

## INTRODUCTION

The aortic arches are embryonic vascular structures that play an important role in the formation of some definitive arteries in the adult. The aortic arches and their resulting structures arise from the migration and reorganization of the cells of the cardiac neural crest (CNC), therefore, variations in the migration or remodelling of the CNC's cells could result in congenital malformations of the aortic arches. These anomalies are uncommon pathologies and affect much more dogs than cats. In spite of their relative poor incidence, some of them represent an important part of the cardiovascular alterations in dogs under one year.

The aims of this bibliographic review were to explain the changes of the aortic arches in order to form other vascular structures, define the most typical anomalies of the arches, research into the possible aetiologies, pathogenies and frequencies of these malformations and describe the clinical symptomatology and treatment.

## FORMATION OF VASCULAR STRUCTURES

Table 1. Remodelling of the aortic arches

AORTIC ARCH	DEFINITIVE STRUCTURE
I	Regression/ Maxillary arteries
II	Regression
III	Common carotid arteries Internal carotid arteries
IV	R: Right subclavian artery, Brachiocephalic trunk L: Aortic arch
VI	R: Regression L: Pulmonary trunk, Arteriosus duct

R: right; L: left

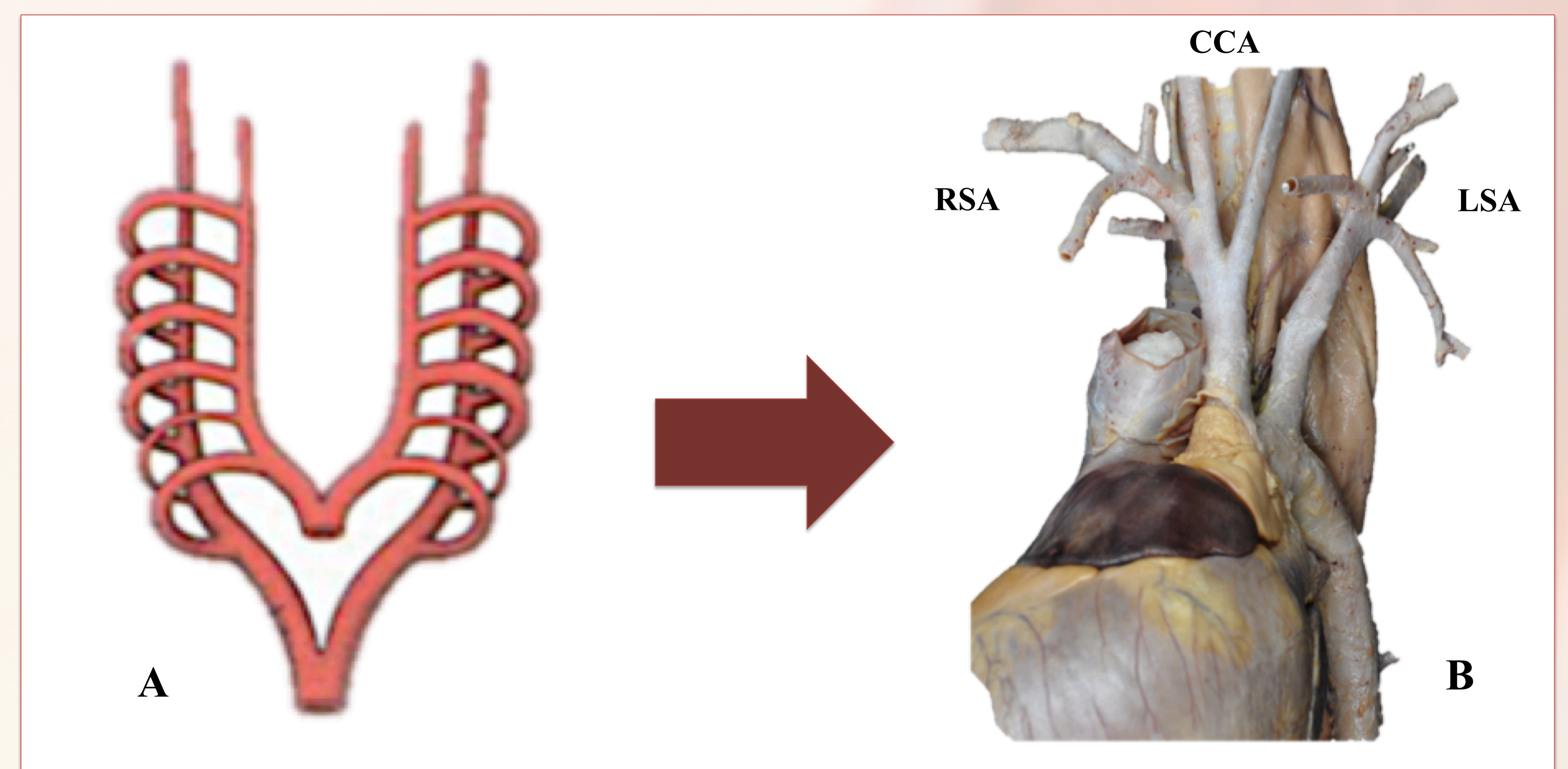


Figure 1. Ventral vision of the initial (A) and final (B) stage of the aortic arches. (A: Gilbert, 2003)  
RSA: right subclavian artery; CCA: common carotid arteries; LSA: left subclavian artery.

