Control of blood pressure mediated by baroreflex changes of heart rate in the chicken embryo (Gallus gallus)

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Altimiras, Jordi, and Dane A. Crossley II. Control of blood pressure mediated by baroreflex changes of heart rate in the chicken embryo (Gallus gallus). Am J Physiol Regulatory Integrative Comp Physiol 278: R980–R986, 2000.—Pharmacological manipulation of peripheral resistance via sodium nitroprusside and phenylephrine was used to study baroreflex function over the latter two-thirds of incubation in embryonic chickens. From day 9 to day 19 of incubation, there is a positive linear relation between heart rate and blood pressure, indicating the feedforward action of arterial pressure on heart rate. A reciprocal relationship between blood pressure and heart rate became pronounced during the last 3 days of incubation. For the purpose of the study, gain of the baroreflex was calculated as maximal gain (only those embryos that demonstrated the response) or average gain (all embryos). Maximal gain increased progressively from 13 ± 7 beats·min⁻¹·kPa⁻¹ at 18 days to 105 ± 83 beats·min⁻¹·kPa⁻¹ in 2-day-old hatchlings. The percentage of embryos older than 18 days with baroreflex responses increased from 33% on day 19 to 56% on day 21, indicating that baroreflex regulation begins late in incubation (~90% incubation time), and the gain of this reflex exhibits a maturation over the final 3 days of incubation.

ontogeny; cardiovascular regulation; negative feedback; baroreflex gain

PRESSURE DIFFERENCES between the arterial and venous sides of the cardiovascular system provide the necessary driving force to ensure adequate gas transfer to tissues. Alterations in arterial pressure that may compromise gas transfer are counteracted by both central and local homeostatic mechanisms. These mechanisms offset the perturbation, thereby avoiding hypertensive or hypotensive episodes and the myriad of pathologies these conditions could induce (28).

Among regulatory mechanisms, the baroreflex is the most prominent short-term compensator during arterial pressure challenges. This compensatory system results in an alteration of cardiac output and peripheral resistance via the cardiac and peripheral limb of the baroreflex, respectively. Although baroreflex responses are well characterized in a variety of adult vertebrates (1), little is known of the maturation of this mechanism.

In the fetal sheep, the standard model in mammalian studies of cardiovascular physiology, an unequivocal baroreflex response is present during the final trimester of gestation (6, 16, 22, 23). Although this model is attractive for its link to clinical pediatrics, an alternative experimental model with short extrauterine development would be instrumental in exploring the course of baroreflex maturation at different organizational levels. Such a model would also be important to the construction of a generalized picture of the ontogeny of cardiovascular regulation in vertebrates.

The chicken embryo may be such a model, with the advantage of a shortened gestational time, ease of embryonic manipulation, and a mammalian-like circulation with an extraembryonic circuit involved in gas exchange (the chorioallantois) analogous to the placenta (17). Furthermore, its use in the analysis of chemoreflexive cardiovascular regulation has recently been shown (18).

Finally, the maturation of many physiological processes is well characterized in chicken embryos, providing essential information for an in-depth study. Previous studies on autonomic cardiovascular regulation indicate that functional vagal innervation appears on day 12 of development (19), implying that a hypertensive baroreflex could be operational during the latter half of chicken ontogeny. The intention of this work was to test the hypothesis that baroreflex function appears during incubation and follows a progressive maturation as it occurs in the fetal lamb.

MATERIAL AND METHODS

Animals and incubation conditions. Experiments were conducted in two separate locations. Chicken embryos up to 19 days of incubation were studied at the Department of Biological Sciences, University of Nevada Las Vegas (referred as Lab 1). Chicken embryos from 19 to 21 days of incubation, as well as hatchlings, were studied at the Department of Zoophysiology, University of Aarhus (referred as Lab 2). Incubation conditions were 38 ± 0.5°C and 60% humidity, and the eggs were turned automatically every 3 h.

Lab 1 studies used White Leghorn chicken eggs of the Hyline strain purchased from Hyline (LakeView, CA) and incubated in a walk-in environmental chamber (Labline Instruments, Melrose Park, IL). Lab 2 studies used Plymouth chicken eggs of the Russ208 strain purchased from Fælles-
rugeriet (Randers, Denmark) and incubated in a custom-made incubator.

