Jugular venous pooling during lowering of the head affects blood pressure of the anesthetized giraffe


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How blood flow and pressure to the giraffe’s brain are regulated when the giraffe, within seconds, changes position from standing, with the brain elevated some 2 to 3 m above the heart, to the drinking position, where the brain is lowered some 2 m below the heart. Although experiments have shown that the adult giraffe is endowed with a mean arterial pressure (MAP) of twice the value of humans (14, 27, 36), the regulatory mechanisms during postural changes are not fully understood.

Despite a high MAP, it has been suggested that cerebral perfusion of the upright giraffe needs to be supported by a siphon mechanism (2), where the energy needed to overcome gravity in the carotid artery is recovered as blood returns to the heart through the jugular vein. However, a siphon mechanism in the giraffes ~2 m long neck would require that the jugular venous pressure at the cranial end is negative by >100 mmHg or a high pressure at the base of the jugular vein. The siphon theory has been addressed in numerous theoretical and mechanical models of which some have reached conflicting conclusions (4, 6, 19, 25, 27, 30, 33). Alternatively, the jugular veins may be collapsed, as in humans when the head is upright (10). In that case, a siphon mechanism cannot operate, and the waterfall analogy has been used to describe venous return (23). The in vivo jugular venous pressure of the giraffe has been reported to be positive close to the head and to approach zero toward the heart (18). This positive pressure gradient negates a functional siphon, but is also not consistent with the common understanding of the waterfall hypothesis (30).

The largest gravitational challenge to the giraffe’s circulatory system is when it lowers the head to drink and raises it again. However interesting this question is, there is only one report of distal carotid arterial pressure from one fully recovered giraffe that voluntarily lowered its head to drink (35). This report shows one brief increase to almost 350 mmHg in distal carotid pressure (35). A threefold rise in the inflow pressure to the cerebral circulation would probably overwhelm cerebral autoregulation (24, 29), as it takes about 3 s for (human) autoregulation to respond to sudden increase in MAP (31). Other mechanisms are, therefore, likely to be involved in protecting the giraffes from cerebral edema or hemorrhage. The rete mirabile, a tortuous network of small veins and arterioles derived from the carotid artery upon entering the skull (15), has been speculated to reduce pressure within the giraffe’s cerebral arterial vessels by expansion of the venous vessels when the head is lowered (28).

An expansion of the highly compliant jugular vein when the head is lowered has also been suggested (15), and some correlation between head posture and MAP have been shown (35). However, the changes in dimension of the jugular vein
during postural changes have not been visualized, and its relation to central venous pressure (CVP) and blood pressure has not been documented. To study this, we determined changes in central and regional hemodynamic variables and made ultrasound images of the carotid artery and jugular veins in five spontaneously breathing anesthetized giraffes. The giraffes were suspended in a sling system designed to avoid pressure to the thoracic and abdominal regions as pressure in these regions was demonstrated to decrease cardiac output and carotid flow. The setup allowed us to lower and raise the head from its normal upright position to heart level by swinging the neck to one side or the other. Heart rate and blood pressure of the anesthetized giraffes were verified by telemetric measurements in one freely walking giraffe.

METHODS

Six young male giraffes (*Giraffa camelopardalis*) bred for trophy hunting in Namibia were caught and transported to a quarantine facility in Hambanskraal, South Africa. The giraffes were adapted to their new setting for 20 days prior to the experiments. The experimental protocol was approved by the Danish Inspectorate for Animal Experimentation, the Danish Ministry of Justice; by the Animal Ethics Screening Committee at the University of Witwatersrand, Johannesburg; and by the Animal Use and Care Committee, University of Pretoria, South Africa. Permission to euthanize the animals after experimentation was granted by the Gauteng Province of South Africa, and the experiments were supervised by local ethical committee members.

Animal handling and anesthesia. Following an overnight fast and 2 h without water, the giraffes were premedicated by remote injection (Daninject, Berkop, Denmark) with medetomidine (~8 μg/kg; dose calculated from the estimated body mass). After ~5 min, the sedated giraffe was guided to a chute where a halter was mounted on the head, and the animal was blindfolded. Anesthesia was induced with etorphine (3.9 μg/kg) and ketamine (0.9 mg/kg im), and after ~2 min the giraffe was led into an adjacent pen where it became recumbent within minutes. A rope connected to the halter and passed through a pulley in the ceiling allowed control of the head to avoid injury when the giraffe became recumbent.

