• سخنرانی کلیدی

ناهنجاری های تنفسی در طیور

هیئت علمی دانشگاه تهران

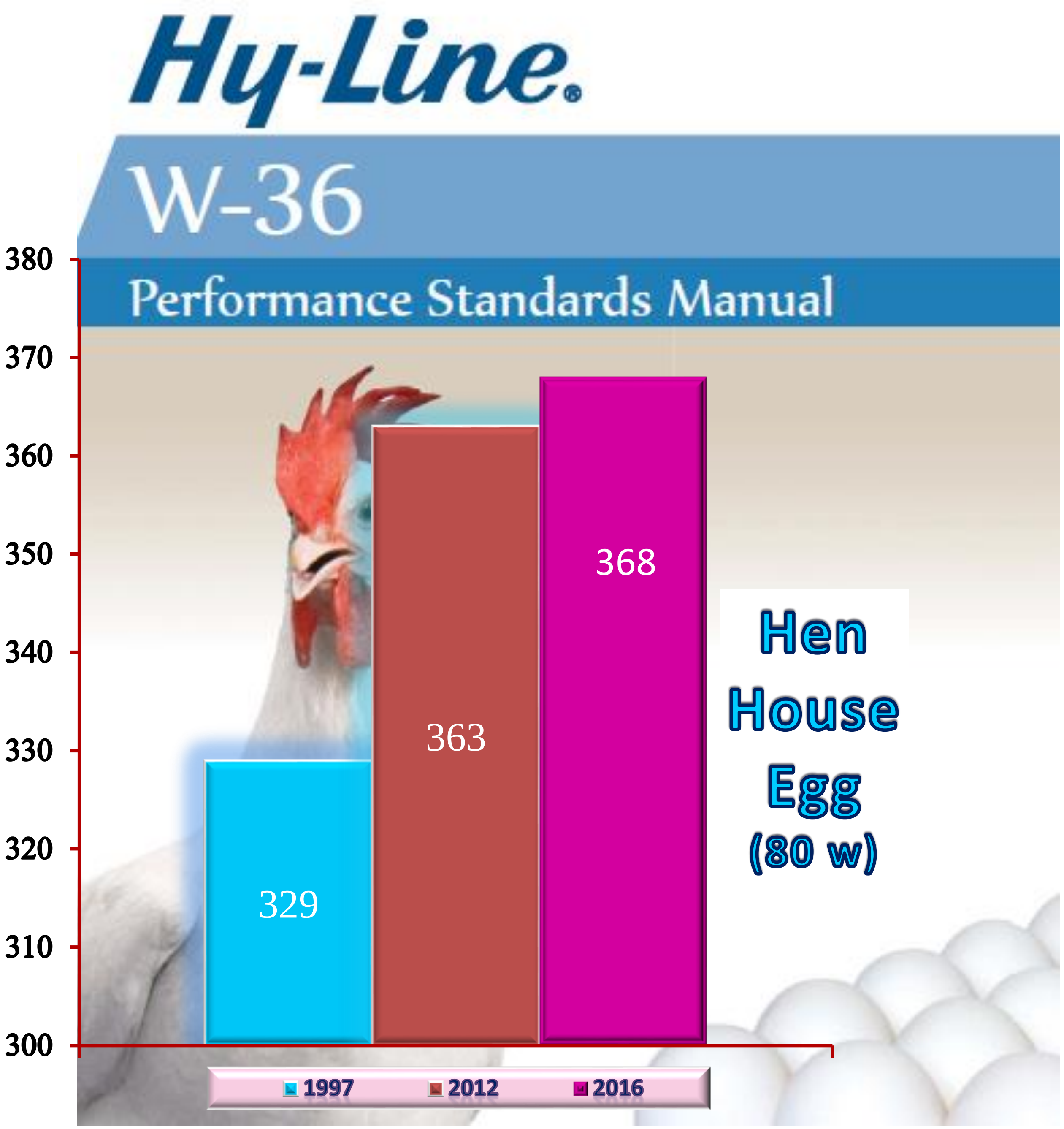
پروفسور مجتـــبی زاغری

اولین همایش ملی
ناهنجاری های متابولیکی
دام و طیور

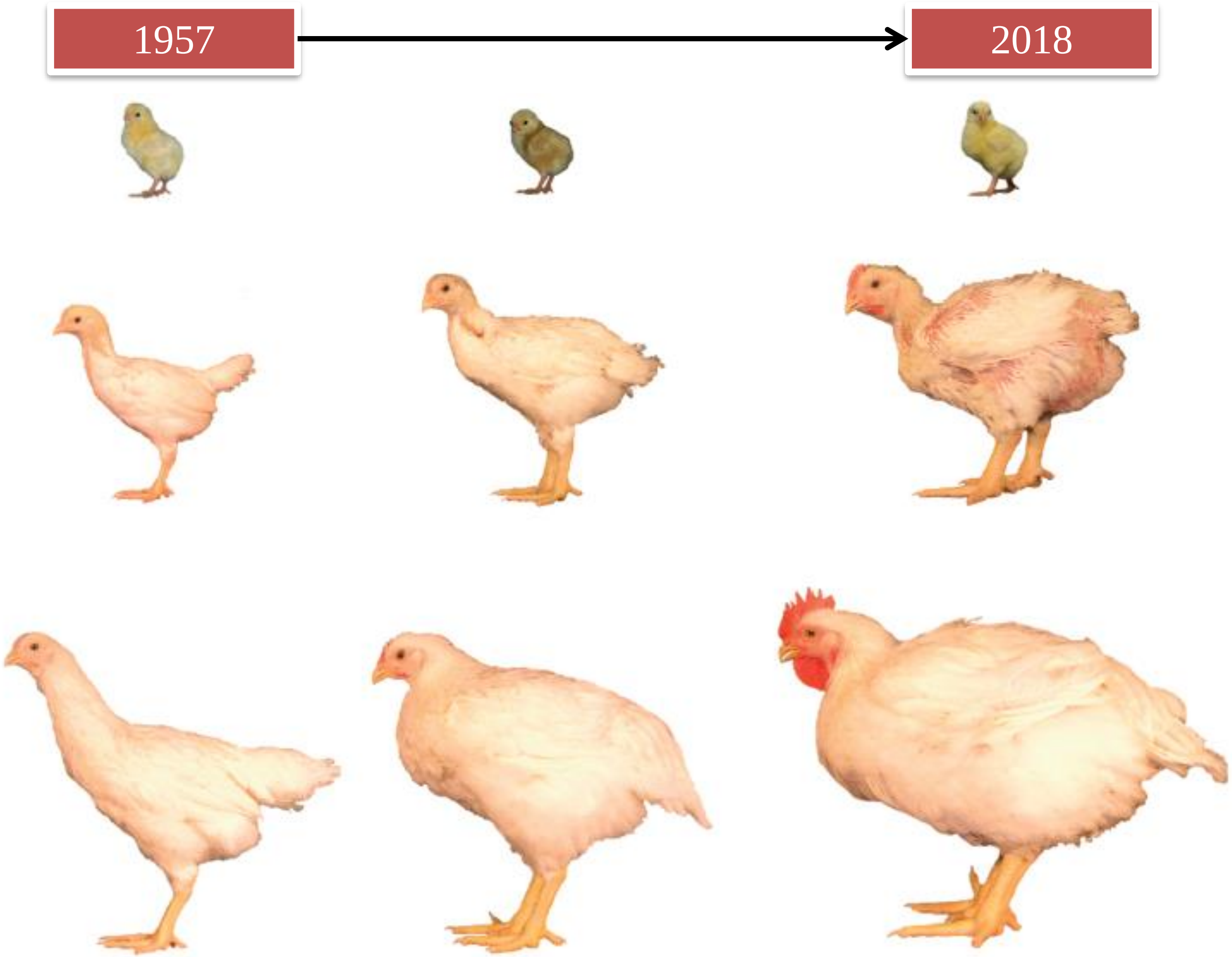
مرکز همایش های بین المللی دانشگاه زنجان / پنج شنبه ـ ۲۲ آذر ماه ۱۳۹۷

مقدمه

Metabolic disorders, may be classed as illness associated with a failure in one of the body hormone or enzyme systems, storage disease related to lack of metabolism of secretory products because of the lack of production of a specific enzyme, or the failure or reduced activity of some metabolic function.



Hy-Line
W-36
Performance Standards Manual
380
370
360
350
340
330
320
310
300
329
363
368
Hen
House
Egg
(80 w)
1997
2012
2016


1957
2018

سویه ژنتیکی	رژیم غذایی	وزن بدن در ۴۲ روزگی (گرم به ازای هر پرنده)	ضریب تبدیل خوراک	درصد سینه	درصد قلب	درصد ریه‌ها
۲۰۰۱	۲۰۰۱	۲۶۷۲	۱/۶۳	۲۰	۰/۴۹۶	۰/۵۲۷
۲۰۰۱	۱۹۵۷	۲۱۲۶	۱/۹۲	۱۷/۴	۰/۵۰۶	۰/۵۶۸
۱۹۵۷	۲۰۰۱	۵۷۸	۲/۱۴	۱۱/۵	۰/۵۷۴	۰/۵۸۹
۱۹۵۷	۱۹۵۷	۵۳۹	۲/۳۴	۱۱/۵	۰/۵۵۳	۰/۵۷۹
خطای معیار		۱۸	۰/۰۳	۰/۳۶	۰/۰۳	۰/۰۴
سطح احتمال						
اثر ژنتیک		۰/۰۰۰۱	۰/۰۰۰۱	۰/۰۰۰۱	۰/۰۰۵	۰/۰۳۵
اثر تغذیه		۰/۰۰۰۱	۰/۰۰۰۱	۰/۰۰۰۱	۰/۸۰۱	۰/۳۸
اثر متقابل		۰/۰۰۰۱	۰/۰۴۵	۰/۰۰۰۱	۰/۴۷۴	۰/۱۴۹

The primary function of the respiratory system

- ❑ Gas exchange
- ❑ Maintaining a constant body temperature in birds
- ❑ Phonation

Respiratory Tract

Parameter			Amount
Air sac gases			(mmHg)
Co_2 mean partial pressure	Abdominal		15
	Clavicular		44
	Thoracic		
	Caudal		24
	Cranial		42
O_2 mean partial pressure	Abdominal		130
	Clavicular		84
	Thoracic		
	Caudal		102
	Cranial		99
Exchenge surface area			14 cm^2/g/body weight
Expaired gases			(mmHg)
Co_2 partial pressure			28
O_2 partial pressure			108
Oxygen uptake			24-26 (ml/kg/min)
Pulmonary ventilation rate (V_E)			0.5-0.7 (l/min)
Respiratory frequency (f_R)		♂	12-21 (breaths/min)
		♀	20-37 (breaths/min)
Volume of respiratory tract			(ml)
Abdominal sacs, paired		♂	180
		♀	110
Clavicular sac		♂	95
		♀	55
Cervical sacs		♂	30
		♀	20
Lungs, paired		♂	70
		♀	35
Thoracic caudal sacs, paired		♂	30
		♀	24
Thoracic cranial sacs, paired		♂	90
		♀	50
Total		♂	500
		♀	300

غلظت اجزای تشکیل‌دهنده خون در پرندگان و پستانداران

متغیر / نوع حیوان	مرغ	گاو	گوسفند	اسب
گویچه‌های قرمز ($\times 10^{6}$)	۳	۷	۱۲	۹
هماتوکریت (٪)	۳۰	۳۵	۳۵	۴۱
هموگلوبین (meq/L)	۹	۱۱	۱۱/۵	۱۴/۴
اسیداوریک (meq/L)	۱–۷	۰/۱–۲	۰/۱–۲	۰/۵–۱
اسیدلاکتیک (meq/L)	۴۷–۹۸	۵–۲۰	۹–۱۲	۱۰–۱۶
کلسترول (meq/L)	۱۲۵–۲۰۰	۸۰–۱۸۰	۶۰–۱۵۰	۷۵–۱۵۰
سدیم (meq/L)	۱۵۱–۱۶۱	۱۳۲–۱۵۲	۱۳۹–۱۵۲	۱۳۲–۱۵۲
پتاسیم (meq/L)	۴/۶–۴/۷	۳/۹–۵/۸	۳/۹–۵/۴	۲/۵–۵
کلر (meq/L)	۱۱۹–۱۳۰	۹۷–۱۱۱	۹۵–۱۰۵	۹۹–۱۰۹

- **تعداد گلبول قرمز در طیور کمتر است.**
- **میل ترکیب اکسیژن با گلبول قرمز طیور کمتر است.**
- **طول عمر اریتروسیت ها ۳۰ تا ۴۰ روز است.**
- **اریتروسیت های مرغ دارای هسته هستند.**
- **اریتروسیت های انعطاف پذیری اندکی دارند.**

Anatomy of the avian respiratory system

i. Small lungs which do not change volume during breathing.

ii. Birds total volume of respiratory system is larger than mammals (15 *vs* 7% of body volume).

iii. Avian lung itself is smaller (1 to 3% of body volume) than mammals.

iv. In contrast to mammals (subatmospheric pressure), the avian thoracic cavity is essentially atmospheric pressure.

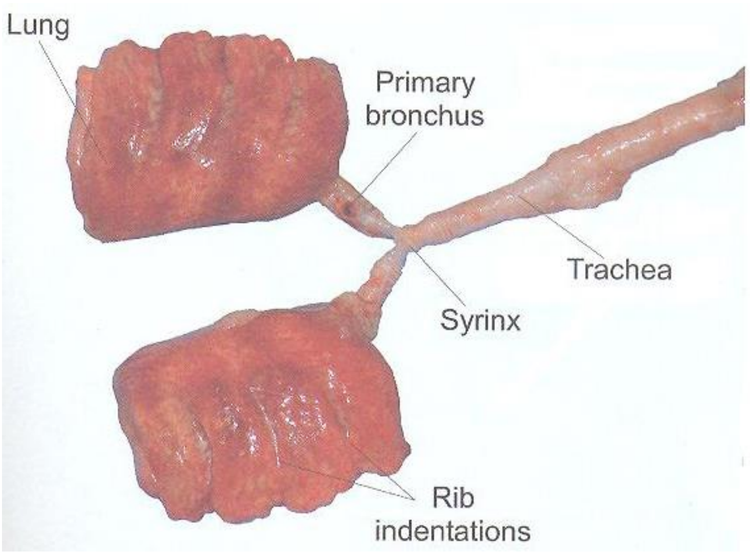
v. In contrast to mammals, the avian trachea is 2.7 (times) longer and 1.3 wider, so the dead air volume is more (4 to 4.5).

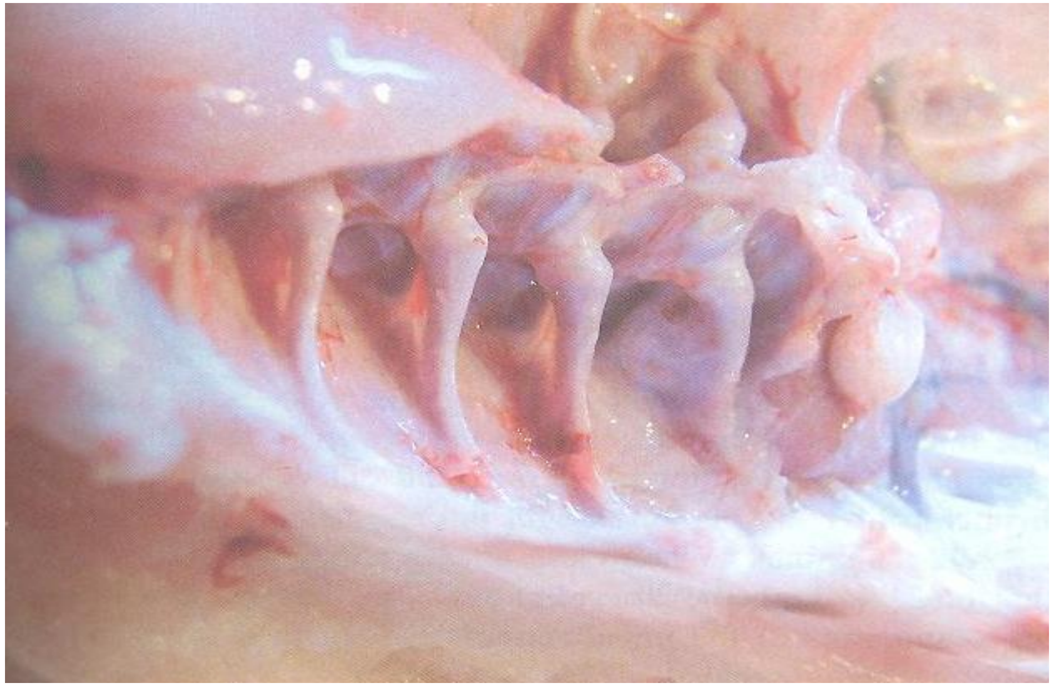
vi. There is no diaphragm.

vii. The air sacs act as bellows, drawing in air through the lungs and expelling the stale air.

viii. The airflow through the lungs is one way, there are no blind-ended sacs as in mammals so consequently there is no ebb and flow of air through the lungs, it is continuous.