Cannulation procedures. At selected developmental ages (see below), eggs were removed from the incubator and candled to locate a major chorioallantoic artery. Arteries were distinguished from veins attending to their course under the shell and the direction of vessel branching. Once the course of the artery had been traced, it was exposed by opening a small window (smaller than 10 × 10 mm) in the eggshell and peeling off the underlying shell membranes. A fluid-filled polyethylene cannula (PE-90, 1.27 mm OD, 0.86 mm ID, Clay-Adams) heat pulled to a tip diameter <0.5 mm OD was used to occlusively catheterize the smallest branching artery. The operation was carried out under a dissection microscope with the aid of fine microsurgery instruments under temperature-controlled conditions. Surgical procedures were completed within 15 min. Special care was taken to avoid bleeding of the chorionic cotyledon. In all procedures a ligature was placed downstream to eliminate retrograde flow, and heat cautery was used for smaller vessels. The catheter was then carefully aligned with the vessel and glued to the eggshell with tissue glue (VetBond 3M). After the procedure was completed, eggs were placed in a temperature-controlled vermiculite bath (38°C) for the duration of each trial, commonly <1 h.

Staging of the late-developing embryos was carried out not only on the basis of incubation time but also on pipping status. Commonly, internal pipping started during day 19 of incubation and external pipping during day 20. To avoid the confounding effects of pipping on cardiovascular hemostasis, we labeled 19-day-old embryos as prepiping embryos, 20-day-old embryos as internally pipped, and 21-day-old embryos as externally pipped.

Studies on recently hatched chicks were conducted at 2 days posthatch under halothane anesthesia. The animals were anesthetized with halothane in a closed chamber until righting and corneal reflexes disappeared. Halothane (0.5–1%; in air) was delivered thereafter through a plastic gas flow chamber adjusted to the top edge of the eggshell (Fig. 1). Once the experiment was completed, the animal was euthanized and quickly frozen at −20°C. After this procedure, the egg was cut along the longitudinal axis to determine the relative position of the embryo and the heart. The distance from the heart to the eggshell was then taken as a pressure head (error) offsetting the measured values.

Experimental protocol and drug infusion. The experimental protocol consisted of pharmacological manipulation of mean blood pressure via a dose-related alteration of peripheral resistances. The α-adrenoceptor agonist phenylephrine (Phe) was used to increase MAP by inducing a generalized vasoconstriction of the peripheral vasculature, while sodium nitroprusside (SNP) induced general vasodilation by the release of nitric oxide, as shown in Fig. 2.

The injection volumes were normalized to 5% of the total blood volume at each embryonic age (obtained from literature data, Ref. 21). After the drug was injected (one-third of the total injection volume), the catheter was subsequently flushed with saline (two-thirds of the total injection volume). The total volumes were as follows: 40, 80, 120, 150, 150, 150, 135

![Fig. 1. Schematic drawing of experimental setup. In particular, notice reference point, calibrated as zero pressure and situated on upper eggshell edge, not at level of atrium. Subsequently, pressures were corrected by adding offset factor as described in MATERIAL AND METHODS.](http://api.genesiscollection.com/981/)

![Fig. 2. Changes in blood pressure (BP; kPa) and heart rate (HR; beat/min) after injection of sodium nitroprusside and phenylephrine. Data from 2 individuals: 9-day-old embryo, dotted line; 21-day-old embryo, continuous line. CAM, chorioallantoic.](http://api.genesiscollection.com/981/)
μ in 9, 12, 15, 18, 19, 20, and 21 days, respectively. Preliminary experiments demonstrated that a bolus of saline of 5% total blood volume had no apparent effects on the variables analyzed. The dead space of the injection system was reduced by shortening the catheter from the point of injection (just outside the shell, leaving a very short length of catheter tubing, between 1-3 cm).

Drug concentrations were based on preliminary tests that established the degree of vasoactivity. All dosages were normalized to the total wet mass of the embryos and egg membranes and ranged between 20 and 100 μg/kg for SNP and Phe.

Increasing concentrations of one drug randomly selected were injected, and the effects on heart rate and blood pressure were recorded until pressure values returned to control conditions. The entire protocol lasted <60 min to avoid excessive water loss from the egg when the eggshell is opened. Embryos were then euthanized with Xylocaine (5 mg) before freezing. Hatchlings were euthanized with halothane as well as injection of potassium chloride through the femoral catheter.

Calculations and statistics. The gain of the baroreflex was calculated as Gain = (–1)·fH/MAP and expressed as beats per minute per kilopascal.