Immediately after the giraffe became recumbent, a cuffed endotracheal tube (ID 20 mm) was inserted, guided by endoscopy to allow for ventilation with 100% oxygen by using a demand valve (Hudson RCI, Research Triangle Park, NC). The giraffe was then moved to an adjacent room and placed in right lateral recumbency with the head elevated ~1 m (Fig. 1A). Electrocardiogram, rectal temperature, end-tidal carbon dioxide tension (PetCO2), and tail cuff arterial pressure were recorded using a portable system (model PM9000Vet; E-Vet, Haderslev, Denmark). Blood gas variables were monitored (Opti CCA Apparatus; Osmetech, Roswell, Georgia) from blood collected from an auricular arterial catheter.

Anesthesia was maintained by intravenous infusion of α-chloralose at 30 mg/kg estimated body mass/h through a catheter in the saphenous vein. Infusion was decreased to 20 mg·kg−1·h−1 after 80 min, 15 mg·kg−1·h−1 after 120 min, and, thereafter, gradually to 3 mg·kg−1·h−1 over 4–5 h depending on reflexes and breathing pattern. Following local infiltration by lidocaine (2%, SAD, Copenhagen, Denmark), vascular sheaths were placed in the carotid artery and jugular vein at the base of the neck and sutured. In addition, two catheters were inserted through the sheaths in the carotid artery and jugular vein ~20 cm below the joint of the jaw and sutured. Straps were placed around each limb leaving the thoracic and abdominal regions free of external pressure, and the giraffe was hoisted to an upright position while the head was supported by a strap connected to the halter (Fig. 1B). This setup allowed the position of the head to be lowered to the level of the heart with a swing to the right or left side (Fig. 1C). It was not possible to lower the head below heart level because that required active ventroflexion.

To avoid influence of the induction agents (medetomidine and etorphine) on the vascular measurements by artificially elevating blood pressures, the effects were antagonized with naltrexone, and measurements were started > 90 min following its administration by which time the effects of ketamine would be minimal (32, 40).

Hemodynamic variables. Blood pressure was determined by inserting tip transducer catheters through the sheaths with a single pressure sensor mounted on the side of the tip (5 French Micro-Tip SPC 350, range 50–400 mmHg; Millar Instruments, Houston, TX). Two catheters were inserted through the sheaths at the base of the neck; one in the carotid artery was forwarded retrograde to the aortic arch for recording of MAP, while the catheter in the jugular vein was advanced toward the heart for recording of CVP. In addition, two catheters were inserted through the sheaths at the cranial end of the neck and were directed toward the heart to record the pressure profiles in steps of 20 cm. Carotid artery and jugular venous flows were recorded by transit time probes (Transonic, Ithaca, NY) inserted at the base of the neck. Cross-sectional area and blood velocity of the vessels were determined by ultrasound (Vivid I; GE Healthcare, Horton, Norway) with a 7-MHz linear transducer. Tissue pressure in the upper and lower part of the neck was assessed by positioning subcutaneous fluid-filled catheters (14 gauge; Arrow Teleflex). Spinal fluid pressure was...
Table 1. Morphological characteristics for the studied giraffes

<table>
<thead>
<tr>
<th>Giraffe No.</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6*</th>
<th>Means ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass, kg</td>
<td>370</td>
<td>490</td>
<td>450</td>
<td>470</td>
<td>280</td>
<td>360</td>
<td>412 ± 80</td>
</tr>
<tr>
<td>Height, cm</td>
<td>322</td>
<td>ND</td>
<td>335</td>
<td>340</td>
<td>290</td>
<td>ND</td>
<td>322 ± 23</td>
</tr>
<tr>
<td>Number of valves†</td>
<td>1</td>
<td>9</td>
<td>10</td>
<td>9</td>
<td>6</td>
<td>ND</td>
<td>7 ± 4</td>
</tr>
<tr>
<td>Heart weight, kg</td>
<td>1.7</td>
<td>1.9</td>
<td>2.3</td>
<td>2.3</td>
<td>1.3</td>
<td>1.8</td>
<td>1.9 ± 0.4</td>
</tr>
<tr>
<td>Heart weight/body weight %</td>
<td>0.46</td>
<td>0.39</td>
<td>0.51</td>
<td>0.49</td>
<td>0.46</td>
<td>0.50</td>
<td>0.47 ± 0.04</td>
</tr>
</tbody>
</table>