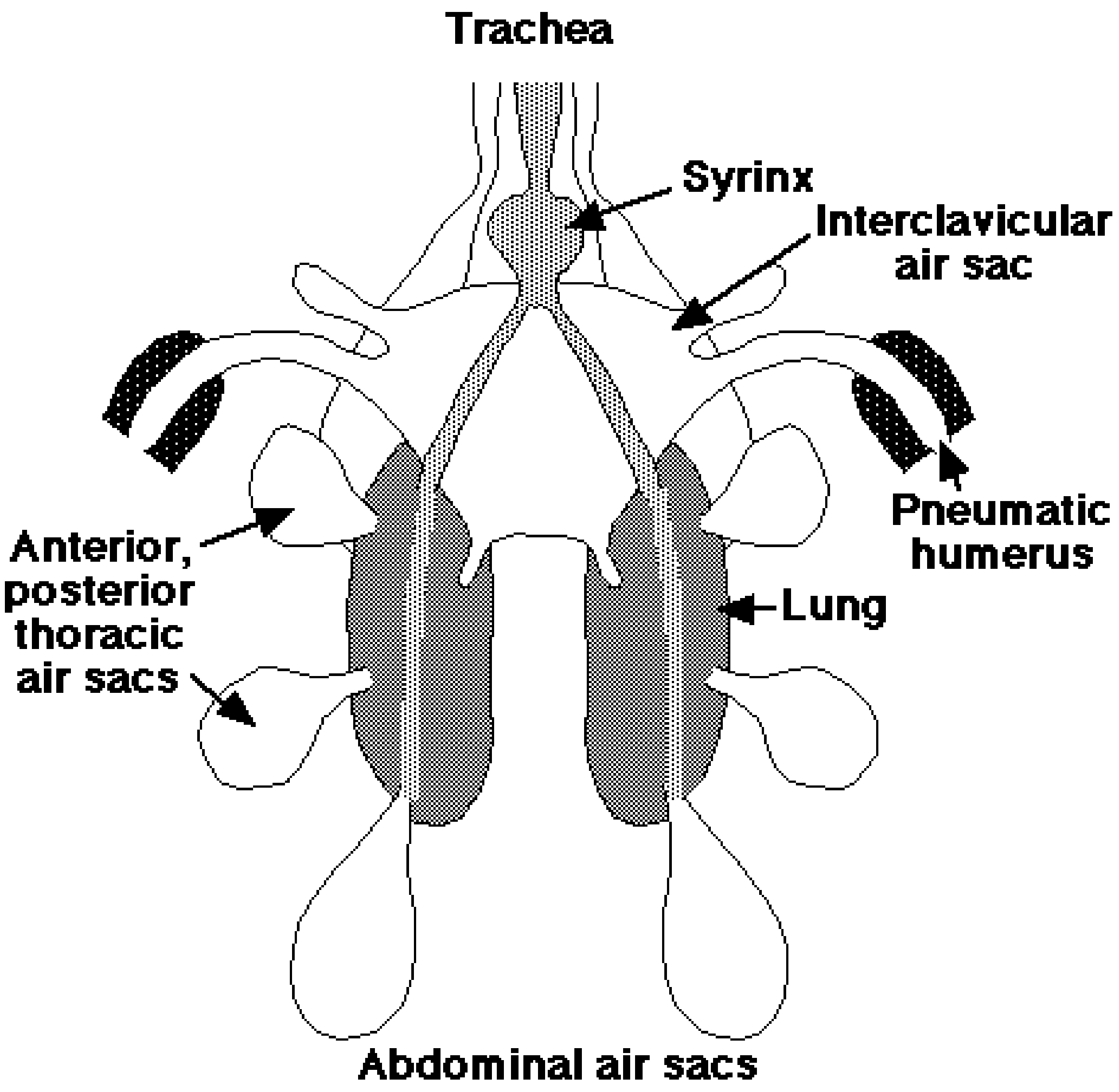
ix. In avian, 25 % of lungs volume occupied by ribs

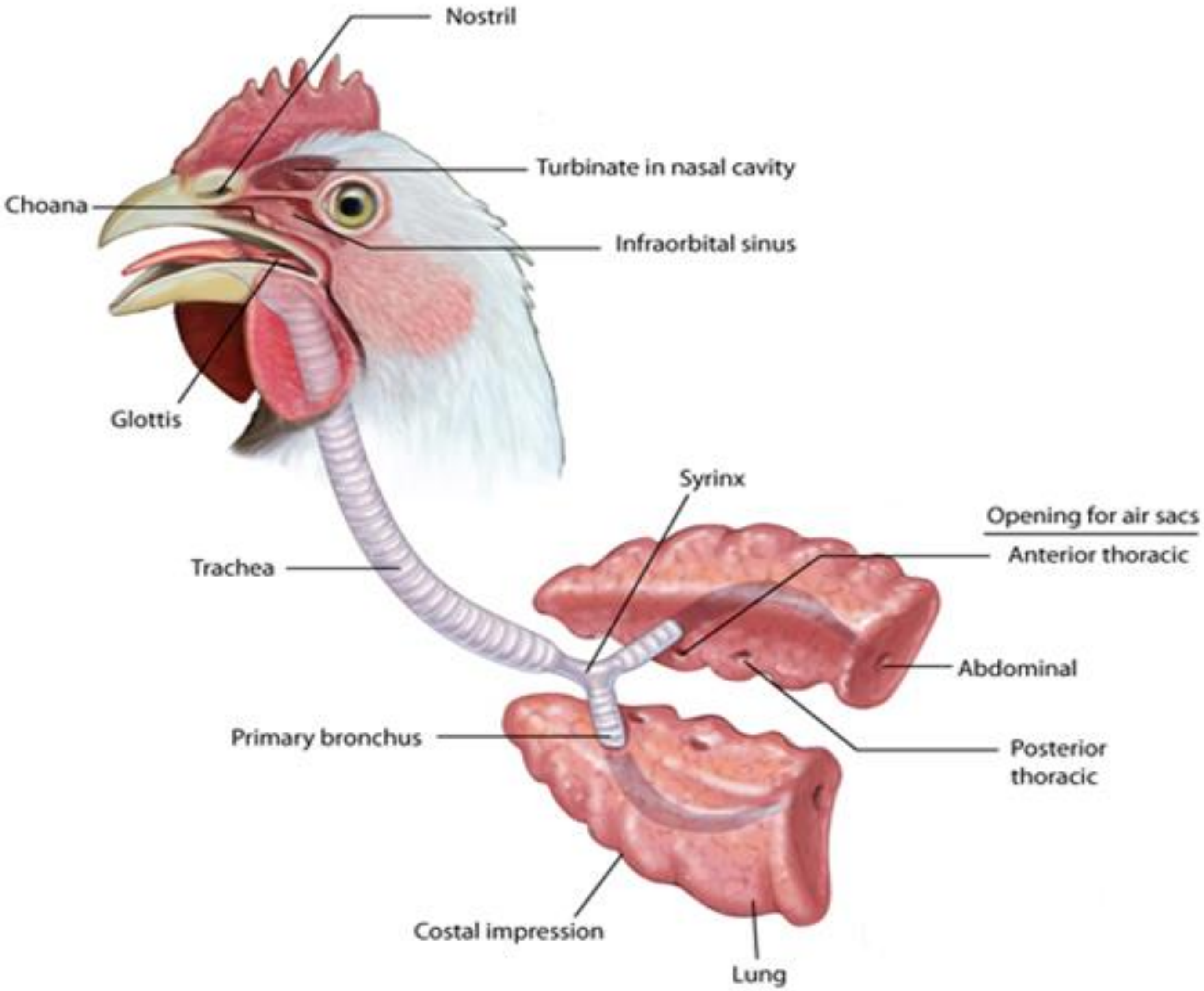

Lung
Primary bronchus
Trachea
Syrinx
Rib indentations



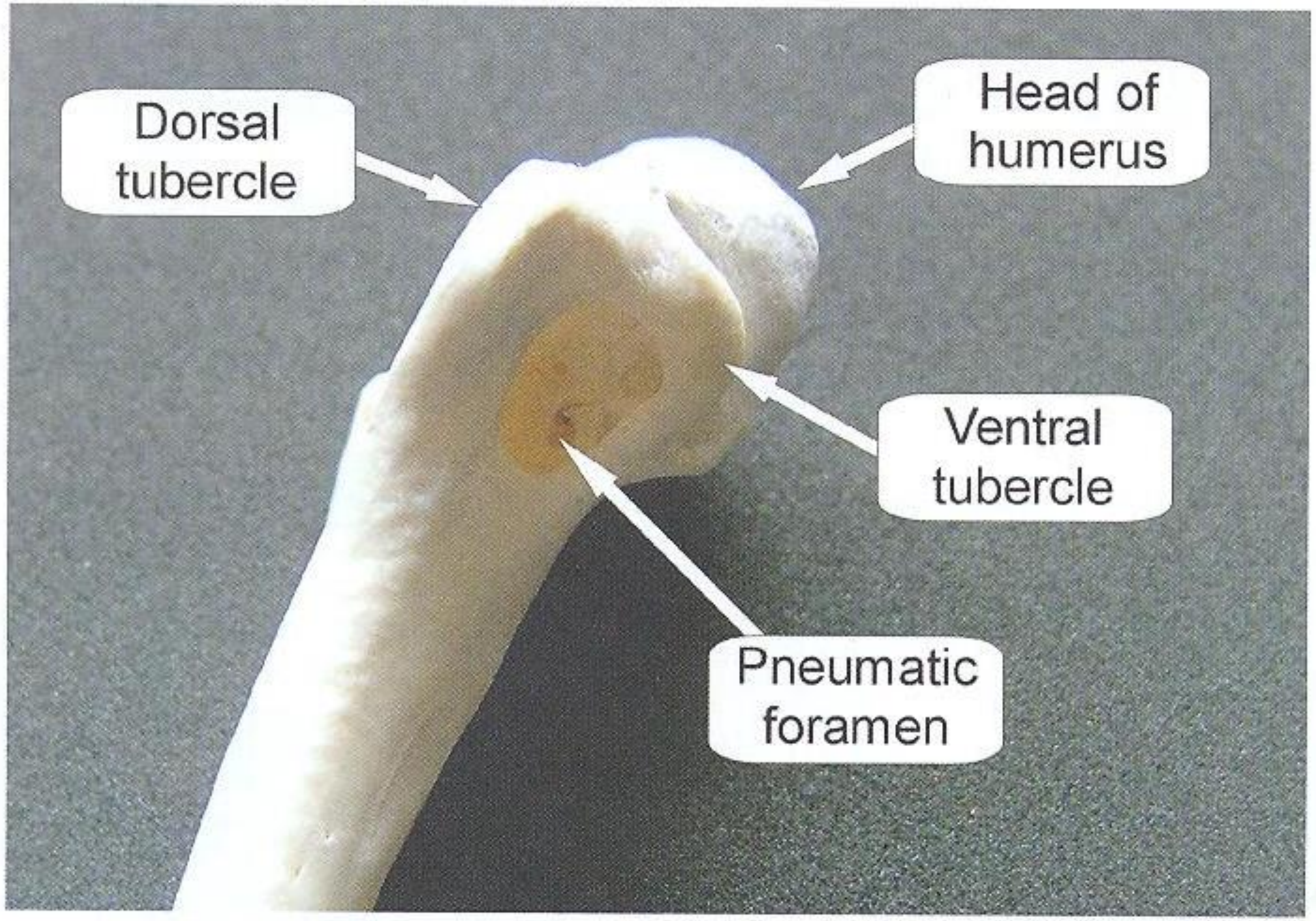
Most birds have 9 air sacs:

One interclavicular sac
two cervical sacs
two anterior thoracic sacs
two posterior thoracic sacs
two abdominal sacs

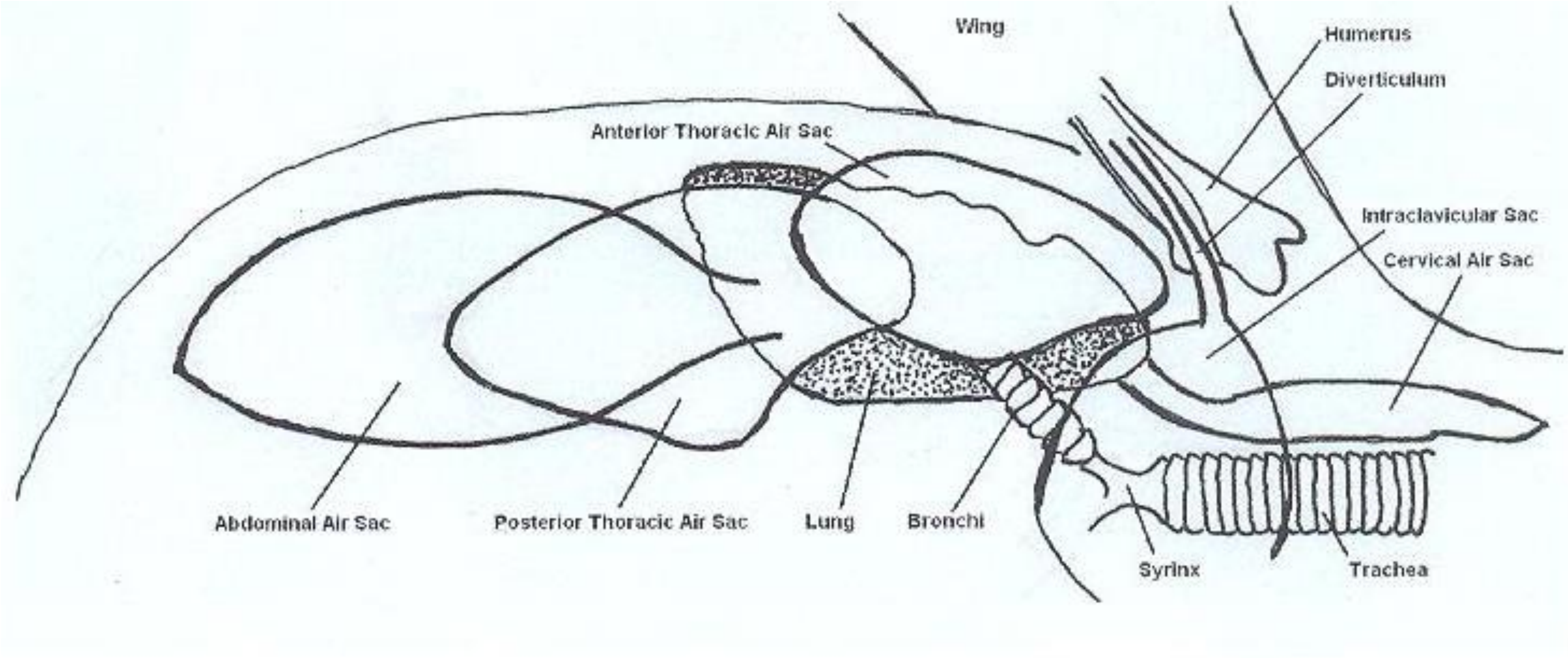




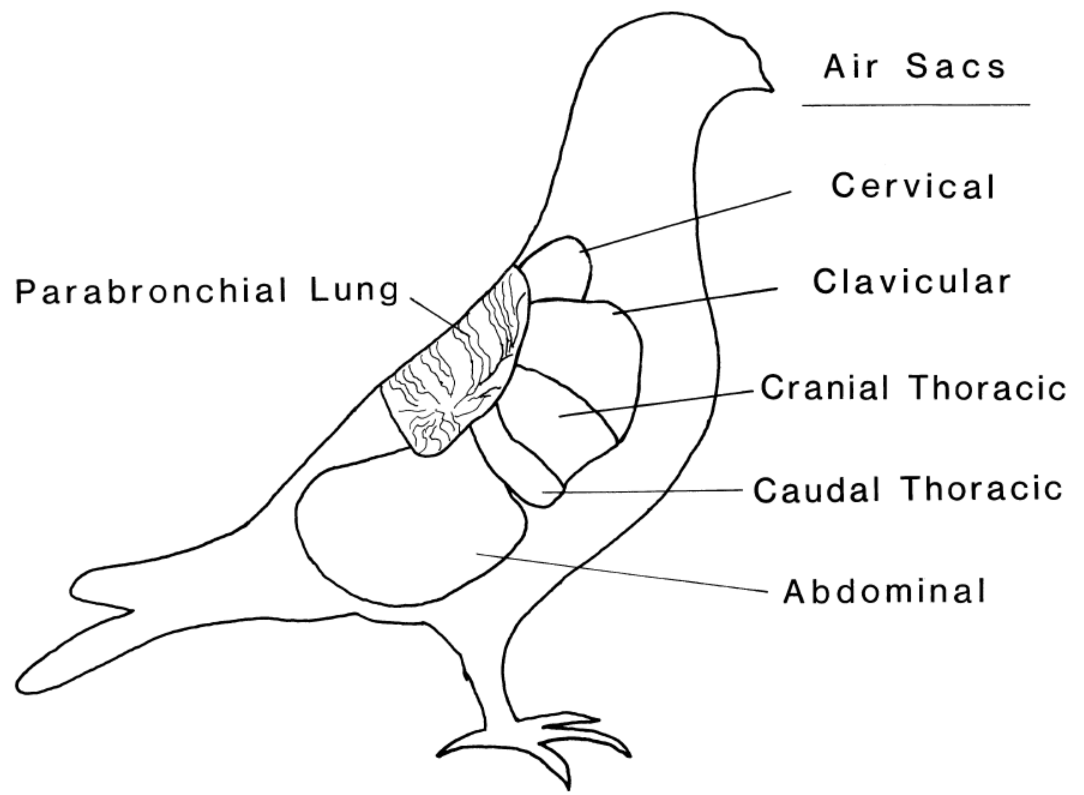
Nostril
Turbinate in nasal cavity
Choana
Infraorbital sinus
Glottis
Syrinx
Opening for air sacs
Anterior thoracic
Trachea
Abdominal
Primary bronchus
Posterior thoracic
Costal impression
Lung



