesthesia—Values recorded during general anesthesia were not different among treatment groups. Values determined from A15 to A60 also did not differ significantly during any anesthetic episode, indicating that all horses reached a steady state before being moved for recovery.

Quality of recovery—Subjective evaluation of the quality of recovery from anesthesia was not different among treatment groups. Mean time from disconnection from the ventilator until standing differed between groups (lateral recumbency, 66 ± 4.5 minutes; sternal recumbency, 56 ± 2.2 minutes; hydropool recovery, 83 ± 11.0 minutes); however, this difference was not significant.

Results at R15 and R30 were calculated from fewer raw data points than at R0 because of animal movement. If horses changed body position (eg, from sternal to lateral recumbency) or struggled, further sampling was discontinued until horses were standing. For results at R15, data were available from 4 of 6 horses in the hydropool and lateral recumbency groups and from 3 of 6 horses in the sternal recumbency group. Results at R30 were calculated from data for ≤ 3 horses. Therefore, R30 results were left out of statistical analyses.

Respiratory effects of recovery protocol—Work of breathing was significantly higher during hydropool recovery, compared with recovery in sternal or lateral recumbency (**Table 1**). During hydropool recovery, W was approximately 3-fold greater than the value determined at A60. However, during recovery in the padded stall, W did not change significantly from values determined during anesthesia. Standing (ie, the final data set) values forW were not significantly different among treatment groups, although values for each horse were increased, compared with baseline values.

Pulmonary resistance was significantly higher in the hydropool group, compared with either the sternal or lateral recumbency groups (Table 1). In immersed horses, R_L (0.93 ± 0.13 cm H_2O/L/s) was approximately twice that of values in either laterally or sternally recumbent horses (0.45 ± 0.01 and 0.44 ± 0.05 cm H_2O/L/s, respectively). Pulmonary resistance during recovery was significantly increased, compared with the A60 value, only for those horses immersed in the hydropool. Following recovery (ie, standing values), R_L was significantly greater in hydropool-recovered horses, compared with horses recovered in either position in the padded stall. Standing values were obtained using a facemask rather than the endotracheal tube, so comparison of standing values with A60 and recovery values was not warranted.

Mean P_{endXes} was significantly higher during hydropool recovery than during recovery in either position in the padded stall (Table 1). Individual values ranged from 5 to 20 cm H_2O in the hydropool and –4 to 4 cm H_2O in the padded stall, regardless of position. For horses recovered in the hydropool, P_{endXes} at R0 was approximately 10-fold greater than at A60. However, P_{endXes} at R0 for horses recovered in the padded stall did not differ significantly from values

Table 1—Respiratory effects of 3 anesthetic recovery protocols in 6 healthy adult horses