*Not included in in vivo measurements because the giraffe died due to fracture of the third cervical vertebra when falling backward following anesthesia.
†Values are for either the right or left jugular vein. Heart mass and jugular vein length were obtained after the tissue was fixed in 4% formaldehyde. ND, not determined.

measured in two giraffes by puncture of the cisterna magna. All measurements were sampled at 100 Hz using data acquisition software (AqKnowledge 3.7.2; Biopack Systems), and an 8-track DAT instrumentation recorder (model D-180; pulse-code modulation, TEAC, Tokyo, Japan) equipped with a microphone to mark events.

In two giraffes, carotid arterial and jugular venous blood samples were withdrawn in both the upright position and after the head had been lowered for measurements of oxygen concentration (Osmotech Opti CCA Apparatus). Assuming that oxygen uptake of the giraffe was unaffected by head position, changes in the arterial-venous oxygen concentration difference allowed for an estimate of changes in cardiac output using the Fick equation.

Protocol. After instrumentation, the giraffes were suspended in an upright position, and carotid and jugular pressure profiles were obtained with the head in the upright position. Subsequently, hemodynamic variables and vessel cross-sectional areas were obtained while changing the head position by bending the neck away from or toward the monitored vessels for ~1 min. All giraffes survived the procedures and were euthanized with intravenous pentobarbital (SAD) and sonication.

Telemetric monitoring. One giraffe was successfully instrumented with an implantable telemetric setup (model E-4311; EndoSomatic Tech) connected to pressure catheters (model SPC 350; Millar) for recording of carotid arterial and jugular venous pressures. This giraffe was awake and freely walking in a 4 × 4-m pen for 48 h before it was resedated, and experiments were performed as described.

Statistical analysis. Pressure and flow recordings in the upright and head-lowered positions were compared using one-way ANOVA for repeated measures. A statistical significance level of P < 0.05 was used, and data are expressed as means ± SE. Telemetric measurements are presented as the average of five recordings.

RESULTS

The morphological characteristics of the giraffes are presented in Table 1. The giraffes were suspended in an upright position within 95 ± 6 min after premedication, and the experiments lasted 343 ± 21 min. Blood pressures increased by ~10% during the protocol, and blood gas variables were assessed 5 min after the giraffe became recumbent and four times at predefined time intervals (Table 2). The head was 141 ± 3 cm above the estimated level of the heart when the giraffes were upright and at about heart level when it was lowered.

Histology. The average heart mass was 1.9 ± 0.4 kg, equaling 0.47 ± 0.04% of body mass (Table 1). Examinations of the jugular veins demonstrated an average of 7 bi- and tricuspid valves, with distance between the valves varying from 2 to 30 cm. One giraffe did not possess any valves in the left jugular vein and only one valve in the right jugular vein. Venous pressure when the head was lowered in that giraffe, however, was not significantly different from that of the others. No valves were observed in the carotid arteries.

Head upright. Arterial pressure in the carotid artery was 76 ± 4 mmHg lower at the cranial end than MAP (Table 3). Within the carotid artery, pressure decreased linearly when the catheter was moved distally along the length of the neck, and pressure in the jugular vein was slightly subatmospheric in the cranial end and close to zero at the lower end of the vein (Fig. 2). The lowest venous pressure recorded at the cranial end of the jugular vein was −21 mmHg. Carotid artery blood flow was similar to jugular venous blood flow, and ultrasound showed that the circumference of the carotid artery changed minimally within the cardiac cycle (see Supplemental Movie 1 posted with the online version of this article), whereas the jugular vein was almost collapsed. The carotid flow was pulsatile, while flow was steady in the collapsed jugular vein (Fig. 3). Spinal fluid pressure just below the head was 2 ± 0 mmHg, and neck tissue pressure was generally positive but varied among recording sites. At the cranial and the lower part of the neck, tissue pressures were 19 ± 18 and 11 ± 12 mmHg (n = 3), respectively.