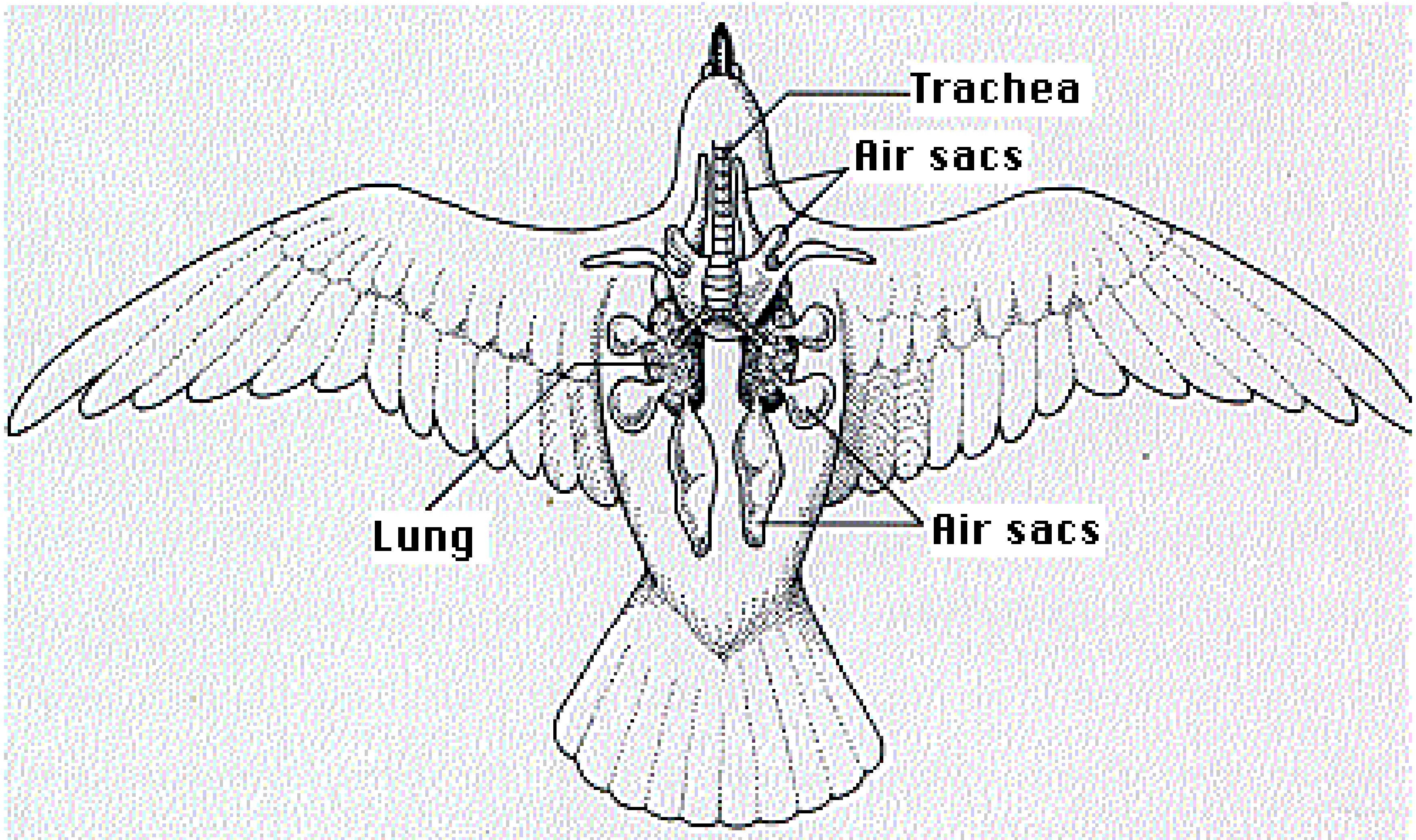
The head of the humerus – detailing the pneumatic foramen that channels the diverticulum of the intraclavicular airsac

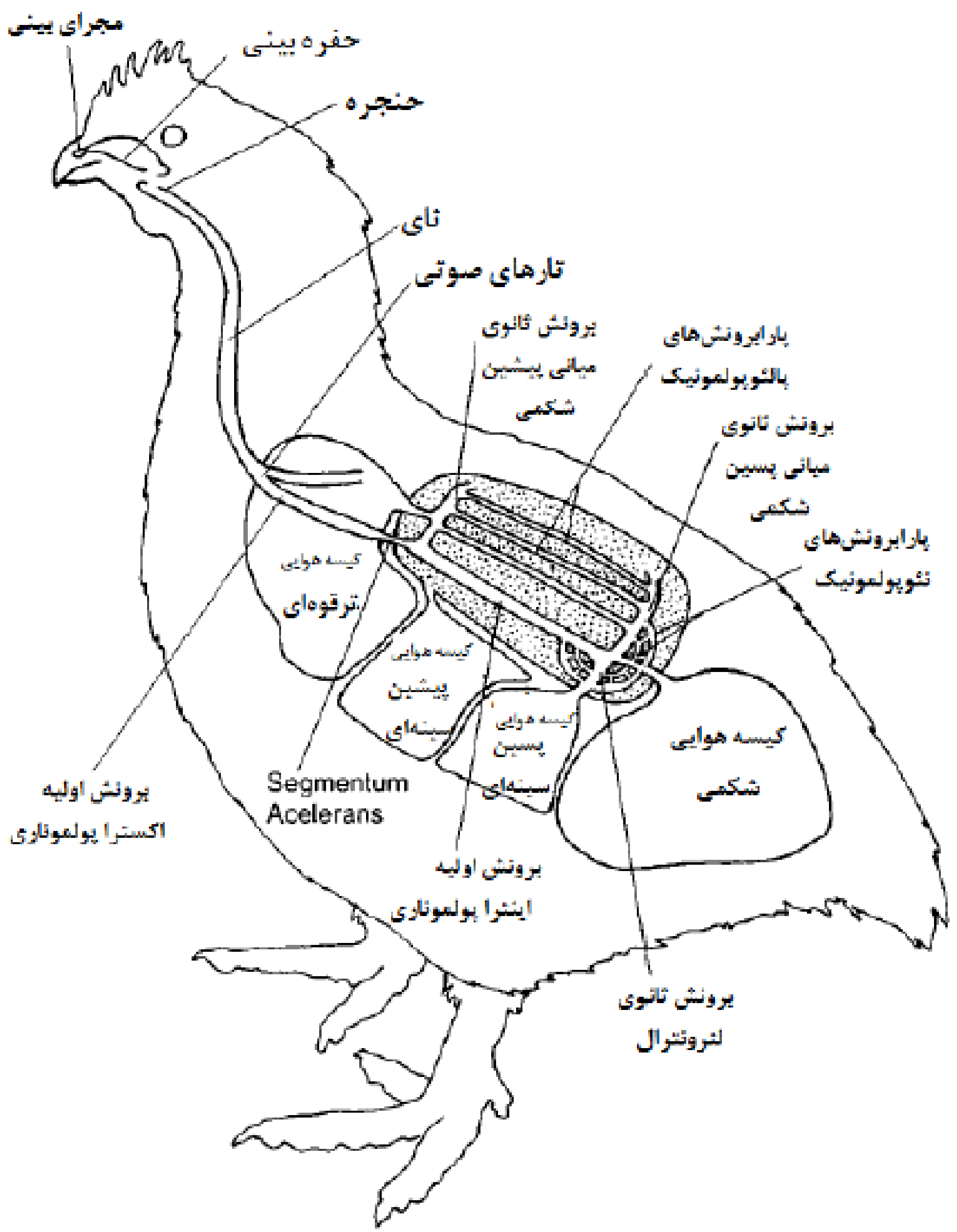


Sketch of the position of the airsacs



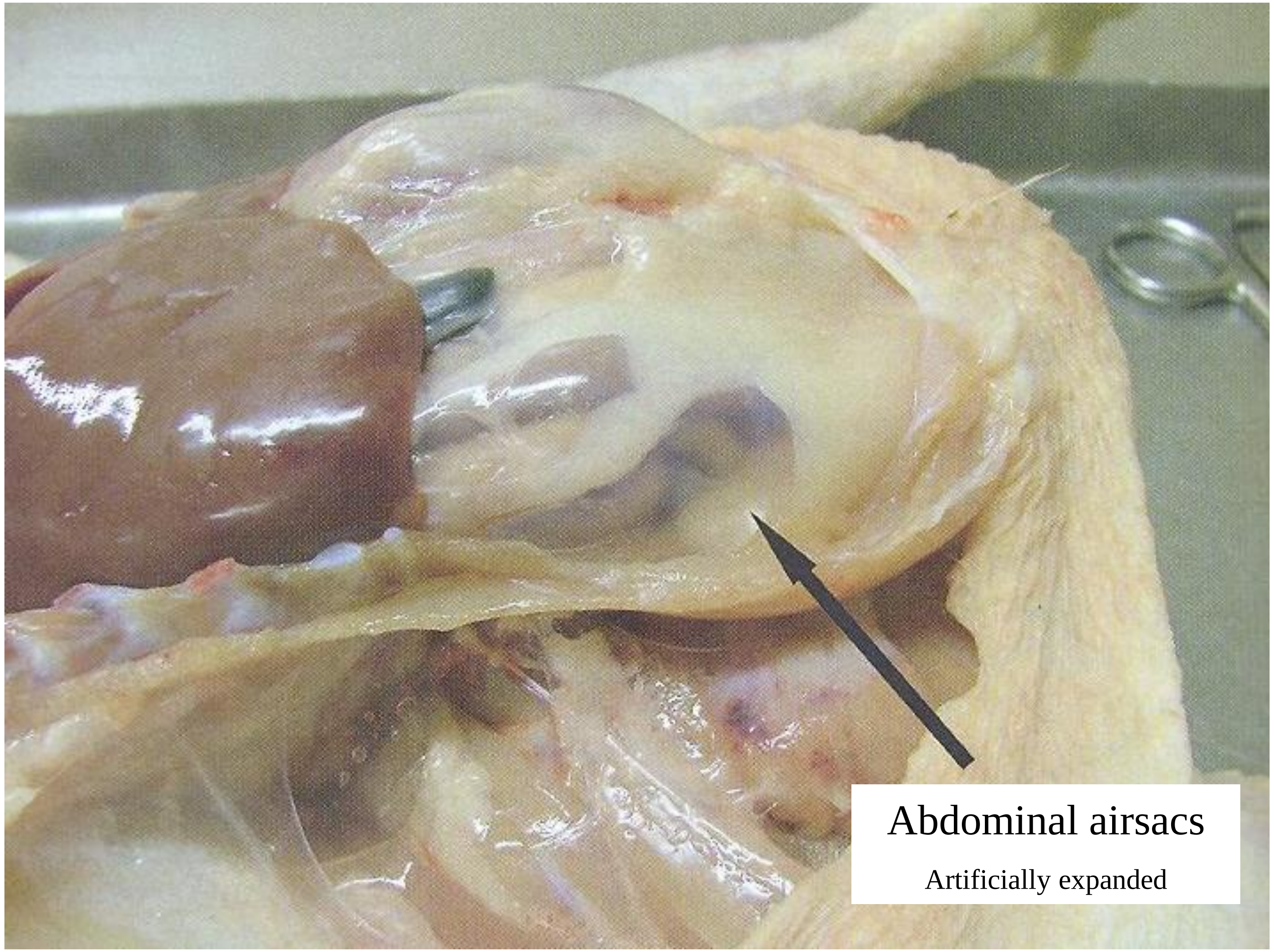
Respiratory system of a pigeon consisting of the parabronchial lung and air sacs.


Trachea
Air sacs
Lung
Air sacs


مجرای بینی
حفره بینی
حنجره
نای
تارهای صوتی
برونش ثانوی میانی پیشین شکمی
پارابرونش‌های پالئوپولمونیک
برونش ثانوی میانی پسین شکمی
پارابرونش‌های نئوپولمونیک
کیسه هوایی ترقوه‌ای
کیسه هوایی پیشین سینه‌ای
کیسه هوایی پسین سینه‌ای
کیسه هوایی شکمی
Segmentum Acelerans
برونش اولیه اکسترا پولموناری
برونش اولیه اینترا پولموناری
برونش ثانوی لتروئترال

کیسه‌های هوایی و ظرفیت حجمی آنها در یک مرغ گوشتی به وزن ۲/۹ کیلوگرم

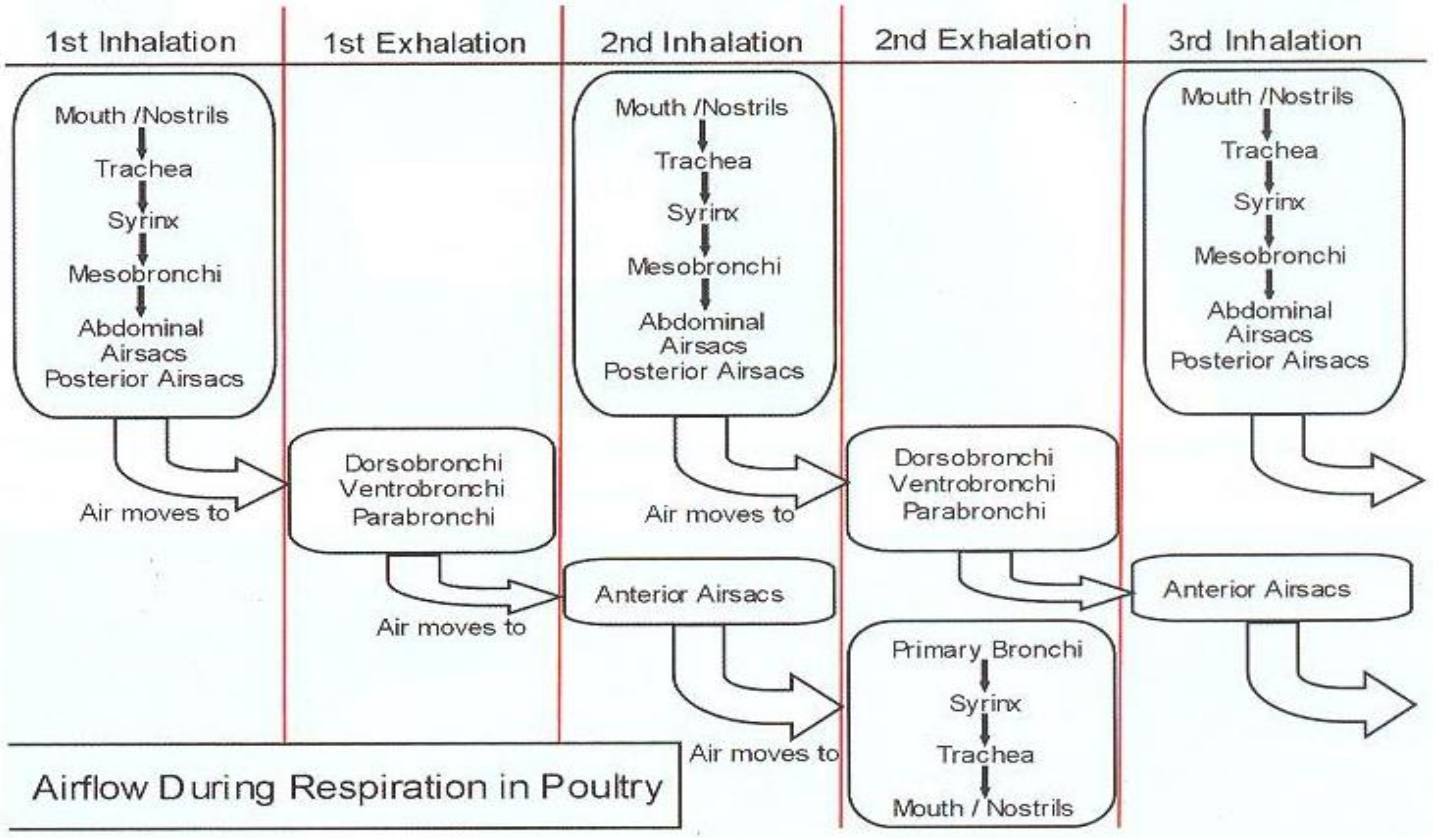
نام کیسه هوایی	ظرفیت حجمی (میلی لیتر)
ترقوه‌ای (clavicular)، یک عدد	۵۵
گردنی (cervical)، دو عدد	۲۰
سینه‌ای پیشین (cranial thoracic)، دو عدد	۵۰
سینه‌ای پسین (caudal thoracic)، دو عدد	۲۴
شکمی (abdominal)، دو عدد	۱۱۰