Group*	$\dot{W}$ (L/s/cm H_2O)	R_L (cm H_2O/L/s)	P_{endXes} (cm H_2O)	$\Delta P_{tp}max$ (cm H_2O)	C_{dyn} (L/cm H_2O)	$\dot{V}_E$ (L/min)	V_T (L)	Pao_2 (mm Hg)	$Paco_2$ (mm Hg)
Baseline									
Hydropool	10.1 ± 2.3	0.29 ± 0.06	–0.9 ± –0.6	3.7 ± 0.4	2.04 ± 0.28	95 ± 21	5.1 ± 0.6	76 ± 2.9	47.5 ± 3.9
Lateral	14.5 ± 2.9	0.30 ± 0.05	–3.3 ± –1.3	5.2 ± 0.7	1.53 ± 0.16	96 ± 10	5.9 ± 0.8	81 ± 6.6	44.8 ± 1.0
Sternal	11.2 ± 2.2	0.40 ± 0.07	0.6 ± 1.7	5.6 ± 0.6	1.62 ± 0.23	73 ± 9	4.8 ± 0.4	81 ± 3.2	45.3 ± 0.6
A60									
Hydropool	30.3 ± 2.4[a]	0.36 ± 0.01[a]	1.2 ± 0.4[a]	7.9 ± 0.17[a]	0.52 ± 0.09	55 ± 4[a]	7.5 ± 0.6	391 ± 27.6[a]	50.3 ± 2.9
Lateral	26.3 ± 2.0	0.37 ± 0.01	0.3 ± 0.5	8.0 ± 0.33	0.56 ± 0.07	55 ± 4	6.9 ± 0.4	378 ± 32.9[a]	55.1 ± 3.3
Sternal	19.2 ± 3.4	0.28 ± 0.05	0.5 ± 0.5	8.2 ± 0.62	0.85 ± 0.18	54 ± 4	7.3 ± 0.4	385 ± 26.7[a]	58.8 ± 2.9
R0									
Hydropool	104.4 ± 40[b,c]	0.93 ± 0.13[b,c]	10.1 ± 2.3[b,c]	25.5 ± 4.5[b,c]	0.38 ± 0.10[b,c]	100 ± 21	5.8 ± 1.1	59.8 ± 3.2	51.2 ± 2.0
Lateral	26.3 ± 4.8	0.45 ± 0.01	1.6 ± 0.7	8.9 ± 1.1	0.83 ± 0.12	67 ± 15	4.8 ± 0.4	61.9 ± 3.7	54.8 ± 3.7
Sternal	32.2 ± 13	0.44 ± 0.05	–2.0 ± 0.8	9.9 ± 1.6	1.09 ± 0.08	65 ± 12	6.9 ± 0.9	67.9 ± 3.1	55.7 ± 1.4
R15									
Hydropool	98.1 ± 26.1[b]	1.19 ± 0.29[b,c]	10.9 ± 2.9[b,c]	23.9 ± 5.1[b,c]	0.45 ± 0.09	106 ± 40	4.3 ± 0.8	43.9 ± 4.7	49.9 ± 2.2
Lateral	20.4 ± 3.9	0.25 ± 0.04	0.3 ± 0.8	7.3 ± 1.0	0.87 ± 0.20	75 ± 2	5.8 ± 0.4	54.9 ± 3.5	47.6 ± 2.2
Sternal	34.6 ± 11.6	0.24 ± 0.03	–4.9 ± 1.5	7.7 ± 1.2	1.19 ± 0.14	90 ± 10	5.9 ± 1.1	71.1 ± 8.8	50.5 ± 3.5
Standing									
Hydropool	49.8 ± 15.7	2.25 ± 0.44[b,c]	–1.1 ± 0.6	16.7 ± 3.5	1.20 ± 0.15	38 ± 8	5.3 ± 0.6	70.6 ± 6.1	45.3 ± 2.2
Lateral	37.5 ± 4.7	1.61 ± 0.07[c]	–2.4 ± 0.9	15.0 ± 1.0	1.47 ± 0.06	37± 4	7.9 ± 0.2	66.6 ± 4.4	50.1 ± 1.5
Sternal	60.1 ± 18.8	0.89 ± 0.30[b]	–1.6 ± 0.8	12.5 ± 1.8	1.40 ± 0.10	99 ± 35	8.5 ± 1.8	75.7 ± 2.1	50.8 ± 1.5

Data are reported as mean ± SEM.

*Baseline measurements were obtained 30 minutes prior to induction of anesthesia, and A60 measurements were obtained 1 hour after end-tidal halothane concentration first reached 1.5%. Immediately after collection of A60 measurements, horses were disconnected from the anesthetic machine and immersed with slings in a hydropool to the level of the withers or positioned in lateral or sternal recumbency in a padded stall. The R0 data set was obtained immediately after positioning horses, and the R15 data set was obtained 15 minutes later. Standing measurements were obtained as soon as each horse was standing or could walk from the pool.