Table 2. Blood gas variables during experimental period

<table>
<thead>
<tr>
<th>Minute, position</th>
<th>pH</th>
<th>PAO2, mmHg</th>
<th>PAO2, mmHg</th>
<th>HCO3−, mM</th>
<th>Base Excess, mM</th>
<th>SaO2, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>5, recumbent</td>
<td>7.1±0.1</td>
<td>58±10</td>
<td>59±15</td>
<td>18±3</td>
<td>−12±3</td>
<td>78±8</td>
</tr>
<tr>
<td>20, instrumentation</td>
<td>7.2±0.1</td>
<td>46±4</td>
<td>146±34</td>
<td>17±3</td>
<td>−12±4</td>
<td>98±1</td>
</tr>
<tr>
<td>60, instrumentation</td>
<td>7.3±0.1</td>
<td>48±2</td>
<td>143±37</td>
<td>22±3</td>
<td>−5±4</td>
<td>98±1</td>
</tr>
<tr>
<td>180, upright</td>
<td>7.4±0.1</td>
<td>53±5</td>
<td>455±25</td>
<td>30±3</td>
<td>3±4</td>
<td>100±0</td>
</tr>
<tr>
<td>300, upright</td>
<td>7.3±0.0</td>
<td>58±6</td>
<td>449±29</td>
<td>31±2</td>
<td>4±2</td>
<td>100±0</td>
</tr>
</tbody>
</table>

Values are means ± SE, n = 5. Variables presented at 5 min after the giraffe became recumbent. Instrumentation at 20 and 60 min is the time of surgical procedures. Upright at 180 and 300 min is the time with the giraffe hoisted to an upright position. PAO2, arterial carbon dioxide tension; PAO2, arterial oxygen tension; HCO3−, arterial bicarbonate; SaO2, hemoglobin oxygen saturation.
Carotid arterial and jugular venous pressures measured telemetrically in a freely walking giraffe were 107 ± 5 and −0.3 ± 0.1 mmHg, respectively. When the same giraffe was anesthetized and suspended in an upright position, telemetrically recorded values were 132 ± 7 and −0.8 ± 0.1 mmHg, respectively. Heart rate of the freely walking giraffe was 41 ± 1 and 42 ± 0 beats/min when the animal was anesthetized.

After all measurements were obtained, the support of one giraffe was moved from the base of the legs to the thoracic and abdominal regions, thereby exerting pressure to these regions. As a result, CVP increased by 14 mmHg, carotid flow decreased by 60% (the detection limit of the apparatus). This indicates that cardiac output fell by > 40%.

**Lowering of the head.** When the head was lowered, spontaneous ventilation ceased, and ventilation was then supported. Yet, an increase in PaCO2 and a 15% reduction in central venous oxygen saturation were observed, while 100% arterial oxygen saturation was maintained (Table 3). Thus, cardiac output decreased by ~30%, provided that oxygen uptake did not change.

Lowering and raising the head caused substantial changes in neck blood flows and pressures but did not affect heart rate (Table 3). Figure 4 shows a recording during the 1-min lowering of the head to the right side (away from the catheters) and subsequent lifting of the head.

At the cranial end of the carotid artery, pressure increased immediately to 203 ± 9 mmHg when the head was lowered and then gradually decreased to 134 ± 7 mmHg. This drop in distal carotid arterial pressure was accompanied by a drop in MAP to 131 ± 13 mmHg (32 ± 7%). Accordingly, with the head lowered, arterial pressure at the base of the skull was similar to MAP (Fig. 4, A and B). The decrease in MAP was paralleled by a decrease in CVP (Fig. 4D) and a 6 mmHg increase in spinal fluid pressure. Carotid blood flow increased immediately when the head was lowered and then returned to the level established when the head was upright (Fig. 4C). Jugular venous flow at the base of the neck ceased for ~30 s after the head was lowered and then recovered (Fig. 4F).

Cessation of jugular flow coincided with a progressive rise in cranial jugular pressure (Fig. 4E) and was associated with distension of the vein while no distension was observed in the carotid artery (Fig. 5). With the head lowered, flow in the
jugular vein, as detected by Doppler, only occurred when the neck was bent away from the catheters.