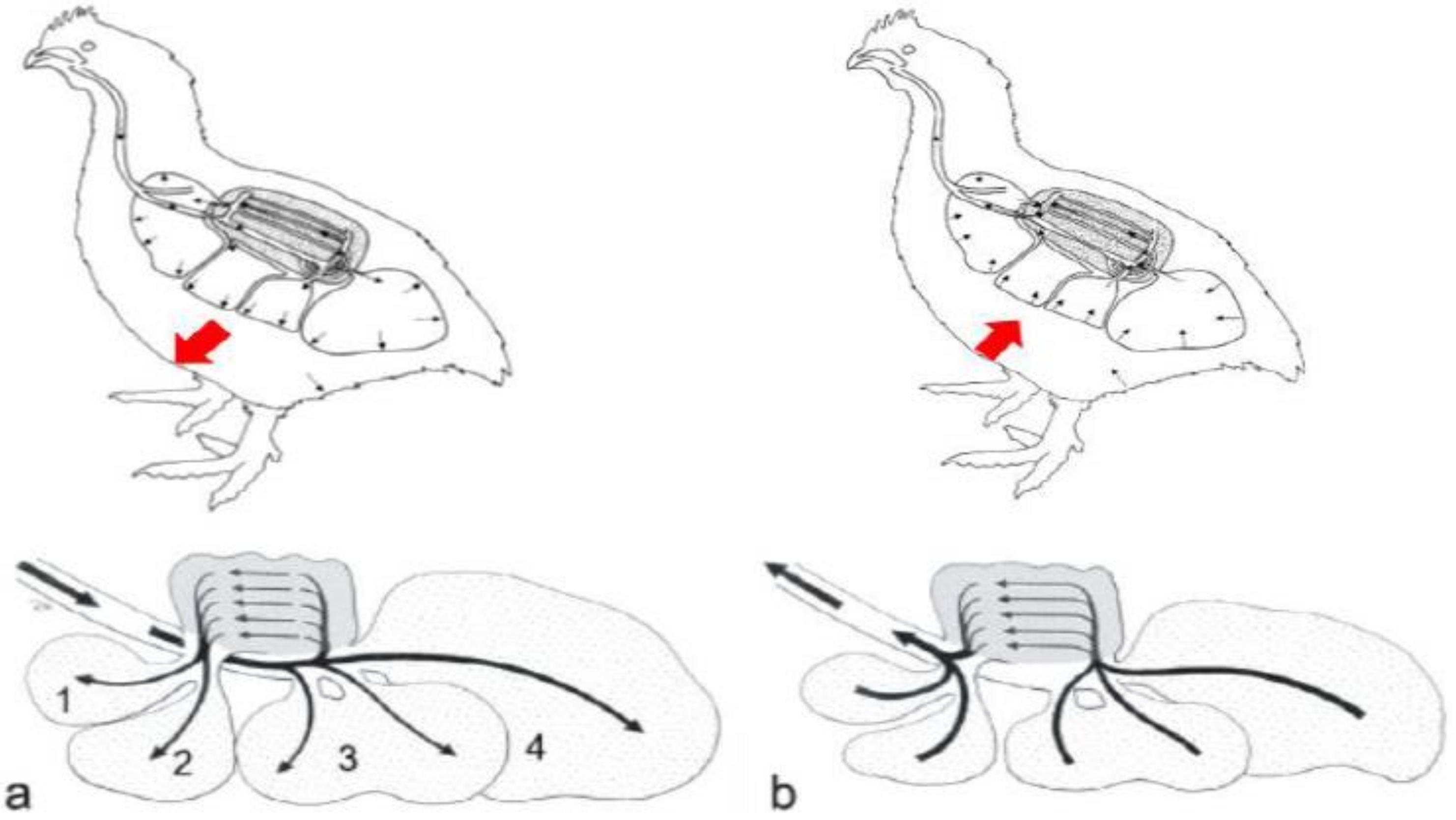

Abdominal airsacs
Artificially expanded

The breathing cycle

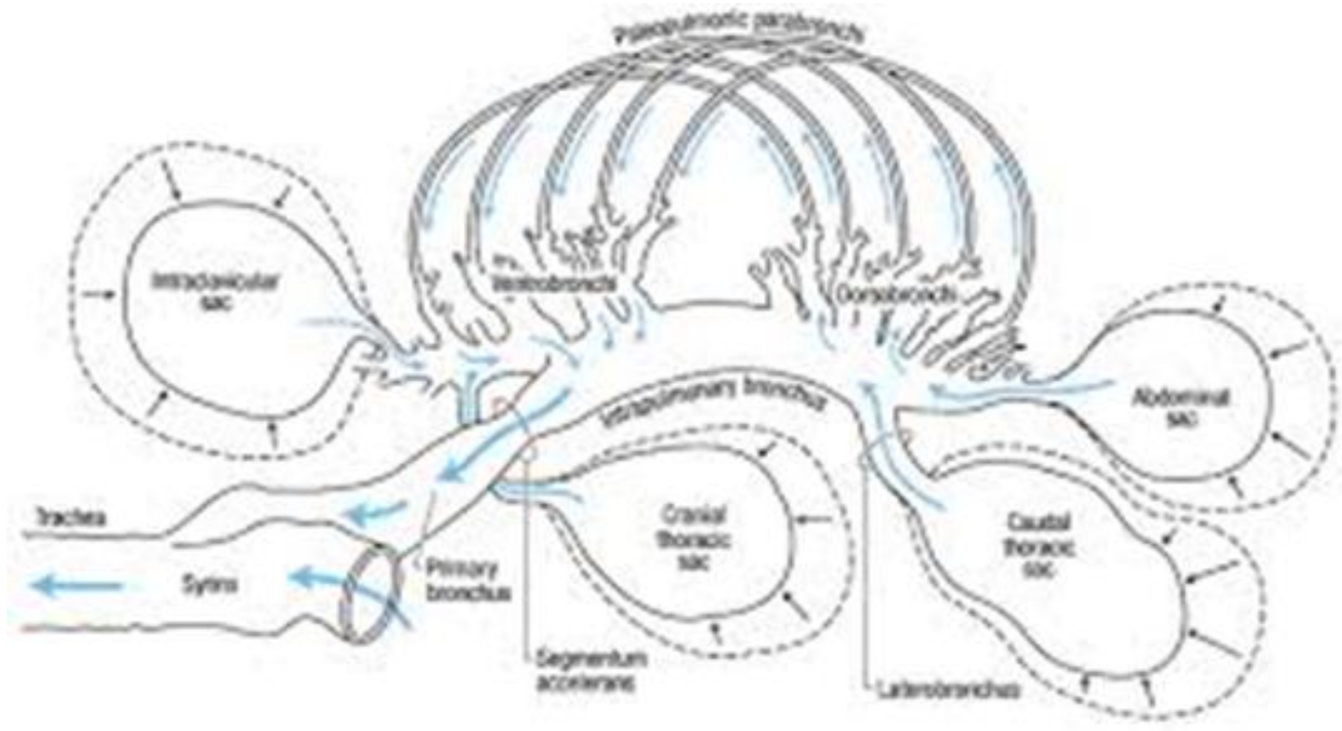
The breathing cycle is basically:

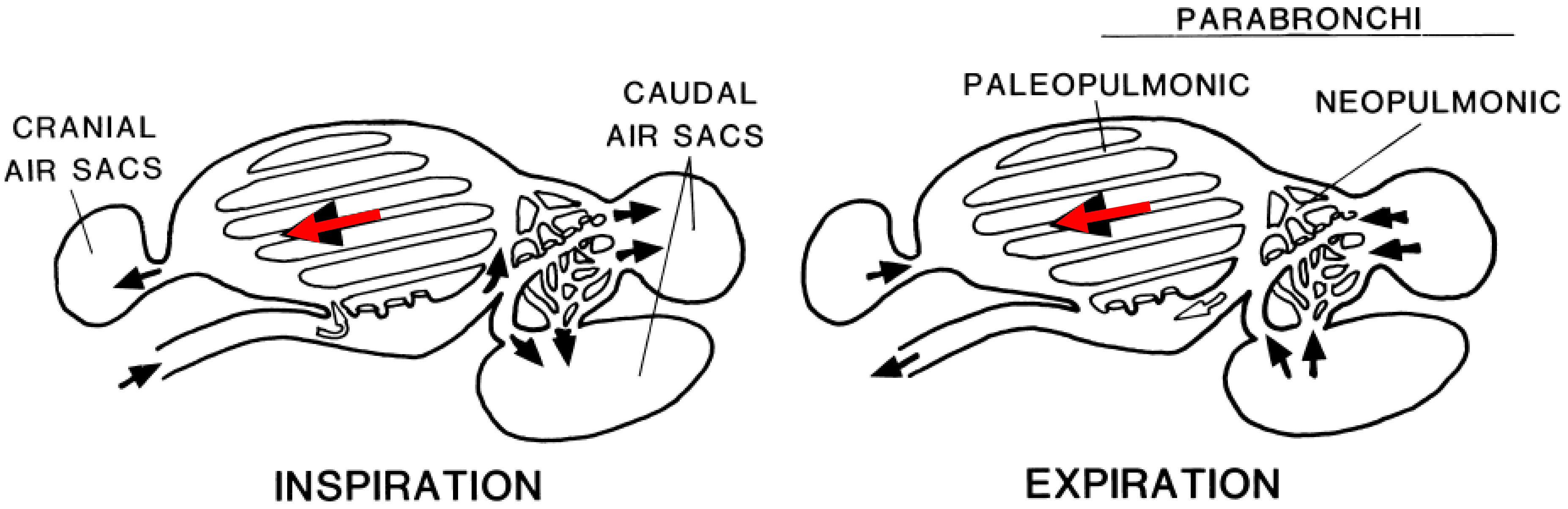
First inhalation	-	Abdominal expansion draws air through to the rear air sacs.
First exhalation	-	Abdomen contracts forcing air through the lungs.
Second inhalation	-	Abdomen expands again, forcing air in lungs into forward airsacs.
Second exhalation	-	Abdomen contracts driving stale air out of trachea.





الگوی جریان هوا در پارابرونش‌های پالئوپولمونیک در ریه پرندگان هنگام دم (a) و بازدم (b)


Paleopulmonic parabronchi
Interclavicular sac
Ventrobronchi
Dorsobronchi
Abdominal sac
Intrapulmonary bronchus
Trachea
Syrinx
Primary bronchus
Cranial thoracic sac
Caudal thoracic sac
Segmentum accelerans
Laterobronchus



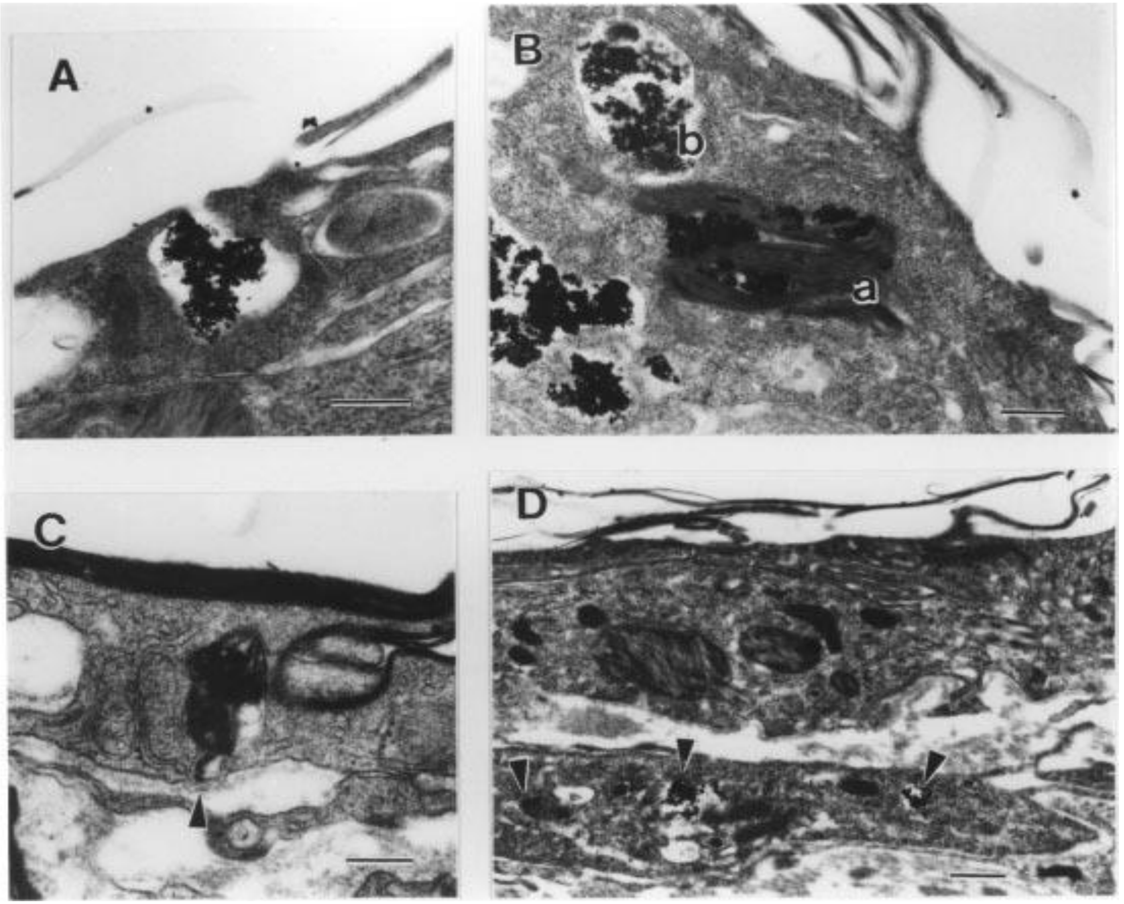
Pathway of airflow in the avian respiratory system during inspiration and expiration. Flow in paleopulmonic parabronchi is always caudal-to-cranial during both phases of breathing (large solid arrows) but neopulmonic flow is bidirectional. Open arrows show possible ventilatory shunts.

Nasal cavity is well designed to

- Heat
- Humidify
- Filter

the inspired air

The impact of poultry house pollutants on particulate clearance from the respiratory system of birds remains largely unknown. However, any substance that reduces ciliary motility or disrupts the ciliated epithelium could be expected to adversely affect the resistance of birds to microorganisms that normally enter their bodies via the respiratory system.



These observations may explain why,

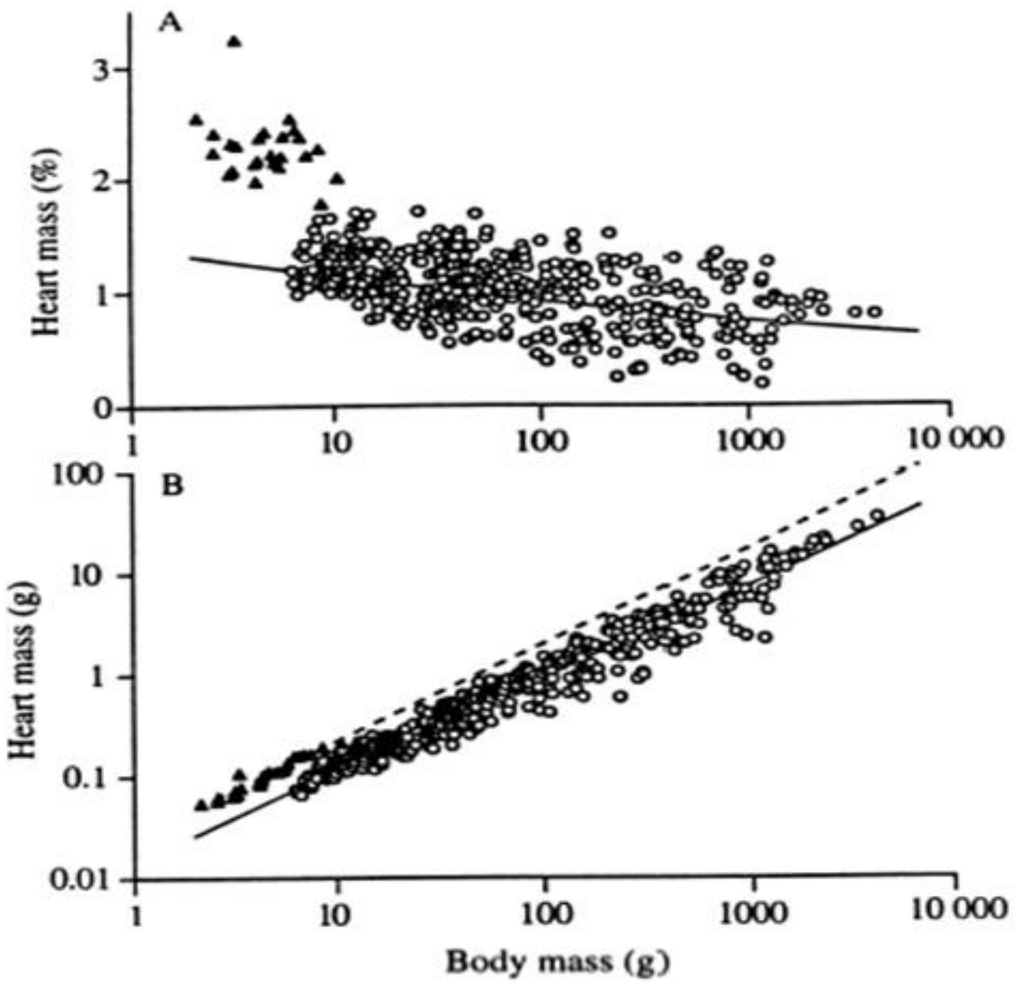
- parabronchial macrophages are not usually seen in the avian lung
- caudal group of air sacs are those most prone to infections while the cranial group of air sacs are less often affected