W = Work of breathing. R_L = Pulmonary resistance. P_{endXes} = End expiratory transpulmonary pressure. $\Delta P_{tp}max$ = Maximal change in transpulmonary pressure. C_{dyn} = Dynamic compliance. V_E = Minute ventilation. V_T = Tidal volume.

[a]Significantly ($P < 0.05$) different from the value obtained at R0 for the same recovery protocol. [b]Significantly ($P < 0.05$) different from the value obtained at the same time for the laterally recumbent group. [c]Significantly ($P < 0.05$) different from the value obtained at the same time for the sternally recumbent group.

determined at A60. Following removal from the pool, P_{endXes} returned to the baseline value and was not significantly different from standing values determined for the other 2 groups.

Mean $\Delta P_{tp}max$ was significantly higher during hydropool recovery, compared with the other 2 recovery protocols (Table 1). Mean values at R0 and R15 in immersed horses (25.4 ± 4.5 and 23.9 ± 5.1 cm H_2O, respectively) were nearly 3 times those in horses in sternal or lateral recumbency. Moreover, R0 and R15 values in immersed horses were significantly different from $\Delta P_{tp}max$ and determined at A60; this difference was not detected in either stall-recovered group. Final (ie, standing) $\Delta P_{tp}max$ was not significantly different among treatment groups. At R0, peak inspiratory transpulmonary pressure was significantly less and peak expiratory transpulmonary pressure significantly greater in immersed horses, compared with horses in sternal or lateral recumbency. However, at R15, only peak expiratory transpulmonary pressure was significantly different among groups.

Mean C_{dyn} was significantly less in the hydropool-recovered horses, compared with the other 2 groups (Table 1). At R0, mean C_{dyn} for immersed horses (0.38 ± 0.1 L/cm H_2O) was approximately half that of laterally and sternally recumbent horses (0.83 ± 0.12 and 1.08 ± 0.08 l/cm H_2O, respectively). Values at R15, however, were not significantly different among groups. In immersed horses, C_{dyn} at R0 decreased significantly from the A60 value, whereas in sternally and laterally recumbent horses, C_{dyn} increased from A60 to R0. Standing values approached baseline values and were not significantly different among treatment groups.

The difference between alveolar and arterial oxygenation at R0 was not significantly different among groups (hydropool recovery, 33.6 ± 4.6 mm Hg; laterally recumbent, 30.47 ± 4.3 mm Hg; sternally recumbent, 20.2 ± 6.6 mm Hg). After 15 minutes of immersion, the alveolar-arterial difference increased but was still not significantly different from values for the laterally recumbent group (41.5 ± 3.8 and 36.4 ± 3.3 mm Hg, respectively). Because of patient movement and difficulty in sample collection at R15 in the sternally recumbent group, there were insufficient data points to include in analyses. Mode of recovery had no significant effect on $\dot{V}_E$, respiratory rate, arterial oxygenation, respiratory exchange ratio, V_T, or end-tidal concentrations of halothane, oxygen, or carbon dioxide.

Cardiovascular effects of recovery protocol— Mean ABP was significantly ($P < 0.001$) higher during hydropool recovery, compared with the other groups (**Table 2**). During immersion, mean ABP (144 ± 6.9 mm Hg) was approximately 36 and 16% greater, compared with lateral or sternal recumbency, respectively. Mean ABP at R0 were significantly greater in all groups, compared with A60 values. Arterial blood pressures returned to baseline values within 5 minutes of standing in all groups, and there were no significant differences among groups at that time.

Mean PAP at R0 was 83% greater in the immersed group than in the lateral recumbent group and 68% greater than in the sternal recumbent group (Table 2). Mean PAP at R15 was also significantly greater in immersed horses, compared with recumbent horses. Mean PAP measured at R0 was significantly increased, compared with the A60 value, only for those horses immersed in the hydropool. Values for PAP determined after recovery (ie, standing) were not significantly different among groups and approximated baseline values in all groups.