Lifting the head to the upright position immediately reestablished the pressure difference between the cranial and central part of the carotid artery and caused a large transient rise in jugular blood flow (peak flow \( \sim 12 \text{ l/min} \)) (Fig. 4). Integration of venous return indicated that 1.2 ± 0.2 l of blood accumulated in the jugular veins when the head was lowered. CVP was restored within 2–4 heart beats after the head was elevated, whereas it took \( \sim 1 \text{ min} \) to normalize MAP.

The surge of venous blood returning to the heart when lifting the head coincided with a decrease in the jugular venous cross-sectional area that demonstrated an immediate emptying of the vessel (Fig. 5). Emptying of the vein’s distal parts was visualized by ultrasound (see Supplemental Movie 2 at online version of this article). The ultrasound recording also showed that the carotid artery had little or no pulse-mediated change of its diameter during movement of the giraffe’s head.

**DISCUSSION**

This study confirmed the high MAP in giraffes (13–16, 18, 24, 35–37, 39) and that pressure in the carotid artery decreases above heart level in accordance with gravity (18, 37). As a result, the inflow pressure to the brain in the upright position corresponds to that of other mammals, including humans (38).

The ultrasound images of the jugular vein revealed a collapsed vein in the upright position and accumulation of venous blood when the head was lowered, which affected MAP.

Histological findings. A somewhat surprising histological finding was that the high blood pressure was not correlated with a large heart relative to body mass. The 0.5% relative heart mass found in the six young giraffes studied is similar to that of other mammals, including humans (11). Previous estimates of the relative heart mass of giraffes has been up to 2.3% of body mass (30). That estimate is based on the one report of a 11.3-kg heart from a fully grown giraffe by Goetz and Keen (15). However, body mass was not reported for the giraffe investigated, and it is possible that their reported heart mass included both blood and pericardial fluid. The relative heart mass that we found also agrees with an early report on giraffes by Crisp (9). An interesting implication from this observation is that the generation of the higher pressures takes place without the development of left ventricular hypertrophy that is commonly observed for humans with hypertension (22). The special architecture of the giraffe heart with a thick left ventricular wall, also referred to as gothic (7), might be involved in the heart’s increased ability to generate pressure, but the architecture of the heart needs further analysis for detailed evaluation of how a large blood pressure can be generated without myocardial hypertrophy.

Histological examinations revealed no valves in the carotid arteries, although such arterial valves have been mentioned in more popular accounts, such as the Encyclopedia Britannica Online. In the jugular vein, no uniform pattern in the distribution of bi- vs. tricuspidal valves was found, and, surprisingly, one giraffe possessed no valves in the (left) jugular vein.

**Jugular venous flow and pressure.** The cerebral circulation of the giraffe has been suggested to be governed by a siphon mechanism where the energy used to overcome gravity in the carotid artery is recovered on the venous side (2, 5). Numerous theoretical (6, 30) and mechanical models (4, 19, 25, 30), however, have not supported this notion and stress that a collapsed vein cannot support a siphon and venous return. Venous return may be better described by a waterfall analogy, where blood seeps through the small lumen of the vein.

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**Fig. 4. Trace of simultaneous venous and arterial measurements of central and cranial pressures (P), and flow in the neck when changing the head position of an anesthetized giraffe suspended in the upright position. Gray bars indicates the time the head was lowered.**
Our recordings of venous pressure close to zero throughout most of the length of the jugular vein demonstrate that the jugular vein had a minute cross-sectional area, in agreement with the waterfall analogy. We also observed a maintained small lumen in the cranial part of the jugular vein of the anesthetized giraffe, implying that a column of blood may be preserved in this part of the jugular vein. Consistent with this column of blood, there was recorded negative venous pressure at the cranial end of the jugular vein, while pressures in the dominant part approached zero. The pressures measured in our study agree with McCalden et al. (24), who measured a jugular venous pressure of 0 mmHg at the level of the first cervical vertebrae, while Mitchell and Skinner (28) measured a jugular venous pressure of 10 mmHg some 30 cm below the head. In contrast, Hargens et al. (18) reported that pressure decreased from 13 mmHg at the base of the skull to 4 mmHg ~30 cm above heart level in sedated giraffes. It is not evident why jugular venous pressure varies so much between studies. The experiment in which one giraffe was supported under the thoracic region suggests that the protocol used for suspension substantially affects the hemodynamic variables, but the key finding of the present study is that jugular venous pressure of the giraffe depends on the position of the neck. Yet, we acknowledge that differences in the reported jugular venous pressures might also reside from differences in protocols for sedation and anesthesia, although such differences are more likely to affect arterial than venous pressure.