Brief Anatomy of the Avian Heart

Heart and Circulation	
Parameter	**Amount**
Blood pressure	(mmHg)
Bronchial artery	25
Common carotid artery	145
Diastolic ♂	150
♀	130
Hepatic portal vein	5
Hepatic vein	1
Systolic ♂	160-180
♀	130-160
Blood volume	70-90 (ml/kg)
Cardiac output	120-270 (ml/kg/min)
Circulating time	2-8 (s)
Heart rate	250-470 (beats/min)

Brief Anatomy of the Avian Heart

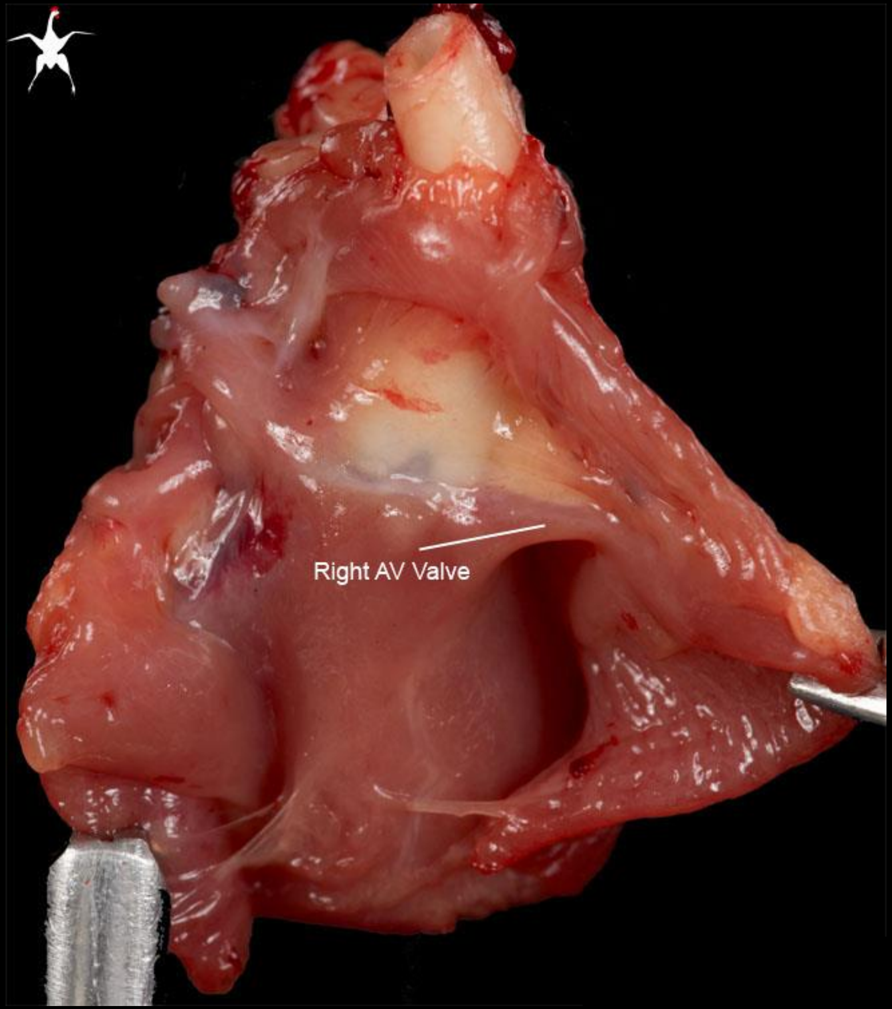
In birds, heart mass scales in respect to body mass as $M_h = 0.014 M_b^{0.91}$ (Bishop and Butler, 1995). In mammals the relationship is $M_h = 0.0058 M_b^{0.98}$ (Prothero, 1979), where M_h is heart mass and M_b body mass.

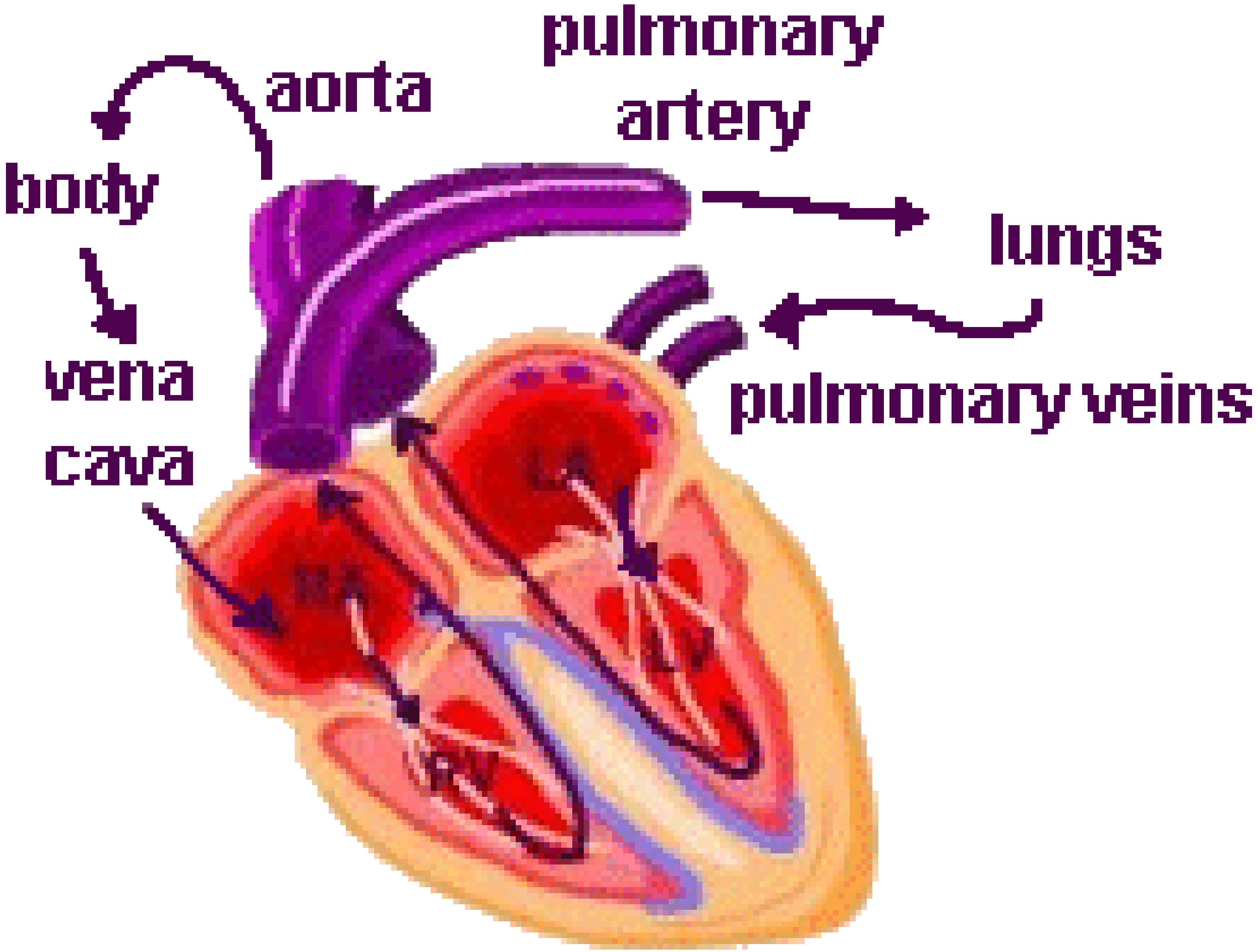


(A) Heart mass as a percentage of body mass. (B) Heart mass in grams, plotted against body mass (g) for 488 avian species, including 25 species of hummingbird. Hummingbird data are represented by the filled triangles and dashed line; all other species are represented by open circles and solid line. (After Physiological modelling of oxygen consumption in birds during flight, C. M. Bishop and P. J. Butler, *J. Exp. Biol.* **198**, 2153–2163, 1995, © Springer-Verlag.)

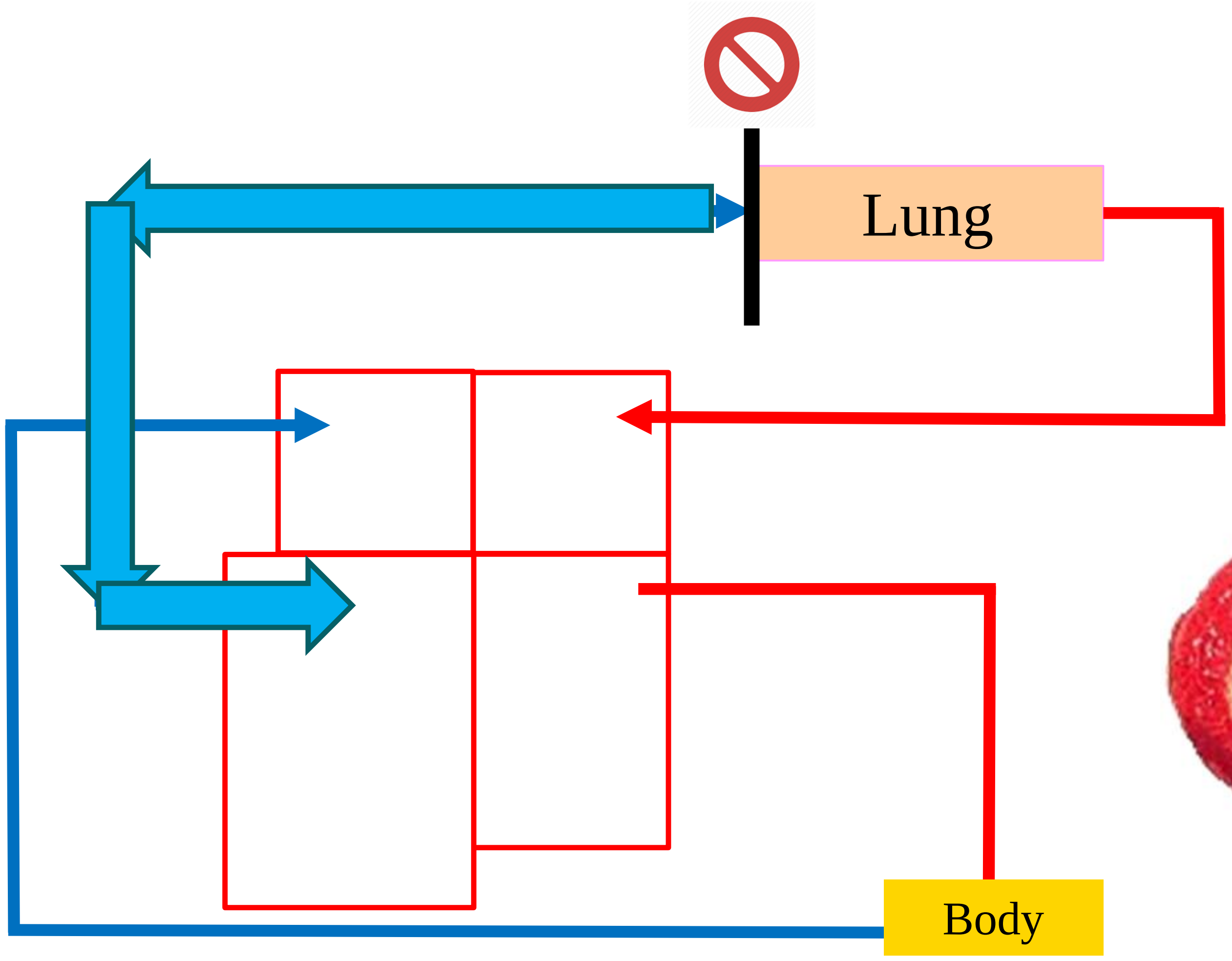
Humming birds whose wings move very quickly

- Avian A-V valve, which is merely a muscular flap unlike the complex structure seen in mammals and so contraction of the heart causes not only increased pulmonary arterial pressure, but also back pressure to the venous system and especially the liver.