Mode of recovery had no significant effect on CO, CI, BV, PV, Hct, total plasma protein concentration, heart rate, or SV. Determination of CO proved to be technically challenging during all recovery protocols. Consequently, 2 horses were dropped from the final analysis for lack of usable data.

Table 2—Cardiovascular effects of 3 anesthetic recovery protocols in 6 healthy adult horses

Group*	Mean ABP (mm Hg)	Mean PAP (mm Hg)	CO (L/min)	PV (ml/kg)	BV (ml/kg)
Baseline					
Hydropool	118 ± 2.4	28.2 ± 2.5	38.7 ± 4.2	40.0 ± 3.9	61.9 ± 6.2
Lateral	135 ± 3.6	30.5 ± 2.0	42.2 ± 5.2	40.2 ± 3.3	65.8 ± 4.8
Sternal	126 ± 4.91	29.3 ± 3.5	42.4 ± 7.7	41.9 ± 2.8	68.6 ± 5.5
A60					
Hydropool	74.8 ± 1.0[a]	36.2 ± 2.3[a]	17.5 ± 2.6	55.2 ± 4.7	80.1 ± 6.9
Lateral	70.3 ± 1.8[a]	31.7 ± 1.6	19.3 ± 3.1	55.5 ± 3.9	81.3 ± 5.9
Sternal	73.0 ± 2.6[a]	32.5 ± 1.1	17.9 ± 1.7	55.2 ± 3.4	81.7 ± 6.2
R0					
Hydropool	144.8 ± 6.9[b,c]	54.6 ± 2.5[b,c]	23.9 ± 3.8	53.2 ± 5.2	81.8 ± 8.5
Lateral	106.2 ± 4.6[c]	29.8 ± 3.5[c]	23.3 ± 2.0	58.4 ± 2.2	74.2 ± 10.6
Sternal	124.0 ± 7.6	32.5 ± 2.7	18.6 ± 2.1	56.8 ± 2.0	91.3 ± 2.5
R15					
Hydropool	157.9 ± 3.6[b,c]	53.0 ± 3.8[b,c]	29.6 ± 4.5	54.2 ± 5.6	80.5 ± 8.0
Lateral	112.5 ± 3.5[c]	28.6 ± 2.1[c]	22.2 ± 1.3	52.4 ± 6.1	75.9 ± 8.7
Sternal	126.3 ± 2.3	23.8 ± 3.2	21.2 ± 3.3	50.2 ± 6.9	76.1 ± 11.2
Standing					
Hydropool	123.4 ± 6.3	23.7 ± 2.5	30.4 ± 4.1	51.6 ± 4.7	78.9 ± 7.7
Lateral	122.2 ± 9.2	27.0 ± 4.1	48.0 ± 9.2	51.8 ± 3.7	76.2 ± 5.2
Sternal	126.4 ± 4.9	24.6 ± 1.9	34.1 ± 2.1	52.2 ± 3.8	81.9 ± 6.9

Data are reported as mean ± SEM.
ABP = Carotid arterial blood pressure. PAP = Pulmonary arterial blood pressure. CO = Cardiac output. PV = Plasma volume. BV = Blood volume.
See Table 1 for key.

Discussion

The results of this study indicated that thermoneutral HOI had significant effects on cardiopulmonary function of horses recovering from general anesthesia. Changes in pulmonary function and cardiovascular dynamics, specifically an increase in R_L, mean PAP, and P_{endXes} and a decrease in C_{dyn}, all contributed to an increase in W. For the most part, these results paralleled results of studies[9-36] in humans and other species subjected to HOI. However, there were some distinct variations in results between horses and humans.