Baroreflexes are characterized by reciprocal responses between heart rate and blood pressure. Thus the slope of this response should be negative and the gain should be positive if baroreflex responses were present.

To test the significance of the measured baroreflex gain, a particular t-test was employed (see Ref. 24 for details). The basis for this test is that if fH and MAP were independent of each other, baroreflex gain should be zero. Thus the experimental gain at each stage (n) was tested against a predicted null gain (μ = 0) using the two-tailed t-distribution and the following t-factor

\[ t = \frac{x - \mu}{0.707 \cdot \frac{\sigma}{\sqrt{n}}} \]

where σ is the standard deviation of the experimental gain and n is the number of observations.

Resting blood pressures between stages were compared using a one-way ANOVA and the Newman-Keuls post hoc test. fH-MAP sensitivity was analyzed using common linear regression methods. Tests were carried out using STATISTICA 98 edition (version 5.1).

For intraspecific and interspecific comparisons of baroreflex gains, gain was normalized with respect to control MAP and fH as follows: Normalized gain = Gain·MAP/fH and expressed as percent change in fH per percent MAP.

Data are shown as means ± SE. Significant differences were all taken at the fiducial level P < 0.05.

RESULTS

As seen in Table 1, MAP shows a consistent increase during incubation, from 1.01 ± 0.02 kPa at day 9 to 3.18 ± 0.28 kPa during external pipping (21 days). Blood pressure in earlier stages (9, 12, and 15 days) was significantly lower than in later stages (P < 0.05). No differences in blood pressure were observed between the two strains of chickens used at 19 days (2.68 ± 0.29 kPa White Leghorn vs. 2.40 ± 0.21 kPa Plymouth).

In early embryos, the patterns of change in fH and blood pressure were similar as vascular tension was manipulated. This trend was altered in late embryos, which exhibited a reciprocal relation between parameters, i.e., increased pressure-induced bradycardia and decreased pressure-induced tachycardia (Fig. 2). Changes in fH-MAP sensitivity over development are detailed in Table 2. With the use of a total of 139 pharmacological injections in 53 embryos and hatchlings, fH-MAP sensitivity can be estimated as the average slope (Fig. 3). Slope of the fH-MAP curve was positive during early development up to 19 days, indicating a lack of baroreflex responses. The largest slope of 204 beats·min⁻¹·kPa⁻¹ was observed on day 9 and decreased to 9 beats·min⁻¹·kPa⁻¹ at 19 days. The coefficient of determination (r²), i.e., the amount of variance explained by a linear relationship between both variables decreased likewise from 88% at 9 days to 17% at 19 days (see Table 2). In late embryos (21 days) and 2-day-old hatchlings, fH-MAP was reciprocal, indicating active baroreflex function. This was best shown by excluding those drug trials where fH and MAP varied concurrently, thus providing a maximal estimate of the fH-MAP slope when only baroreflex responses occurred. In this case, the maximum slope was negative from 18 days onward and peaked on day 20 at –34 beats·min⁻¹·kPa⁻¹ (P < 0.05; Table 2).

These results were mirrored when calculated on a per animal basis as shown in Fig. 4A and Table 3. The

<table>
<thead>
<tr>
<th>Days of Incubation</th>
<th>Strain</th>
<th>MAP</th>
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<tbody>
<tr>
<td>9</td>
<td>WL</td>
<td>1.01 ± 0.02 (3)*</td>
</tr>
<tr>
<td>12</td>
<td>WL</td>
<td>1.08 ± 0.08 (4)*</td>
</tr>
<tr>
<td>15</td>
<td>WL</td>
<td>1.15 ± 0.08 (5)*</td>
</tr>
<tr>
<td>18</td>
<td>WL</td>
<td>2.22 ± 0.17 (6)†</td>
</tr>
<tr>
<td>19</td>
<td>WL</td>
<td>2.68 ± 0.29 (4)†</td>
</tr>
<tr>
<td>19</td>
<td>Ply</td>
<td>2.40 ± 0.21 (8)†</td>
</tr>
<tr>
<td>20</td>
<td>Ply</td>
<td>2.80 ± 0.13 (9)†</td>
</tr>
<tr>
<td>21</td>
<td>Ply</td>
<td>3.18 ± 0.28 (9)†</td>
</tr>
<tr>
<td>Hatch</td>
<td></td>
<td>2.40 ± 0.33 (5)†</td>
</tr>
</tbody>
</table>

Data are means ± SE; n in parentheses. MAP, mean arterial pressure (kPa); WL, White Leghorn strain; Ply, Plymouth strain. Dissimilar symbols indicate significant differences in MAP.