aorta
pulmonary artery
body
lungs
vena cava
pulmonary veins


Lung
Body

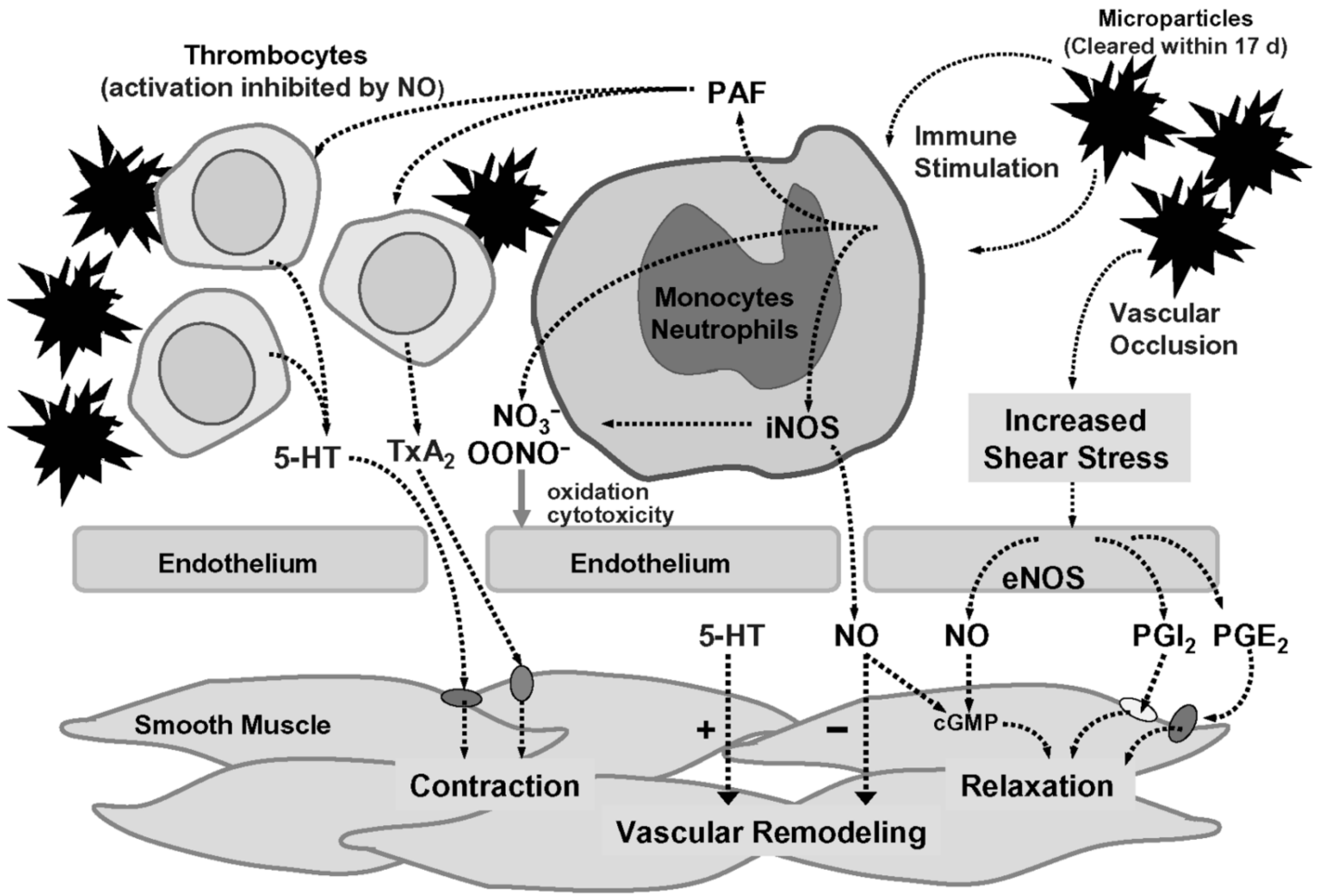
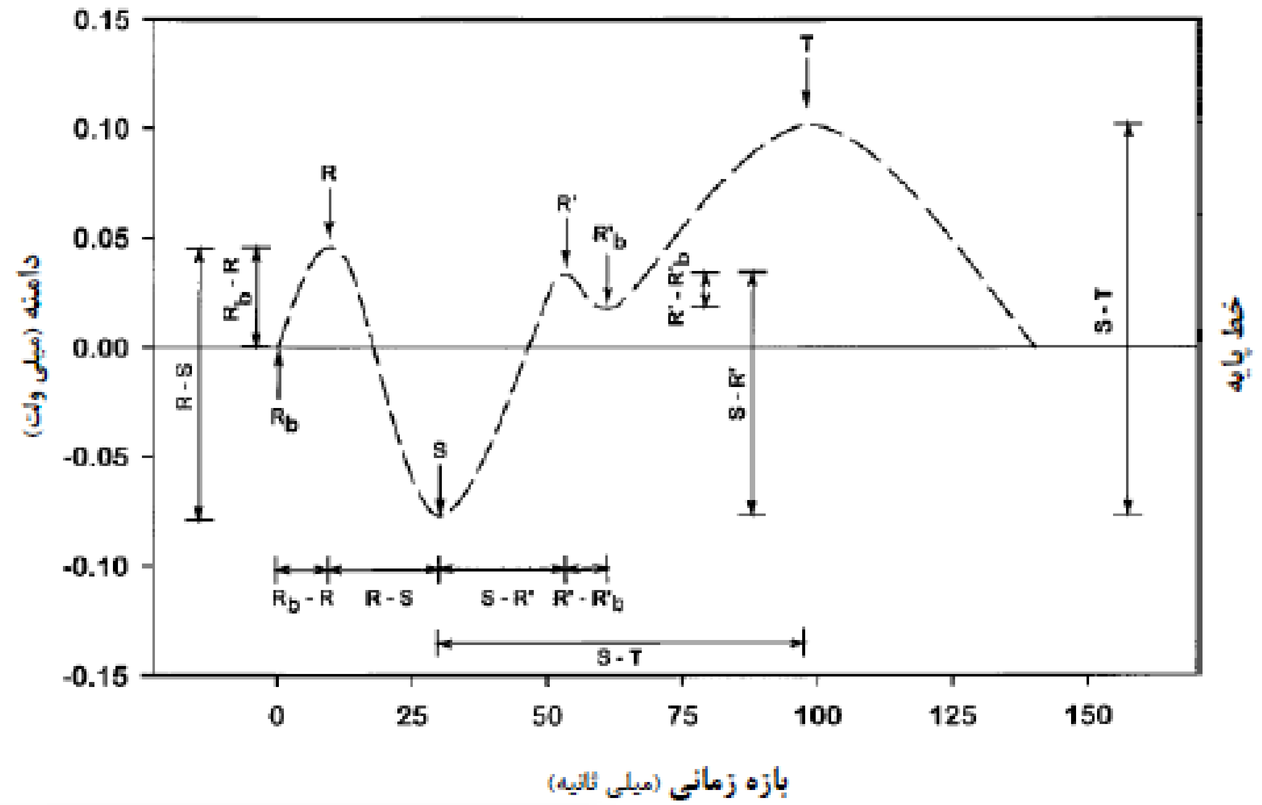
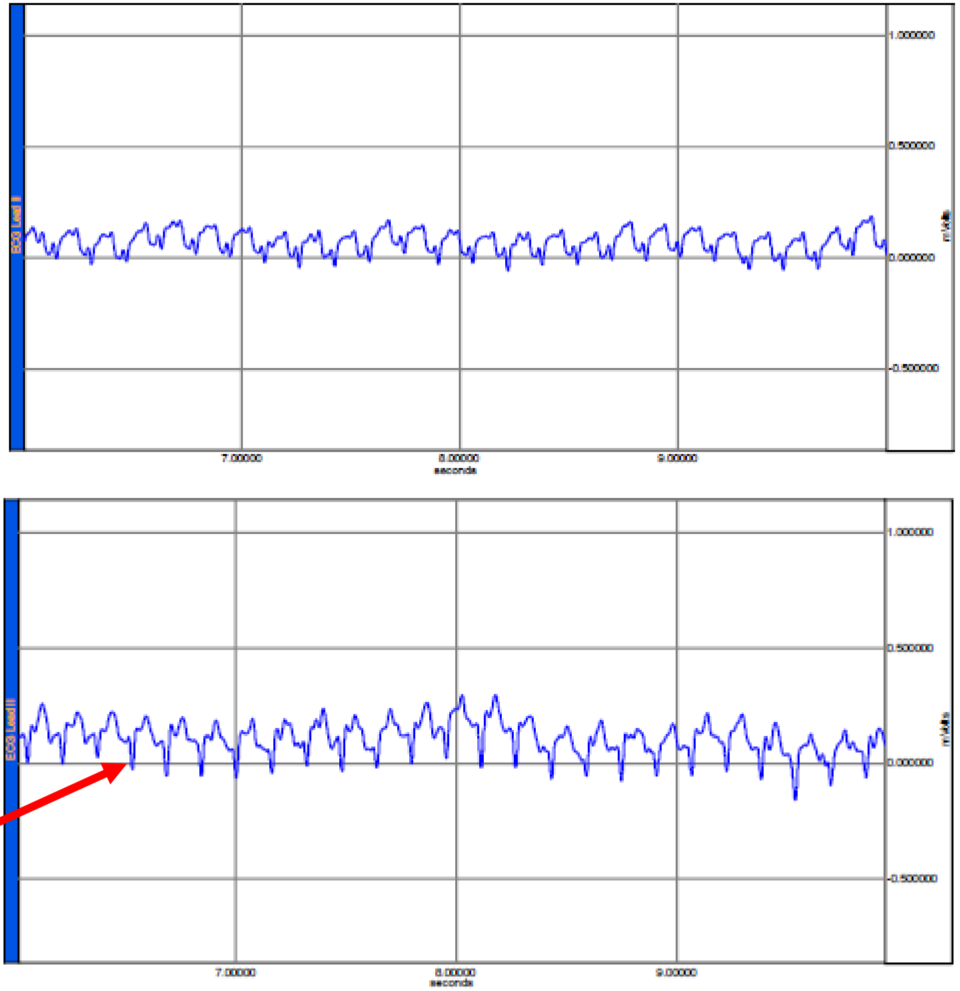
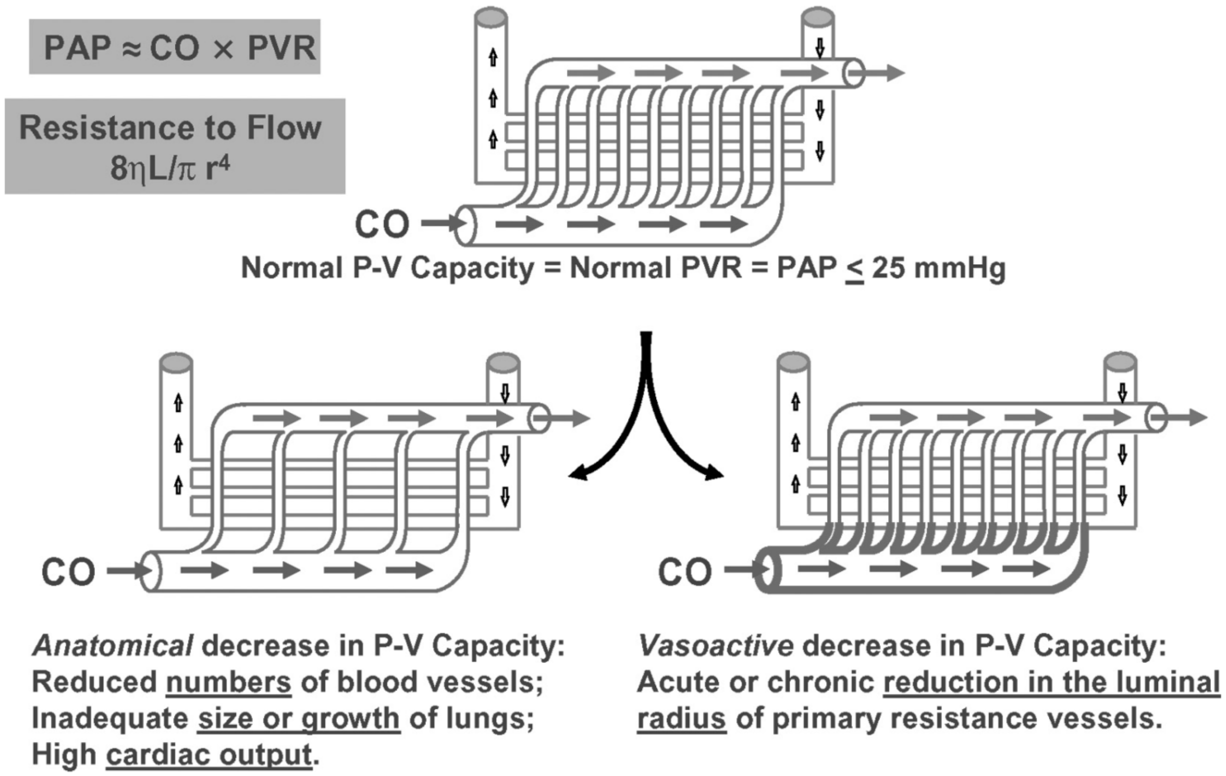



Figure 6. Microparticle occlusion of pulmonary arterioles increases blood flow and shear stress through unoccluded channels, with the resulting increase in shear stress activating endothelial NO synthase (eNOS) to produce the potent vasodilator NO as well as the putative eicosanoid vasodilators prostacyclin (PGI_2) and prostaglandin E_2 (PGE_2). Entrapped microparticles activate monocytes and macrophages, triggering a cascade of intracellular signaling events including the release of platelet-activating factor (PAF) and expression of inducible NO synthase (iNOS). Entrapped microparticles and PAF stimulate thrombocytes to release the pulmonary vasoconstrictors thromboxane (TxA_2) and serotonin [5-hydroxytryptamine (5-HT)]. The iNOS enzyme produces copious quantities of NO and derivative reactive O^2-N species [e.g., nitrite (NO_3^-) and peroxynitrite ($OONO^-$)] that are nonspecifically cytotoxic. Nitric oxide relaxes pulmonary vascular smooth muscle, NO modulates (inhibits) PAF activation of thrombocytes and the release of TxA_2 and 5-HT, and NO and PGI_2 inhibit platelet aggregation and the formation of obstructive microthrombi. Vascular remodeling (hypertrophy, hyperplasia, and distal extension of pulmonary arteriole smooth muscle cells) is inhibited by NO (Tan et al., 2005), whereas 5-HT stimulates vascular remodeling. cGMP = cyclic guanosine monophosphate (adapted from Wideman et al., 2004).

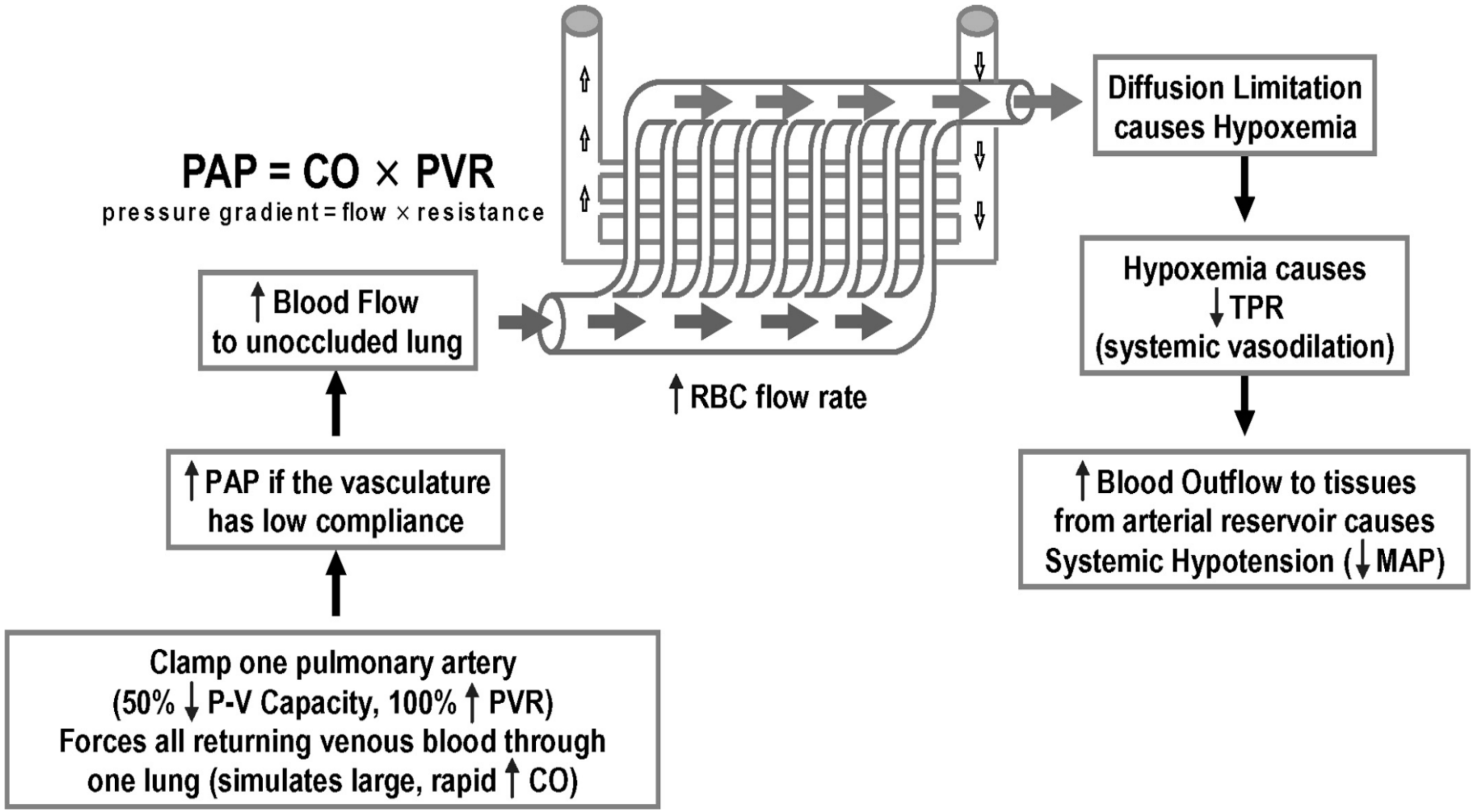

0.15
0.10
0.05
0.00
-0.05
-0.10
-0.15
0
25
50
75
100
125
150
دامنه (میلی ولت)
بازه زمانی (میلی ثانیه)
خط پایه
R
S
T
R'
R'b
Rb
R - S
Rb - R
S - R'
R' - R'b
S - T



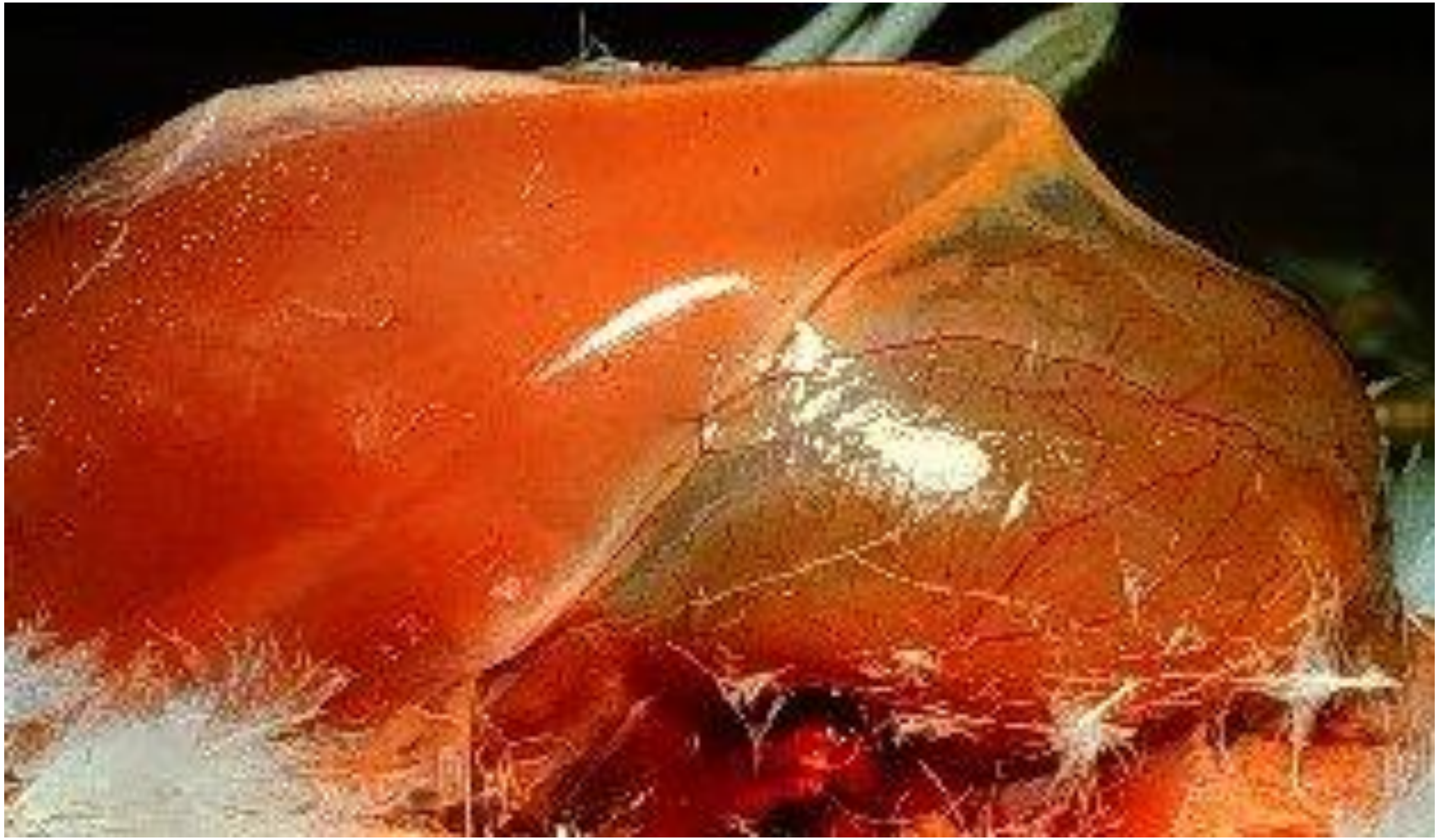
موج S یا دی پولاریزیشن
بطن افزایش یافته است