Horses submerged to the midcervical region in thermoneutral water experience added hydrostatic pressure that acts most profoundly on the thoracic cavity. Hydrostatic pressure is dependent on water depth, such that pressure increases approximately 1 **atmosphere (ATM)** for every 10 m of depth. For horses recovered in the hydropool, the cranioventral lung fields and heart were under approximately 10% (or 0.1 ATM) more pressure than horses recovered in the padded stall. This increased pressure affected several aspects of cardiopulmonary function. Extrathoracic pressure primarily acts to narrow and close small airways, which results in increased R_L. In resting nonintubated horses, 50% of airway resistance is attributable to the nasal passageways, 30% to the larynx and trachea, and the remaining 20% to the intrathoracic airways. In the present study, horses were still intubated at R0 and R15, so R_L measured at these times was attributable to resistance in airways distal to the aboral end of the endotracheal tube as well as resistance created by the endotracheal tube itself. Changes in airway resistance caused by extrathoracic hydrostatic pressure are most likely to develop at the level of the bronchioles and alveoli, because these structures lack cartilaginous support. The increase in R_L that we detected in immersed horses contributed to the increase in W during hydropool recovery.

Studies[9-12] using humans and dogs have closely examined the initial cardiovascular and pulmonary changes induced by HOI. Many of the findings of these studies can be related to 2 major physiologic effects of water immersion on mammals. First, water immersion results in an equilibration of hydrostatic pressure gradients of body fluids. Secondly, immersion results in an enhancement of transcutaneous thermal exchange.[13] The increased hydrostatic pressure that a mammal experiences during immersion is associated with shifts in blood flow from the periphery to the intrathoracic region, thereby significantly increasing pulmonary capillary BV and increasing left cardiac preload.[14-17] In humans undergoing thermoneutral water immersion, this increase in venous return is reflected by an increase in mean PAP.[18,19] We also detected an increase in PAP during hydropool recovery in our horses.

Pulmonary hypertension resulting in pulmonary capillary hyperemia can lead to a disturbance in the balance of capillary hemodynamics and lymphatic drainage in the parenchyma. Immersion increases cardiac preload, which, combined with overhydration, has been shown to result in acute pulmonary edema in human athletes during episodes of maximal exertion.[20,21] An increase in fluid in the interstitium results in wetter and heavier lungs that, in turn, decrease C_{dyn}. In the present study, C_{dyn} was significantly decreased during immersion, whereas in horses recovered in sternal or lateral recumbency, C_{dyn} increased.

Mean P_{endXes} was significantly increased during hydropool recovery. This variable measures the transpulmonary pressure at the end of expiration and is usually slightly negative (–1 to –3 cm H_2O) in resting horses. A negative P_{endXes} helps to make the first phase of inhalation passive. A positive P_{endXes} may reflect small airway closure attributable to external forces (eg, an increase in extrathoracic hydrostatic pressure). An increase in P_{endXes} also suggests a decrease in end-expiratory lung volume that further results in small airway closure. The increase in W that we detected during hydropool group, compared with stall recovery, can be partially explained by the extra effort required to overcome the increase in extrathoracic hydrostatic pressure.

In humans, vital capacity, expiratory reserve volume, and maximum pulmonary ventilation decrease with immersion, whereas closing volume increases.[18,22,23] Tidal volume and $\dot{V}_E$ decrease as a consequence of reductions in peak airflows. Therefore, greater transpulmonary pressures must be generated to produce a given V_T, which results in an increased in $\dot{W}$. Although V_T and $\dot{V}_E$ were not significantly different among groups in the present study, $\Delta P_{tp}max$ was significantly higher in immersed horses, compared with recumbent horses, and this increase also contributed to the increase in W detected during hydropool recovery.