<table>
<thead>
<tr>
<th>Days of Incubation</th>
<th>Slope</th>
<th>r</th>
<th>Slope max</th>
<th>r</th>
</tr>
</thead>
<tbody>
<tr>
<td>9</td>
<td>204 (7)</td>
<td>0.94*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>91 (7)</td>
<td>0.76*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>160 (8)</td>
<td>0.83*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>16 (9)</td>
<td>0.73*</td>
<td>–2 (2)</td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>9 (33)</td>
<td>0.41*</td>
<td>–25 (7)</td>
<td>0.92*</td>
</tr>
<tr>
<td>20</td>
<td>0 (36)</td>
<td>0.00</td>
<td>–34 (12)</td>
<td>0.74*</td>
</tr>
<tr>
<td>21</td>
<td>–5 (31)</td>
<td>0.26</td>
<td>–16 (14)</td>
<td>0.56*</td>
</tr>
<tr>
<td>Hatch</td>
<td>–21 (8)</td>
<td>0.44</td>
<td>–13 (4)</td>
<td>0.28</td>
</tr>
</tbody>
</table>

Slope-heart rate (fH)-MAP slope is in beats·min⁻¹·kPa⁻¹. No. of trials in parentheses. r indicates goodness of fit to a linear regression of data. Maximum slope (Slope max) fH-MAP slope given when only baroreflex responses were considered. * Significant difference from zero sensitivity (P < 0.05).
average baroreflex gain (Gain) was negative during early development up to 19 days, with a minimum value of $-246 \pm 75 \text{ beats} \cdot \text{min}^{-1} \cdot \text{kPa}^{-1}$ at 15 days and increased progressively to $-37 \pm 0 \text{ beats} \cdot \text{min}^{-1} \cdot \text{kPa}^{-1}$ at 19 days (Fig. 4A). Values from days 9 and 15 were significantly different from all other stages ($P < 0.05$). Later in development, gain became positive, ranging between 25 ± 19 at 20 days and 53 ± 43 beats·min$^{-1}$·kPa$^{-1}$ at 21 days. Only the highest sensitivity on day 15 was significantly different from zero ($P < 0.05$).

The maximal gain of the baroreflex, obtained by averaging baroreflex gain of the animals with prevalent baroreflex, was positive from day 18 to the end of development, increasing progressively from 13 ± 7 at 18 days to 105 ± 83 beats·min$^{-1}$·kPa$^{-1}$ in 2-day-old hatchlings. In Fig. 4B, the relative proportion of animals with prevalent baroreflex responses is shown. Responses with sensitivities ±10 beats·min$^{-1}$·kPa$^{-1}$ were considered uncertain. No animals had a prevalent baroreflex until day 18. The frequency increased to 33% at 19, 44% at 20, and 56% at 21 days.

**DISCUSSION**

The present investigation was undertaken in an effort to establish the critical period at which baroreflex regulation becomes operational in chicken embryos. In addition, the gain of the reflex was estimated to provide a maturational picture of its development. The data definitely show that the baroreflex is inactive throughout 90% of ontogeny, and it demonstrates a progressive maturation of function in late prenatal and early neonatal periods.

**Table 3. Baroreflex gain in the latest stages of development**

<table>
<thead>
<tr>
<th>Days of Incubation</th>
<th>Gain</th>
<th>%Gain</th>
<th>Gainmax</th>
<th>%Gainmax</th>
</tr>
</thead>
<tbody>
<tr>
<td>18</td>
<td>-10 ± 8 (6)</td>
<td>-0.09</td>
<td>13 ± 7 (2)</td>
<td>0.06</td>
</tr>
<tr>
<td>19</td>
<td>-37 ± 0 (12)</td>
<td>-0.41</td>
<td>21 ± 7 (4)*</td>
<td>0.22</td>
</tr>
<tr>
<td>20</td>
<td>25 ± 19 (9)</td>
<td>0.25</td>
<td>40 ± 21 (7)*</td>
<td>0.44</td>
</tr>
<tr>
<td>21</td>
<td>53 ± 43 (9)</td>
<td>0.48</td>
<td>60 ± 48 (8)</td>
<td>0.55</td>
</tr>
<tr>
<td>Hatch</td>
<td>40 ± 38 (5)</td>
<td>0.34</td>
<td>105 ± 83 (2)</td>
<td>0.93</td>
</tr>
<tr>
<td>Adult</td>
<td></td>
<td></td>
<td></td>
<td>0.92</td>
</tr>
</tbody>
</table>