## AORTIC ARCHES ANOMALIES

### Vascular ring anomalies

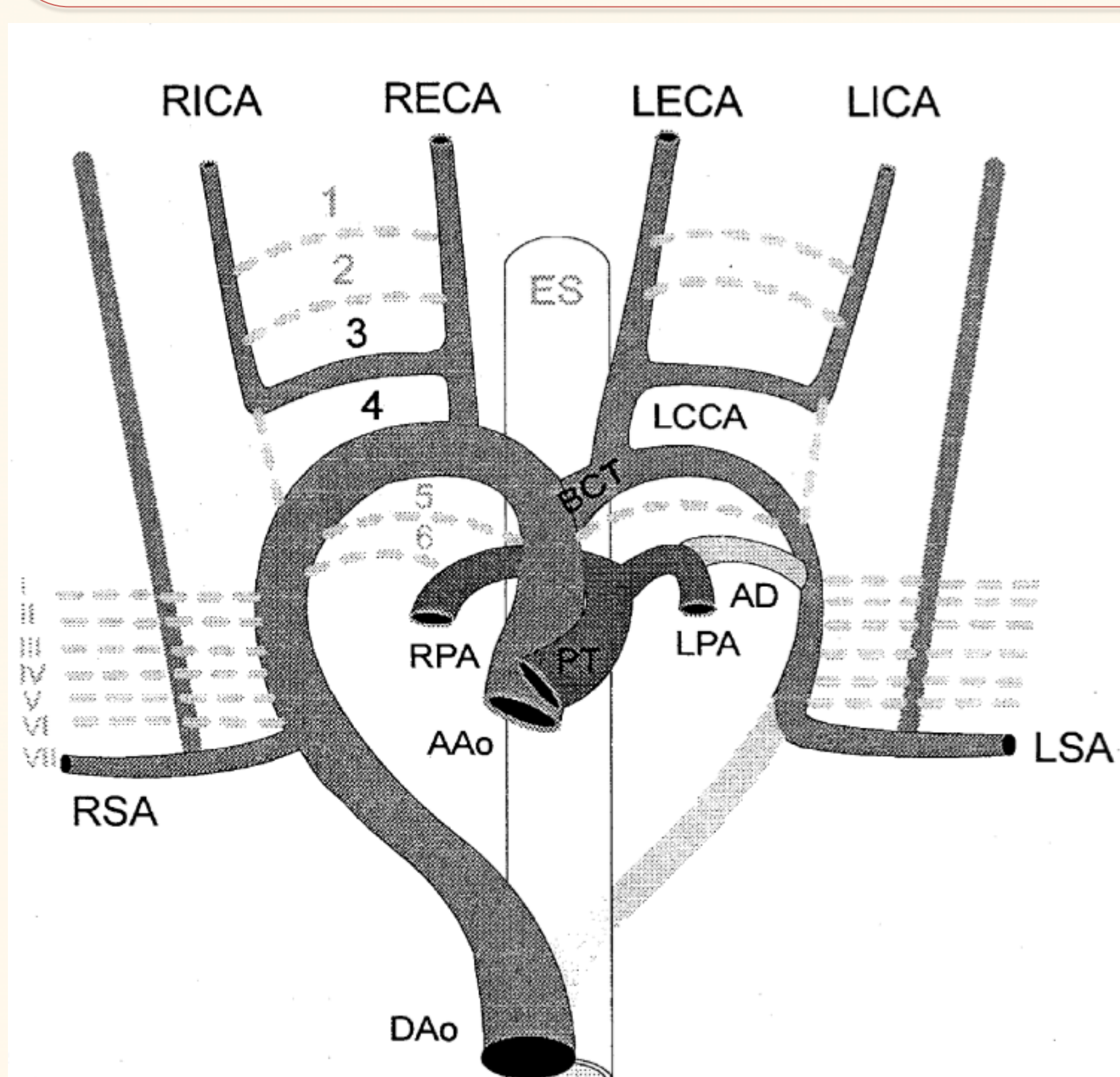


Figure 2. Persistent right aortic arch.

RSA: right subclavian artery; RICA: right internal carotid artery; RECA: right external carotid artery; LECA: left external carotid artery; LICA: left internal carotid artery; LSA: left subclavian artery; AD: ductus arteriosus; Aao= ascendant aorta; Dao: descendent aorta; PT: pulmonary trunk; RPA: right pulmonary artery; LPA: left pulmonary artery; ES: oesophagus. (Craatz *et al.*, 2003)

### Persistent right aortic arch

#### Aetiology

- Pinscher, German Shepherd, Greyhound
- 22q11 chromosome in human
- CFA26 region in chromosome 26 in canine
- Felines?
- *Neuropilin1* in mice

**Frequency:** dogs < cats

**Pathophysiology:** constriction of the oesophagus and trachea

**Symptomatology:** regurgitation, dyspnoea

**Diagnosis:** Rx

**Treatment:** surgery

### Patent ductus arteriosus

#### Aetiology

- German Shepherd, Cocker Spaniel, Maltese, Poodle, Shetland and others. Female > male
- Concentration of oxygen and prostaglandins
- Interaction between actin and myosin
- Transcription factor AP-2 $\beta$
- Environmental factors

**Frequency:** common in dogs.

**Pathophysiology and symptomatology:**

- Left to right: A $\rightarrow$ P. Congestive left failure
- Right to left: P $\rightarrow$ A. Caudal cyanosis, polycythaemia.

**Diagnosis:** echocardiography

**Treatment:** surgery

A: aorta; P: pulmonary trunk

## DISCUSSION AND CONCLUSIONS

The aetiology of these alterations is still unclear due to the shortage of studies into the veterinarian field. According to the review, genetic aetiology was by far the most observed in all anomalies. These genetic alterations affect the formation and migration of the cardiac neural crest and the remodelling of the aortic arches. The lack of information in this field and the lower incidence of these anomalies are responsible for the way these pathologies are confront. Instead of preventing such alterations, the goal of most veterinarians is to diagnose and to treat these congenital malformations.

To sum up, even though some of these congenital anomalies are diagnosed in a higher frequency than others, in veterinary medicine this field remains unknown and it bases its management in the treatment of these malformations.

#### References:

- Craatz, S. *et al.*. 2003. Right-sided aortic arch and tetralogy of Fallot in humans- a morphological study of 10 cases. *Cardiovascular Pathology*, 12: 226-232.
- Gilbert, S.F. 2003. *Developmental Biology*. 7a Edición. Sinauer Associates Inc, USA. pp: 838.