Jugular viscous resistance. As pointed out by Badeer (3), pressure at any given location in the jugular vein is the sum of the viscous flow pressure and the gravitational (hydrostatic) pressure. Thus, the low pressure in the jugular vein close to the brain supports a certain venous drag on cerebral blood flow, but along the length of the jugular vein, viscous resistance counterbalances the influence of gravity on pressure. A negative pressure, therefore, implies that a siphon mechanism is active in the upper part of the jugular vein, whereas the pressure 1 m above the heart indicates that the vein is functionally collapsed. From that perspective, the lowest recording of cranial jugular pressure (~21 mmHg) indicates that the open part of the vein may reach some 30 cm. The collapsed vein was confirmed by ultrasound, and the flow measurements suggest that flow is steady through the small lumen of the collapsed vein. Spinal fluid pressure below the level of the brain was close to zero, in accordance with the estimate for humans (10).

By integrating flow, cross-sectional area of the jugular vein, and the blood viscosity for the giraffe (15) in Poiseuille's law, we estimated the relationship between jugular venous cross-sectional area and the viscous pressure drop in a 1-m long segment of the vein (Fig. 6). The estimated viscous pressure drop is well within a range where it can counterbalance the
depressed in the anaesthetized giraffes. Although peripheral resistance, as expected if the autonomic reflexes are mated from the oxygen saturation. This could indicate that the matches the calculated 30% reduction in cardiac output esti-
and, hence, cardiac output. The percentile reduction in MAP CVP, both indicating reduced cardiac filling and stroke volume
was lowered. This could reflect a depression of autonomic anesthesia. Furthermore, the 30% reduction of was lowered. This could reflect a depression of autonomic reflexes by the anesthesia. Whether the slow return of MAP could be explained by the volume of translocated blood or is a consequence of lacking baroreceptor function is not known. Furthermore, the muscular structure in the lower part of the vena cava (15) or pooling of blood in the pulmonary circulation due to altered breathing patterns in the suspended position could be involved. To evaluate these possibilities requires a setup that allows for measurements in naturally drinking, awake giraffes.

**Perspectives and Significance**

In line with the August Krogh principle that suggests that for many physiological problems there will be an animal of choice on which it can be most conveniently studied, giraffes and other long-necked animals can provide fundamental insights to the effects of gravity on the circulatory system of all animals.

Simultaneous measurements of blood flow and pressure show that cerebral perfusion of anaesthetized upright positioned giraffes is maintained by the high arterial blood pressure and that the jugular vein is collapsed in the upright position. This implies that the vascular resistance of the jugular vein

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**Figure 6.** Change in viscous pressure drop per meter as a function of jugular venous cross-sectional area for jugular blood flow at 0.6 l/min in the giraffe. Blood viscosity is taken to be 0.0031 kg·m⁻¹·s⁻¹ (15). Dotted lines indicate cross-sectional area of the jugular vein as measured by ultrasound in the upright position (0.14 ± 0.04 cm², n = 4; mean ± SE) and when the head was lowered (3.19 ± 0.5, n = 4) in anesthetized giraffes.

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**Graph:**
- **X-axis:** Cross-sectional area (log cm²)
- **Y-axis:** ΔP (mmHg/cm)
- **Data points:** Head up and Head lowered

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**Equation:**
- Blood viscosity = 0.0031 kg·m⁻¹·s⁻¹
- Cross-sectional area of jugular vein measured by ultrasound:
  - Upright position: 0.14 ± 0.04 cm² (mean ± SE)
  - Lowered position: 3.19 ± 0.5 cm²

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**Table:**
- Blood viscosity: 0.0031 kg·m⁻¹·s⁻¹
- Cross-sectional area:
  - Upright position: 0.14 ± 0.04 cm²
  - Lowered position: 3.19 ± 0.5 cm²

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**Notes:**
- The maintenance drug in our study, provides stable hemodynamic variables over long periods in a wide range of mammals, including domestic ruminants (8, 12, 34), we acknowledge that baro- and volume receptor function might have been affected. However, neither can we exclude the possibility that the baro- and volume receptors have sensed the ~4% reduction in blood volume. It, therefore, remains to be determined whether changes in overall peripheral resistance contributes to the reduction of MAP when the head is lowered in awake giraffes.