Data are means ± SE; n in parentheses. Gain, average baroreflex gain (beats·min$^{-1}$·kPa$^{-1}$); %Gain, normalized Gain (unitless variable); Gainmax, maximal baroreflex gain (see MATERIAL AND METHODS for details) (beats·min$^{-1}$·kPa$^{-1}$); %Gainmax, normalized maximal baroreflex gain (unitless). *Significant differences from zero gain ($P < 0.05$).
periphery, would be ±0.2 kPa, equivalent to a range of ±20–6% difference in pressure from days 9 to 21. A similar method has also been used in the fetal lamb by adjusting the zero reference to the midline of the uterus (23). Therefore, the reference method proposed here or an alternative should be considered if working with embryos prior to 9 days of age.

An additional criticism could be the use of pharmacological tools to manipulate peripheral resistance in an attempt to invoke the cardiac limb of the reflex. This experimental approach eliminates an important component of the baroreflex and potentially could bias our conclusions. Indeed, several studies in the fetal lamb established that the use of Phe for baroreceptor loading renders higher gains in comparison to the use of vascular occluders (6, 16). It is important to recognize that although sensitivities of the cardiac limb might be overestimated, a reflex was not masked by pharmacological challenge in the fetal lamb, indicating that this approach is valid.

Comparison with previous studies and between strains. Blood pressure values reported in this study are in good agreement with previous work on chicken embryos (Fig. 5) and display a progressive rise throughout development. No significant pressure differences were evident between the two strains of chicken used (White Leghorn and Plymouth) at 19 days of incubation (Table 1). Subsequently, the results were pooled and the two strains were considered identical in relation to blood pressure.

Arterial pressures measured in neonates (2 days posthatching) in this study were considerably lower than previously measured in nonanesthetized animals (10). This could be attributed to a depression in cardiac function usually associated with halothane anesthesia (3).

Onset of baroreflex function. As shown in Fig. 2, cardiovascular responses were markedly different between early and late embryos. Late embryos, starting at 18 days, demonstrated a reaction consistent with a functional adult baroreflex response. However, considering the low gains of the baroreflex at this stage (Table 3), the onset of baroreflex regulation is more appropriately placed at 19 days of incubation. This is equivalent to 90% of incubation in chicken embryos, which is within the range of baroreflex onset in several mammalian species. It should be noted, however, that little information can be gained from a direct comparison, because the range between species is from 60% of gestation in the fetal lamb (23) to neonatal appearance in the rabbit (11).

In chicken embryos, the baroreflex activation at 90% incubation time does not appear to be an all or none event. Activation was progressive, with the percentage of embryos demonstrating a baroreflex gradually increasing over the last 10% of development. Mild reflexive responses were first evident in 18-day-old embryos, whereas the average slope ΔfΔMAP was positive due to the limited number showing a negative slope. If positive slopes are excluded, the average gain in 18-day-old embryos is positive (13 ± 7 beats·min⁻¹·kPa⁻¹). By 19 days, 33% of embryos showed consistent baroreflex responses, with an increasing proportion on days 20 and 21. This finding was similar to that reported in the fetal lamb (23), in which the frequency of animals displaying a baroreflex response increased from 60 to 80%. The variability of activation appearance of the baroreflex between individuals was unexpectedly wide. This result suggests that the onset is less reliant on developmental age, possibly indicating that other factors, such as MAP, the onset of lung ventilation, or the relative contribution of changes in vascular resistance, may vary between individuals.

Data from fetal sheep provide evidence for the importance of other factors determining the onset of a baroreflex. Results from the fetal lamb have shown that the baroreflex is triggered only when a set point for arterial pressure is reached. At the same time, the operational set point increases with development, simultaneous to the blood pressure increase (4, 22). Although it seemed possible that individual differences in resting arterial pressure could explain the absence of baroreflex responses, we found no correlation between reflex gain and control MAP. Thus this may not explain the variation in onset age demonstrated in chicken embryos.