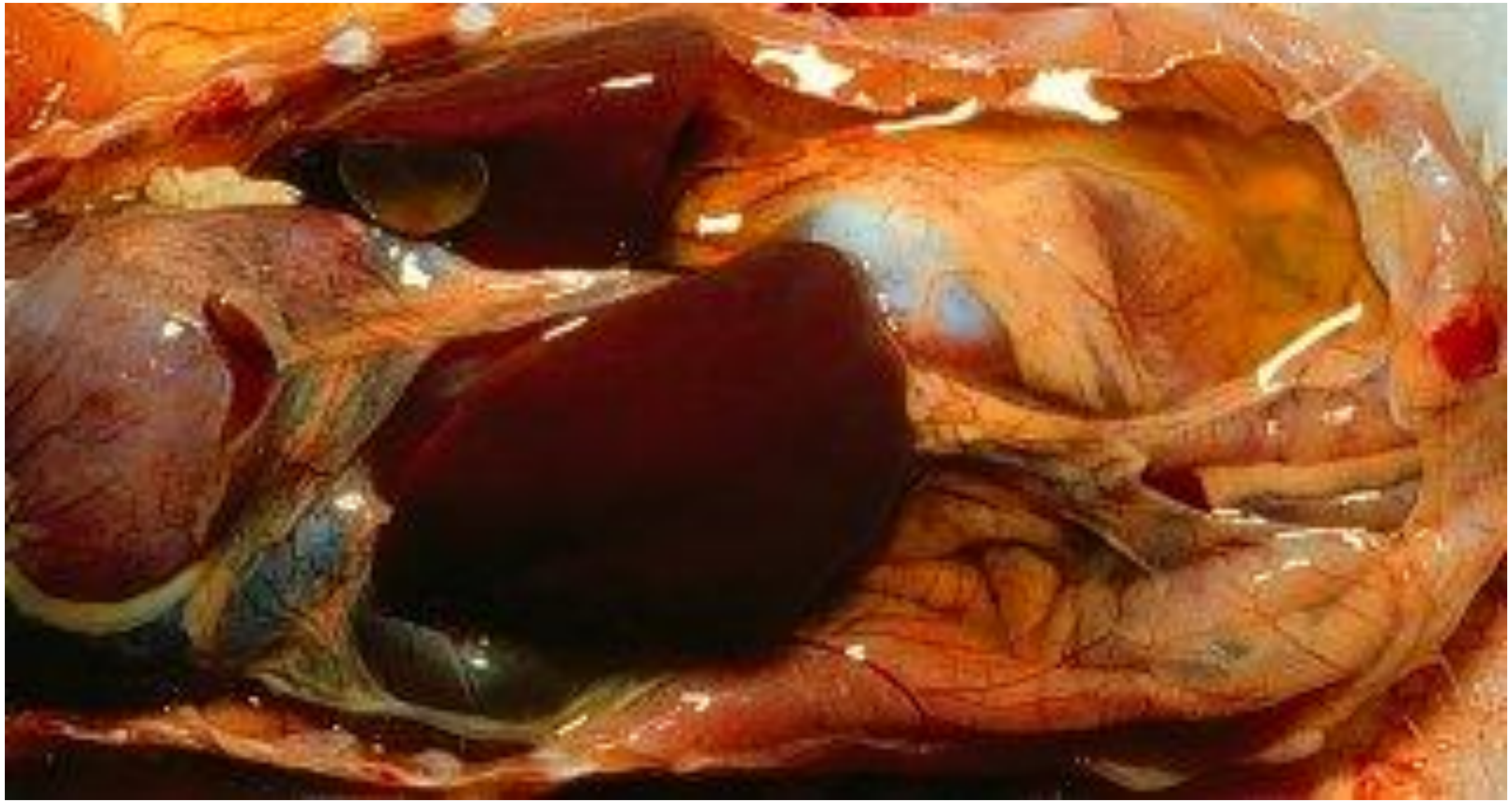


The pulmonary vascular (P-V) capacity encompasses anatomical components such as the compliance (elasticity), volume, and cumulative cross-sectional radius of the blood vessels, as well as functional components including vascular responsiveness to vasoactive mediators affecting the tone maintained by the primary resistance vessels. Pulmonary arterial pressure (PAP) is (approximately) equal to the cardiac output (CO) multiplied by the pulmonary vascular resistance (PVR). Resistance to flow through blood vessels is principally determined by the vessels' radius (r^4) rather than by length (L) or the viscosity of blood (η). Increases in PAP can be attributed to increases in CO, to anatomical inadequacies of pulmonary vascular capacity (increased PVR), or excessive vasoconstriction (increased PVR; adapted from Wideman and Bottje, 1993).



When the pulmonary vasculature is relatively noncompliant and fully engorged with blood, then experimentally reducing the pulmonary vascular (P-V) capacity by occluding 1 pulmonary artery doubles the pulmonary vascular resistance (PVR) and forces the right ventricle to double the pulmonary arterial pressure (PAP) to propel the entire cardiac output (CO) through the unoccluded lung. The rapid (within minutes) onset of systemic arterial hypoxemia (reduced partial pressure of O_2 on arterial blood) and hypercapnia (elevated partial pressure of CO_2 in arterial blood) are attributable to the onset of a diffusion limitation that is revealed when erythrocytes [red blood cells (RBC)] are forced to flow too rapidly past the pulmonary gas exchange surfaces to permit full blood-gas equilibration of O_2 and CO_2. Hypoxemia dilates the systemic vascular resistance vessels, reducing total peripheral resistance (TPR) and thus the mean systemic arterial pressure (MAP; adapted from Wideman, 2001).





Factors influencing pulmonary arterial pressure

PH

Factors influencing pulmonary arterial pressure

- 1- Increased pulmonary blood flow or cardiac output
  - A- Metabolic rate (growth rate)
  - B- Hypoxia
    - -Altitude
    - - Ammonia, dust
    - - Carbon monoxide
- 2- Organic vascular obstruction
  - A- Blockage of capillaries
  - B- Megalocytosis
  - C- Polycythemia
    - -Young birds
    - - Diet sodium
    - - Erythrocyte flexibility
- 3- pulmonary vasoconstriction → High altitude
- 4- Increased pulmonary venous pressure → Rarely seen in birds

Factors influencing pulmonary arterial pressure

A- Metabolic rate (growth rate)

1- Increased pulmonary blood flow

-Altitude

3.4 Kg broiler consume 2500 liters O_2

- 19% BM
- 36% BM+Maintenance+Activity
- 64% Gain

- Diet sodium

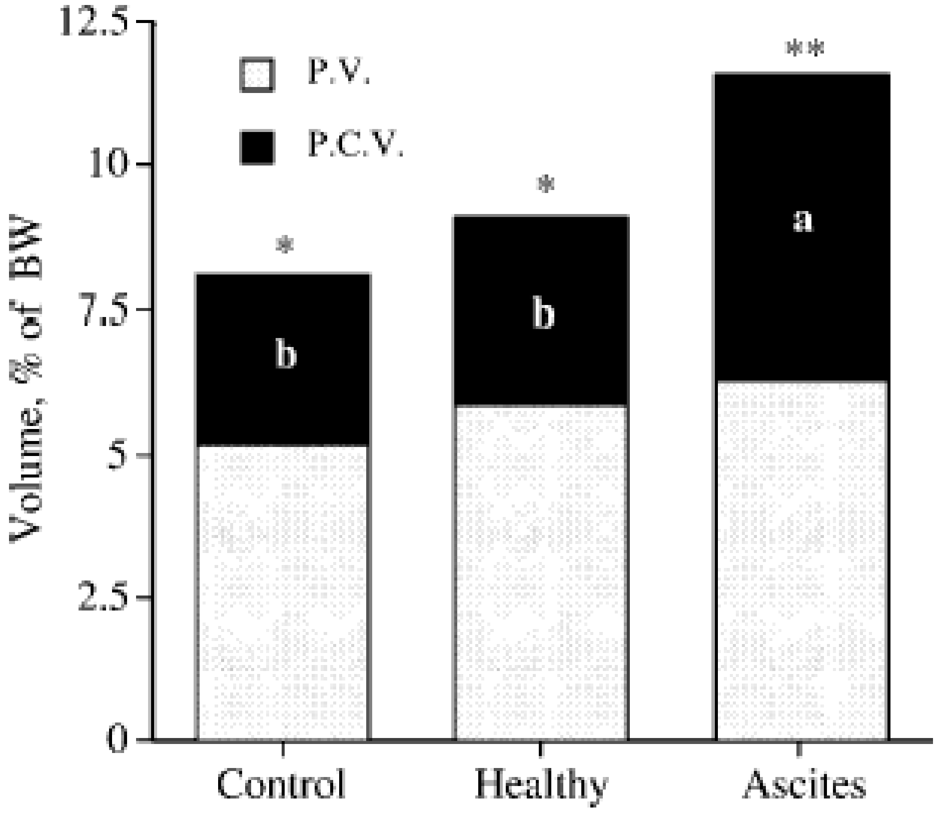
- Erythrocyte flexibility

3.1 liters O_2 per gram protein gain

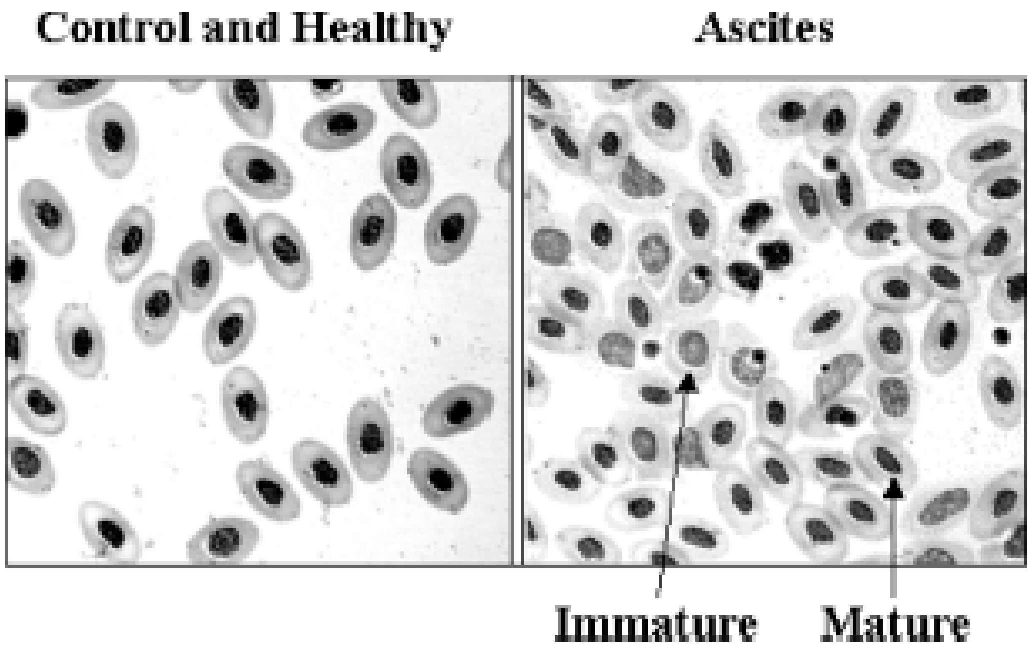
0.82 liters O_2 per gram fat gain

4- Increased pulmonary venous pressure → Rarely seen in birds

Erythropoiesis regulation during the development of hopoxemia



Blood (BV), plasma (PV), and packed cell (PCV) volumes in control, healthy, and ascitic broilers, presented as percentages of BW. For PCV and BV, different letters and asterisks, respectively, designate significant differences ($P \leq 0.05$); n = 10. For BV, SEM = 1.57; for PV, SEM = 1.35; and for PCV, SEM = 1.1.

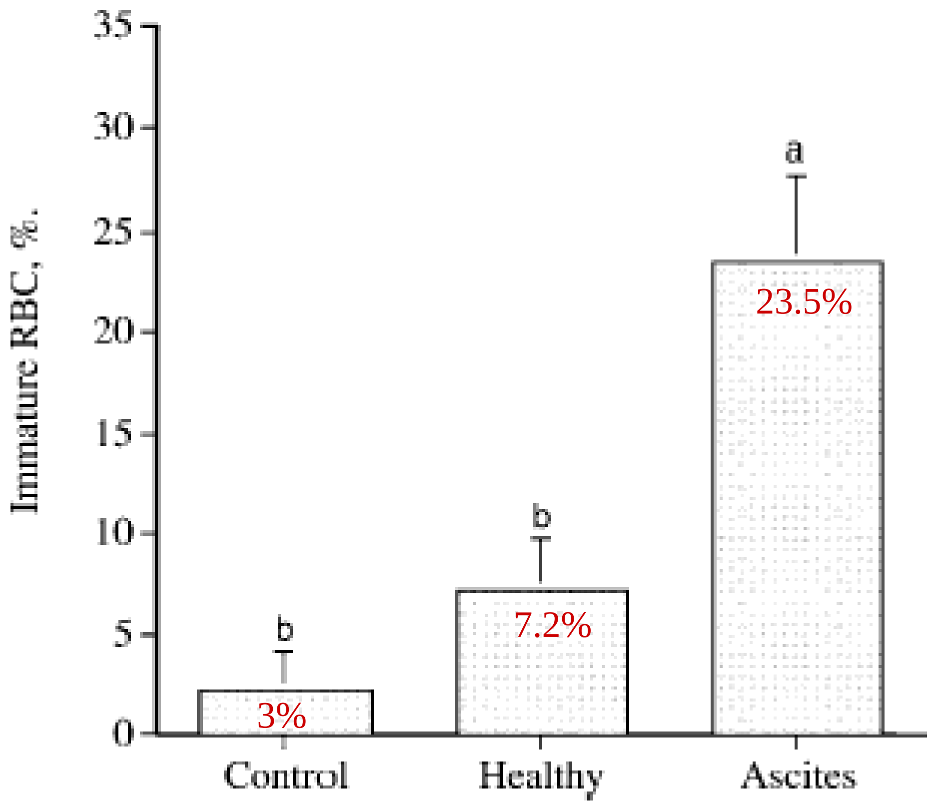


Blood smears from ascitic and nonascitic chickens were stained by the May Grünwald Gimsa method. Immature erythrocytes were characterized by large, rounded, and lightly stained nuclei, and resembled polychromatic erythrocytes. Immature and mature erythrocytes were counted in a total of 10 slides, with six different regions in each.

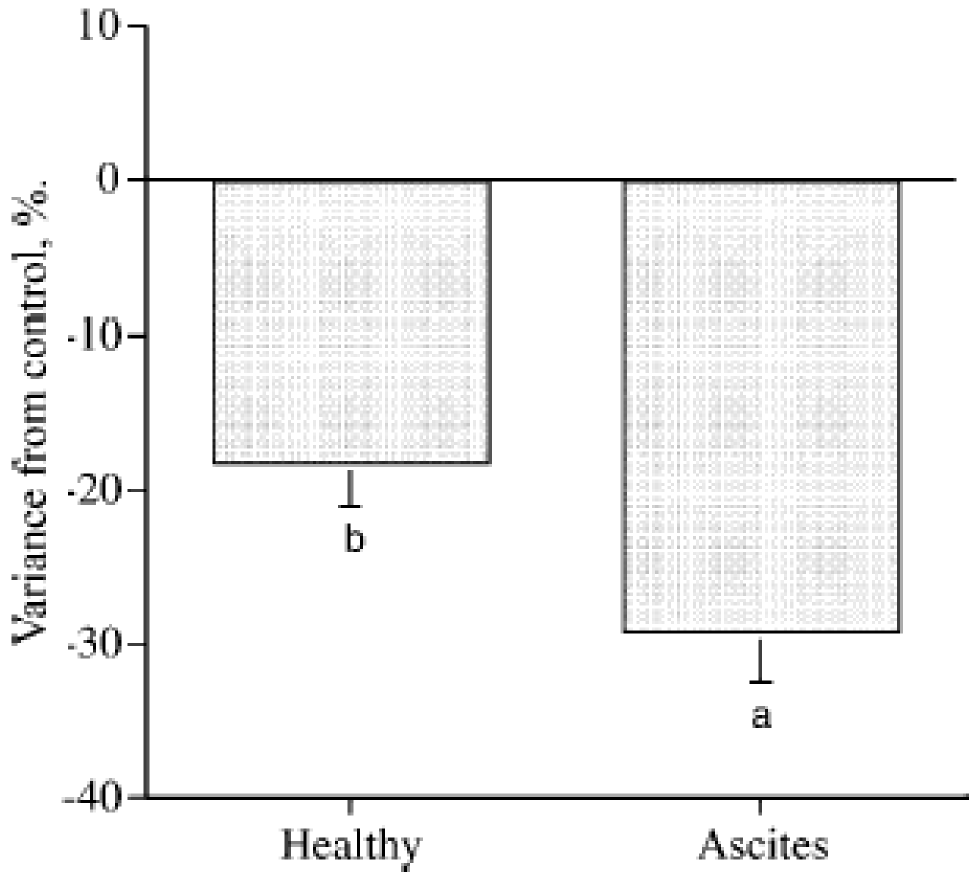
Corticostrone accelerating erythrocetes proliferation

Ascitic birds showed Hypothyroidism

(T_3 as a significant controller of erythrocyte diferentiation)



Percentage of immature cells in the red blood count (RBC). Immature and mature erythrocytes were counted in a total of 10 blood smear slides, with six different regions in each. Between columns, different letters designate significant differences ($P \leq 0.05$).