The ultrasound images revealed a pronounced expansion of the jugular vein when the head was lowered, and the volume of blood that accumulated in the veins was determined to be 1.2 liters, or ~4% of the estimated blood volume (16). The volume in the jugular vein and the increase in spinal fluid pressure may well have been larger if a lower head position had been obtained, as by the drinking position. Nevertheless, the accumulated volume is considerably smaller than previously suggested (26, 27), and it remains to be established whether translocation of this relatively small volume of blood to the jugular vein is sufficient to significantly decrease cardiac filling when the head is lowered. Alternatively, lowering of the head may reduce cardiac filling by altering the hydrostatic indifference point, and it is possible that blood pressure regulation by the arterial baroreceptors positioned at the base of the skull in the giraffe (21) are important. Although these mechanisms warrant further evaluation, we suggest that the reduction in MAP decreased the rise in blood pressure at the level of the head and, thereby, contributed to protecting brain capillaries when the head is lowered. Furthermore, subdural venous collapse (17), along with the increase in spinal fluid pressure when lowering the head, may act to protect the cerebral vasculature by decreasing the pressure difference between the capillary blood and spinal fluid.

**Lowering the head.** When lowering the head to drink, the giraffes spread and bend their front legs and, thereby, also lower the level of the heart. Because the giraffes in this study were suspended, the head could only be lowered to heart level. Lowering of the head to this position caused an immediate increase in carotid pressure at the inlet to the brain that corresponded to the change in gravitational force. Thus, it would be expected that the rise in carotid pressure would have been even higher if the head had been lowered to the ground, as when the giraffes drink. The initial steep rise in pressure was followed by a progressive decrease in carotid pressures at the inflow to the brain, as well as in MAP, and (after ~45 s) restoration of jugular flow. A consequence of these observations is that the pressure difference between the cranial part of the carotid and jugular vein is decreasing by ~20%, while jugular flow is increasing. This scenario indicates a reduction in cerebral vascular resistance with lowering of the head, as suggested by Mitchell et al. (26). This is somewhat surprising and not consistent with precapillary vasoconstriction during lowering of the head as part of an autoregulatory response (24).

The one telemetric recording of carotid pressure in a drinking giraffe also recorded a fall in MAP during drinking (36), but this was accompanied by bradycardia, presumably as a result of stimulation of carotid arterial baroreceptors (21). In the anesthetized giraffes, heart rate did not change when the head was lowered. This could reflect a depression of autonomic reflexes by the anesthesia. Furthermore, the 30% reduction of MAP after lowering the head coincided with a reduction in CVP, both indicating reduced cardiac filling and stroke volume and, hence, cardiac output. The percentile reduction in MAP matches the calculated 30% reduction in cardiac output estimated from the oxygen saturation. This could indicate that the reduction in MAP was independent of a reduction of total peripheral resistance, as expected if the autonomic reflexes are depressed in the anaesthetized giraffes. Although ΔP = ∆V/

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**Equation:**
- Blood flow rate = 0.6 l/min
- Viscosity = 0.0031 kg·m⁻¹·s⁻¹
- Cross-sectional area of jugular vein:
  - Upright position: 0.14 ± 0.04 cm²
  - Lowered position: 3.19 ± 0.5 cm²

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**Conclusion:**
- The reduction in MAP is not explained by a reduction in total peripheral resistance, as expected if the autonomic reflexes are depressed in the anaesthetized giraffes. Although ΔP = ΔV/

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**Further Reading:**
- Mitchell et al. (26)
- Subdural venous collapse (17)
- Hydrostatic indifference point

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**References:**
counterbalances the gravitational pressure profile and, thus, there is no indication of a functional siphon. The pronounced accumulation of blood in the jugular vein when the head was lowered affected MAP. This points to an important role of venous distensibility in influencing how gravitational pressures affect the cardiovascular system during postural changes. It is, therefore, interesting to study whether similar mechanisms exist in other long-necked animals such as ostriches and camels, as well as to repeat this study on free-ranging animals (1) to avoid any effect of anesthesia and to obtain measurements from naturally behaving unstressed animals.

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DISCLOSURES

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