A possible coupling between the onset of lung ventilation and the onset of baroreflex control is unlikely given

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**Fig. 5.** Comparison between mean blood pressure (MAP) and baroreflex gain in chicken (A) and sheep (B) from start of incubation to adulthood. Blood pressures shown by dotted line. ○, Blood pressure values from this study (means ± SE) in chicken plot; notice that most of the error bars are inscribed within symbols. ▲, broken line, baroreflex gain. Mean arterial pressure data obtained from previous studies (10, 12, 25–27). Data on baroreflex gain based on results from this study and previous literature (2, 6, 16, 22, 23). Hatching time is indicated by shaded line.
that a baroreflex is present in fetal sheep before the onset of breathing. Furthermore, a significant portion of chicken embryos during days 20 and 21 showed no baroreflex activity, despite the fact that these animals were ventilating.

A change in the relative contribution of each limb of the baroreflex between embryos remains a viable possibility, but without new experimental data, this is only a speculation. It is possible that embryos relying on the peripheral limb of the reflex show a diminished heart rate response during pressure changes. In fact, studies in the fetal lamb have suggested that the baroreflex is more dependent on peripheral resistance than on heart rate (4). Clearly, this could also be the case in chicken embryos, but, as stated, extensive experimentation is necessary to clarify this issue.

Pre- and postnatal maturation of the baroreflex. Baroreflex maturation began during late embryonic development (Fig. 4), with maximal baroreflex gain increasing progressively from 18 days to hatching (Table 3). With the use of literature values for adult chickens (2), this maturation is incomplete at hatch and must continue rising to reach a normalized gain of 0.92 from 0.55 \( \% \Delta f_p \% \Delta MAP^{-1} \) at 21 days. Before this developmental age, \( \Delta f_p/\Delta MAP \) slope was positive, indicating a feedforward influence of arterial pressure on heart rate. The feedforward effect evident in this work has also been observed in a prior study on the fetal lamb (8) and is partially attributed to pharmacological alterations in arterial elastance and venous compliance, which changed venous return, stretch on the sinoatrial node, and, ultimately, heart rate. These characteristics may also be present in embryonic chickens, possibly explaining the pattern found in early stages of development. However, the feedforward effect was less accentuated in 18-day-old embryos and older, indicating the appearance of reflex cardiovascular regulation, via baroreflexive and chemoreflexive regulation (18).

Comparing the development of arterial pressure and baroreflex regulation in the chicken embryo and the fetal lamb (Fig. 5), the baroreflex in the lamb appears when arterial pressure is 50% of adult blood pressure, whereas in chicken embryos, blood pressure is only 21% of the adult arterial pressure. Thus it appears unlikely that baroreflex maturation follows a similar pattern in different vertebrate species. Although this conclusion has been formulated before (11), it remains undemonstrated, prompting for more detailed interspecific studies to understand the driving forces of baroreflex regulation.

Perspectives

This work has provided the basis for two conclusions. First, there is a late onset of baroreflex regulation, and, second, the gain of the reflex exhibits maturation over the final 3 days of incubation in embryonic chickens. Although the maturation of baroreflexive cardiovascular control is clearly significant for newly hatched precocial chicks, its late appearance questions its importance in ovo. Indeed, the pharmacological manipulation of blood pressure shows the potential for embryonic baroreflex regulation, but fails to reveal if baroreceptors are stimulated during normal development.

In the fetal lamb, neural recordings from baroreceptor afferents in the carotid nerve are in synchrony with the pressure pulse (4), indicating their role in cardiovascular regulation. In addition, baroreceptor denervation in fetal sheep causes an increased variability in blood pressure, suggesting that blood pressure homeostasis is impaired without baroreflex feedback (13, 29). Thus baroreflex control of blood pressure is relevant for the correct development of peripheral resistance (13) and aids in the proper development of organ systems (7).

Finally, baroreflex control has been implicated in cardiovascular homeostasis during fetal movements, specifically fetal breathing movements (5, 9). Although movements in the chicken embryo occur as early as day 5 and increase substantially at the end of development (14), distinct individualized movements of the thoracic muscles do not occur until 17–18 days of incubation (15). Given the space restriction due to the size of the embryo, the compression of vessels and sudden changes in cardiovascular resistance would be more likely at those late stages. In addition, the initiation of increased pulmonary blood flow, which must occur during internal and external pipping, could result in resistance changes requiring fast regulation from the baroreflex.

The correlation between baroreflex onset, as well as gain, and embryonic breathing movements or proper organ development needs to be verified experimentally.