In the present study, neither Pa_{O_2} nor Pa_{CO_2} were significantly different among groups during recovery. Results of previous studies[18,24,25] are inconsistent with respect to the effect of immersion on arterial blood gases; some studies revealed an immersion-induced decrease in Pa_{O_2} and an increase in Pa_{CO_2}, whereas others failed to reveal any significant change. The proposed mechanism for poor gas exchange induced by water immersion involves a direct response to increased extrathoracic hydrostatic pressure, as well as compression of airways that leads to an increase in R_L and functional dead space and a decrease in C_{dyn}. Together these changes have been postulated to decrease ventilation-perfusion ratios (V_A/Q) and diminish gas exchange,[18,26] although Derion et al[25] demonstrated that V_A/Q was not altered during HOI. The authors of the latter study concluded that increases in mean PAP and apical lung perfusion resulted in an increased surface area for gas exchange that offset negative influences of immersion on ventilation.

Ventilation-perfusion mismatching is an expected consequence of anesthesia in horses.[27] Dependent lung fields are underventilated because of narrowing of small airways and an increase in R_L. Any mismatching is likely to be compounded when horses are placed in a hydropool for recovery, because additional hydrostatic pressure results in a further decrease in C_{dyn}. However, V_A/Q was not significantly affected by immersion in the present study, suggesting that the change in position from lateral recumbency to upright in the hydropool offset the negative effects of hydrostatic pressure.

None of the horses in the present study developed clinical signs of pulmonary edema. However, others have reported that 10 of 60 horses recovered in a hydropool following surgery or other procedures requiring general anesthesia developed signs of pulmonary edema.[a] The lack of clinical signs (eg, fluid sounds in trachea, froth or fluid from the nostrils or mouth, respiratory distress, coughing) in the horses in the present study was likely attributable to several differences between results of our study and those reported by others.[a] Anesthesia times and fluid volumes administered in the present study were less than previously reported. In addition, horses in the present study were healthy, and we controlled body position during anesthesia and recovery and limited the use of α_2 agonists. A single dose of xylazine was administered 15 minutes into each recovery episode to closely mimic the clinical situation, in which horses typically receive variable amounts of sedatives during recovery.

Results of hemodilution studies[28,29] indicate that fluid movement across capillary walls in the limbs may be the most important mechanism resulting in a decrease in colloidal oncotic pressure in humans undergoing HOI. Plasma volume increases significantly in humans and dogs within 20 minutes of immersion and is accompanied by a decrease in Hct and total protein concentration.[16,29-32] Although PV steadily increased throughout anesthesia and recovery in horses in the present study, we did not detect a significant difference in PV among groups. In those studies that revealed an immersion-induced hemodilution in humans, PV was determined by use of precision densitometry, a technique that was not available for use in our study. It is possible that either the Evans blue dye dilution technique was not sensitive enough to reveal differences among groups or that if immersion induced significant changes in PV, those changes did not occur during the sampling period (ie, first 30 minutes of immersion).

Cardiac index and CO increase in humans experiencing thermoneutral and above-neutral water immersion.[16,19,24,26,33-35] Unlike results of those studies, however, neither CO nor CI was significantly different among groups in the present study. Cardiac output was slightly greater during hydropool recovery, compared with stall recovery, but these differences were not significant. We were concerned that CO determinations for horses in the hydropool were not accurate, because the delivery catheter was submerged over much of its length in thermoneutral water. Thus, the dextrose solution was not maintained near 0 C, and this could have altered the final time versus temperature curve and resulted in erroneous values for CO. In humans, several different methods are used to determine CO and CI, including Doppler ultrasonography, impedance cardiography, and a rebreathing technique. Although none of these methods were applicable for use in this study, their use may have yielded more consistent data.

The difference between alveolar and arterial oxygen tension during hydropool recovery was not significantly different from that during stall recovery. Time, however, had a significant effect on the difference between alveolar and arterial oxygen tension in immersed horses. This change in immersed horses is consistent with impairment of oxygen diffusion and may indicate an increase in interstitial or alveolar fluid volume. Other sources of an increase in the difference between alveolar and arterial oxygen include ventilation-perfusion mismatching and right-to-left vascular shunting.