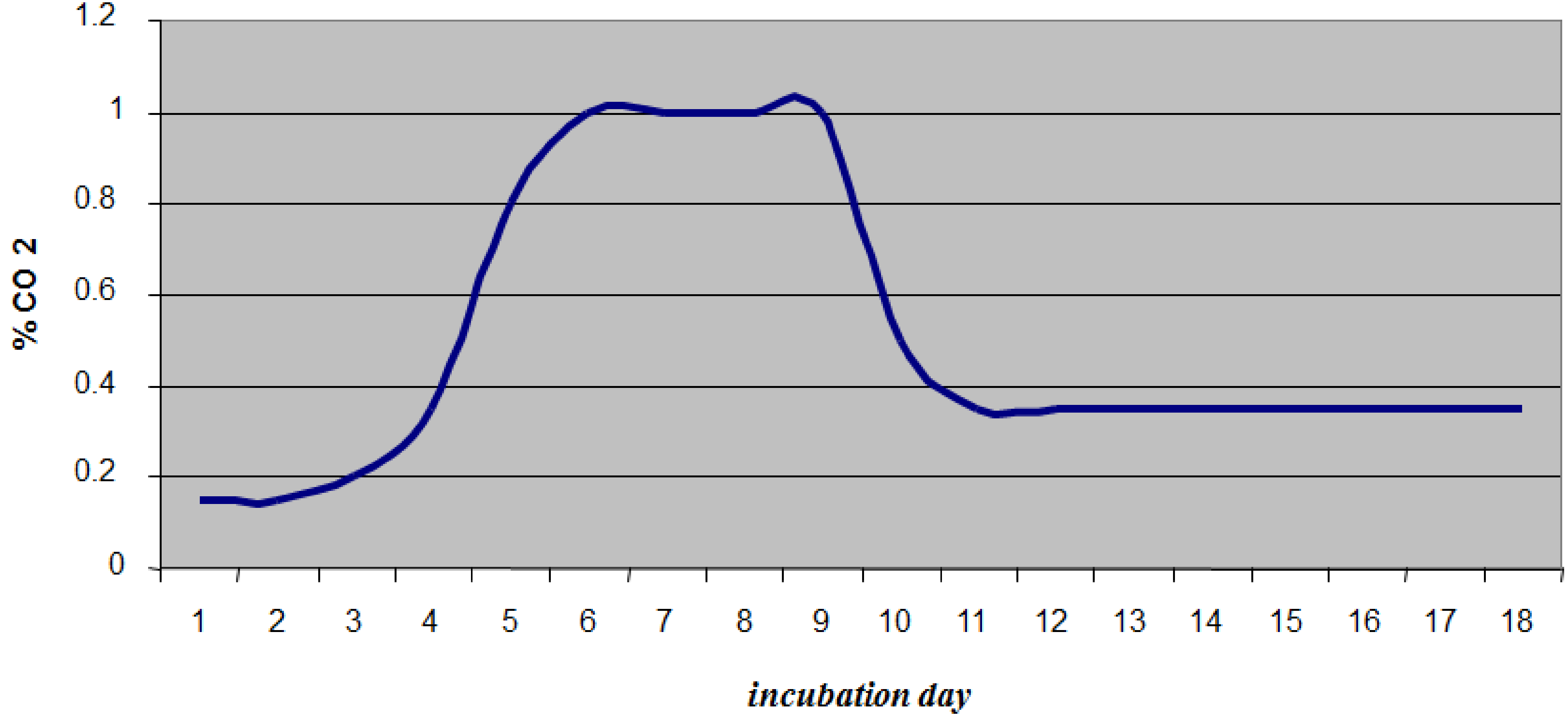
Hemoglobin content in blood cell count (1,000) of healthy and ascitic broilers, represented as variance (%) from control. Between columns, different letters designate significant differences ($P \leq 0.05$).

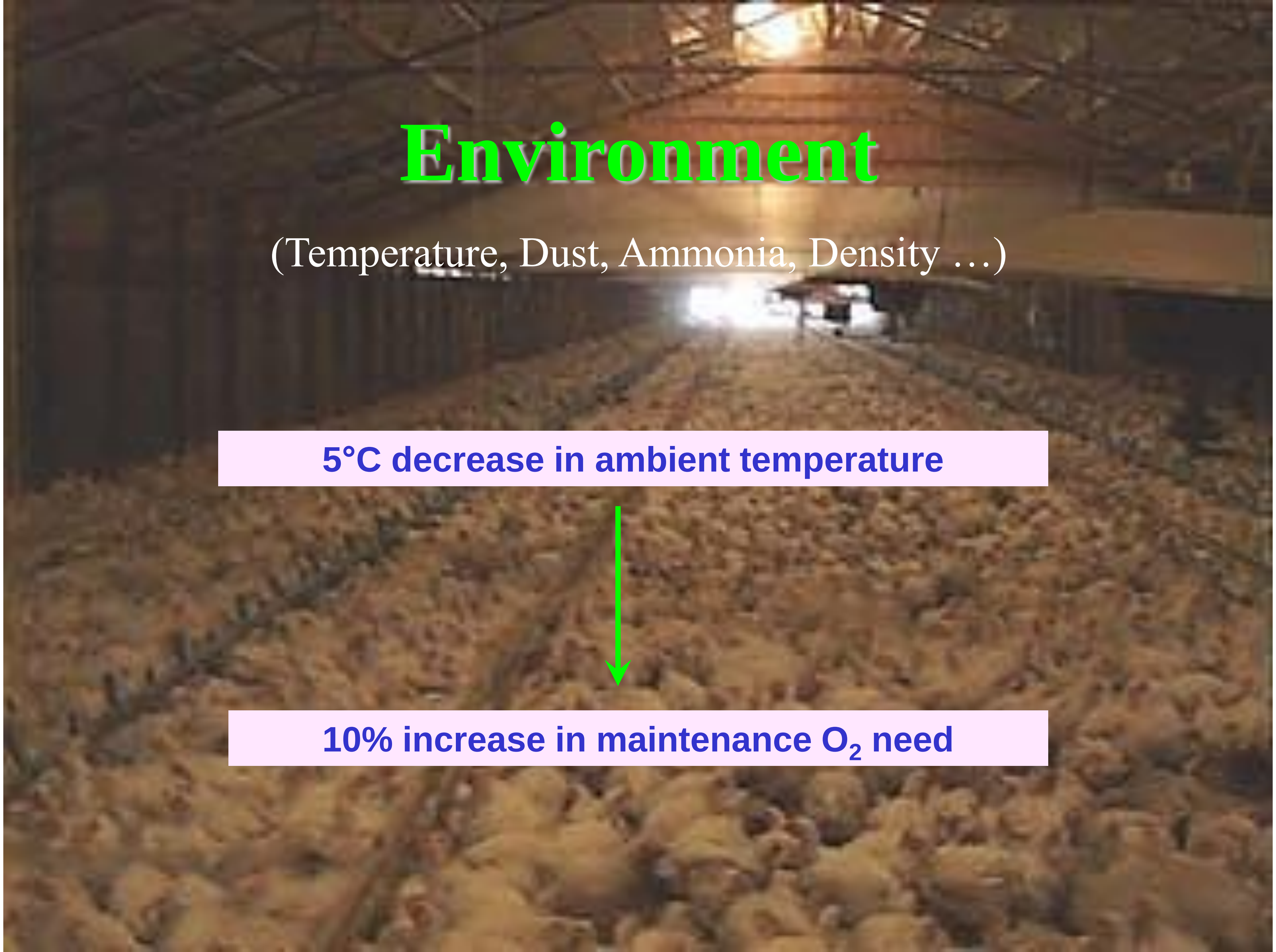
The development of mature peripheral red blood cells from pluripotent stem cells in the bone marrow is a complex process, regulated by several hormones.

- Erythropoietin
- Corticosterone
- Triiodothyronine
- Growth factors

پیشگیری

High early CO_2 levels





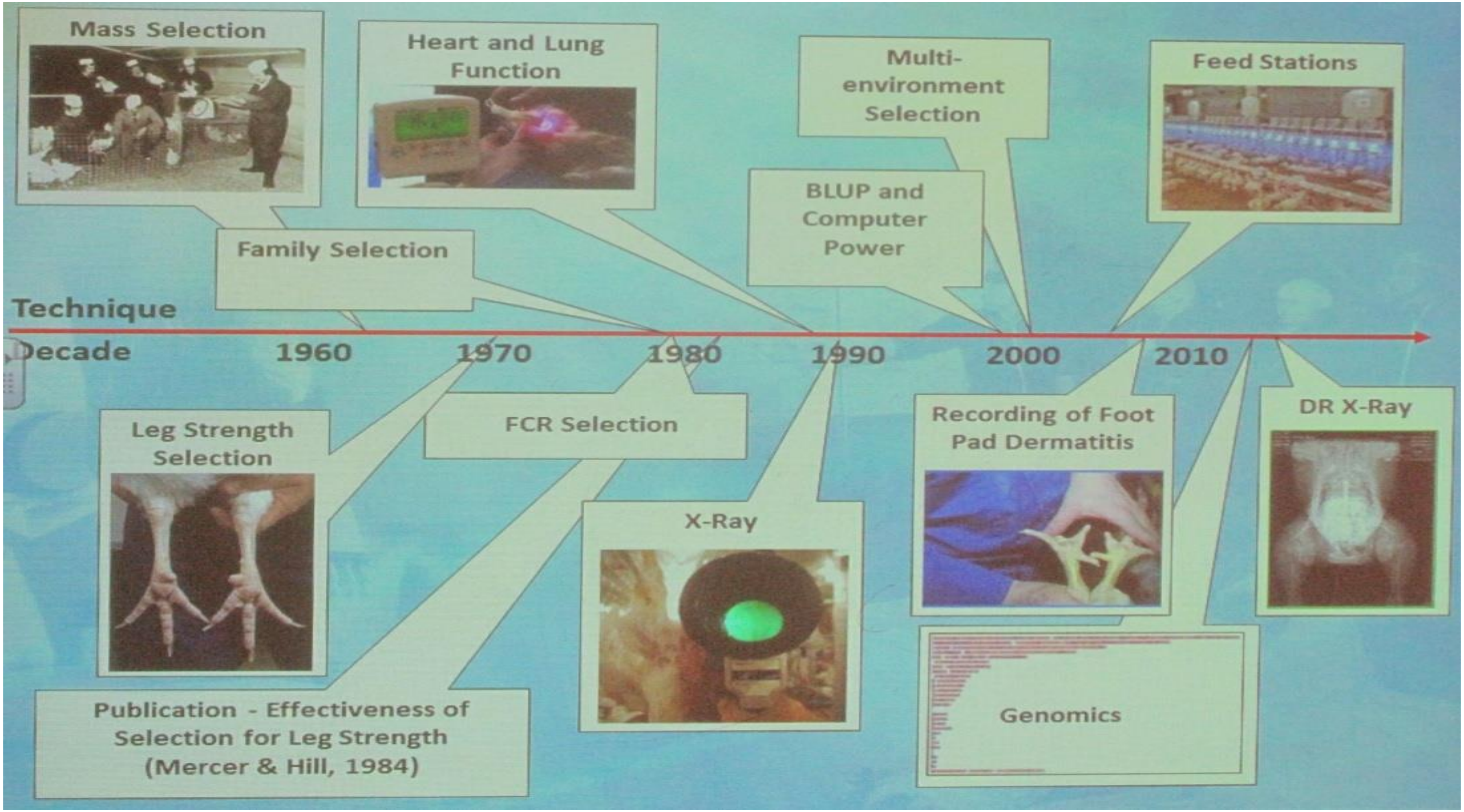
Environment

(Temperature, Dust, Ammonia, Density …)

5°C decrease in ambient temperature

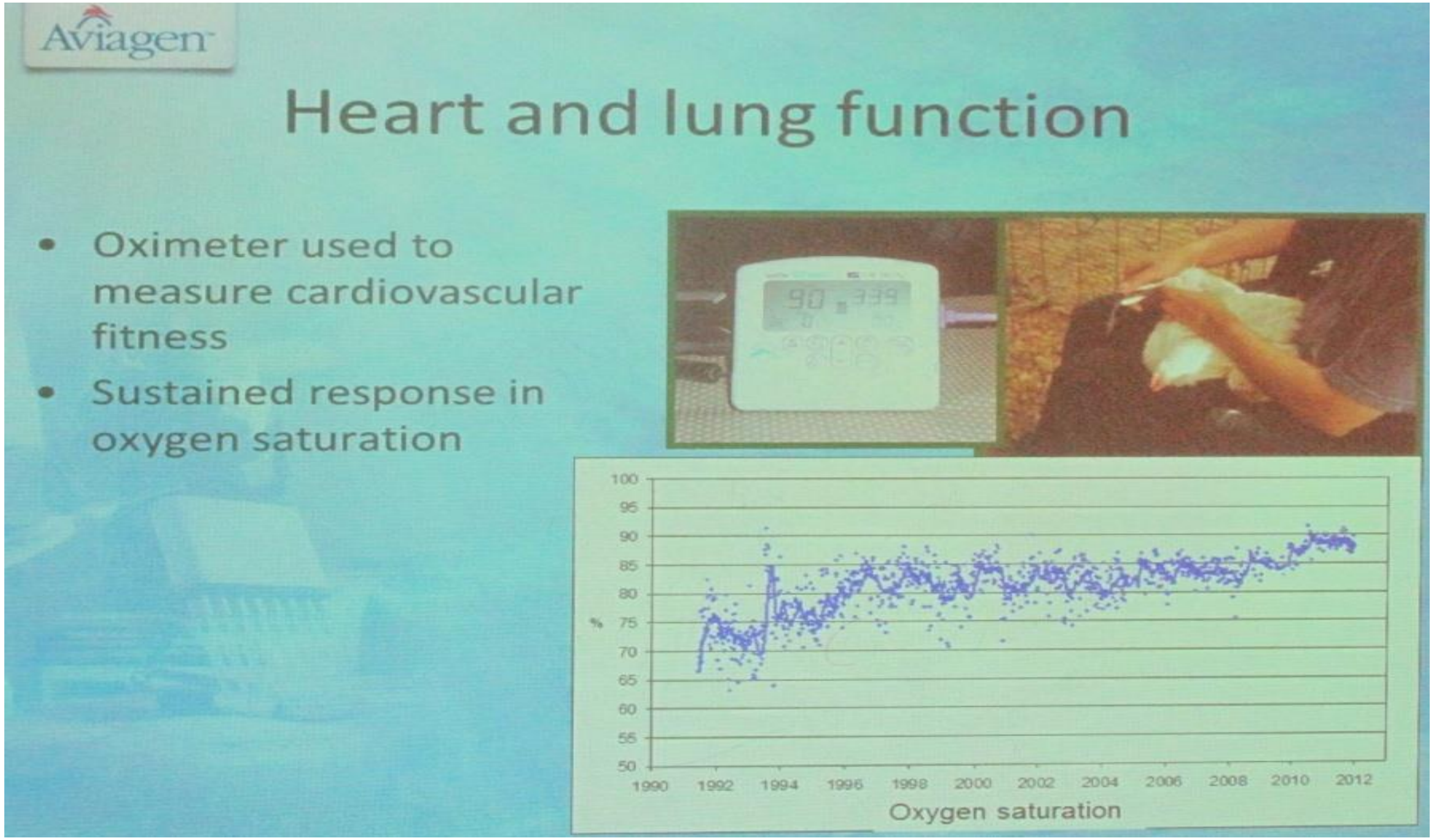
↓

10% increase in maintenance O_2 need



Mass Selection
Heart and Lung Function
Multi-environment Selection
Feed Stations
Family Selection
BLUP and Computer Power
Technique
Decade
1960
1970
1980
1990
2000
2010
Leg Strength Selection
FCR Selection
X-Ray
Recording of Foot Pad Dermatitis
DR X-Ray
Genomics
Publication - Effectiveness of Selection for Leg Strength (Mercer & Hill, 1984)

Selection criteria in primary breeding flock



NaHCO3
RESULTS
Hb + O2
Diet NaCl