Body position has a known effect on cardiopulmonary function in horses undergoing general anesthesia and may play a role in differences in cardiopulmonary function between horses recovered while positioned in lateral recumbency and horses recovered while immersed in water.[36] We included a third recovery protocol in the present study, in which horses recovered while positioned in sternal recumbency. This positioned the lungs in as close to a normal position as possible without the use of slings and enabled us to evaluate the effects of immersion separate from the effects of body position. Interestingly, we did not find significant differences in any variable between laterally and sternally recumbent horses. Subjective observations, however, suggested that recovery times (time from disconnection of ventilator to standing) were less in sternally recumbent horses, and sternally recumbent horses made earlier attempts to move or stand, compared with laterally recumbent horses. In addition, mean PAP was greater than in the laterally recumbent group, and P_{endXes} was consistently negative, whereas this value was consistently positive in the other 2 groups.

In humans, CO increases progressively as the temperature of water used in HOI protocols increases.[19,24] This increase in CO for a given oxygen consumption resulted in hyperperfusion of peripheral tissues during HOI in thermoneutral water.[37] However, systemic ABP and peripheral resistance decreased progressively with increasing water temperature.[19] In a recent study,[37] **systemic vascular resistance (SVR)** decreased 37% in humans undergoing thermoneutral HOI, compared with dry conditions. The authors of that study concluded that the high CO during HOI was necessary to maintain adequate arterial pressures in the face of a reduced SVR. Water temperature did not have a significant effect on fluid and cation changes in healthy humans undergoing HOI, suggesting that immersion per se is the primary stimulus for hemodilution. With this information in mind, we exposed our horses to thermoneutral water conditions (37.5 to 38.6 C).

Work of breathing, R_L, mean PAP, and P_{endXes} significantly increased and C_{dyn} significantly decreased as a result of HOI for recovery of horses from general anesthesia. However, hydropool recovery had no significant effect on CO, PV, or BV. Although we did not detect clinical signs of pulmonary edema in immersed horses, the effects of increased hydrostatic extrathoracic pressure on both the cardiovascular and pulmonary systems may contribute to an increased incidence of pulmonary edema in some horses recovering from general anesthesia in a hydropool.

[a]Tidwell SA, Veterinary Teaching Hospital, Washington State University, Pullman, Wash: Personal communication, 2000.

[b]Percutaneous introducer sheath, Arrow International Inc, Reading, Pa.

[c]Swan-Ganz catheter, 93A-131H-7F, American Edwards Laboratories, Irvine, Calif.
[d]Baxter TD injectate catheter, Baxter Customer Defined Products, Irvine, Calif.
[e]Abbocath-T, Abbott Laboratories, North Chicago, Ill.
[f]Novalon, Becton-Dickinson, Sandy, Utah.
[g]Validyne DP 45–34, Validyne Engineering, Northridge, Calif.
[h]Fleisch No. 5, Instrumentation Associates Inc, New York, NY.
[i]Hemocapillary tubes, Fisher Scientific, Chicago, Ill.
[j]Micro-MB, International Equipment Co, Needham Heights, Mass.
[k]Fluothane, Ayerst Laboratories Inc, New York, NY.
[l]Narkovet-E, Drager Medical Inc, Telford, Pa.
[m]VSM 5, Medtronic Physio-control, Redmond, Wash.
[n]995 Blood gas analyzer, AVL Scientific Corp, Roswell, Ga.
[o]PCM-DAS16S/16, Computer Boards Inc, Middleboro, Mass.
[p]Rascal II respiratory gas monitor, Datex-Ohmeda, Tewksbury, Mass.
[q]Vacutainer, Becton-Dickinson, Sandy, Utah.
[r]Ultraspec III, Pharmacia Laboratories, Peapack, NJ.
[s]Vital signs monitor 5, Physiocontrol Corp, Redmond, Wash.
[t]Transpac II transducer, Abbott Laboratories, Abbot Park, Ill.
[u]SigmaStat for Windows, version 2.03, Access Softek Inc, Berkeley, Calif.

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