J. exp. Biol. **158**, 539–564 (1991)
Printed in Great Britain © *The Company of Biologists Limited 1991*

THE PHYSIOLOGY OF THE ALLIGATOR HEART: THE CARDIAC CYCLE

BY G. SHELTON

School of Biological Sciences, University of East Anglia, Norwich NR4 7TJ, UK

AND D. R. JONES

Department of Zoology, University of British Columbia, 6270 University Boulevard, Vancouver, BC, Canada V6T 2A9

Accepted 27 February 1991

Summary

Pressure recordings from the heart and major arteries of the alligator show that a conventional relationship exists between the left ventricle and the right aorta. Pressure gradients from ventricle to aorta during systole are very small. Right aortic blood flow rises rapidly to a single peak and then falls more gradually until aortic valve closure.

The right ventricle is connected both to the pulmonary arteries and to the left aorta. Right ventricular pressures show that systole is a two-stage process. Initially, blood leaves to the low-resistance lung circuit, though appreciable pressure gradients exist across the pulmonary outflow tract. Active contraction of the pulmonary outflow tract stops pulmonary ejection and a second-stage pressure rise is seen in the right ventricle.

When systemic blood pressures are high, this second-stage pressure does not reach the levels recorded in the left aorta, and the left aortic valves remain closed so that lung and body circuits are functionally separate. An alternation of flow is found in the left aorta under these conditions, with reversed flow during systole and forward flow during diastole. Flow rates are extremely low, compared with those in the right aorta or pulmonary arteries, and the foramen of Panizza has very little significance in the cardiac cycle.

If the systemic blood pressures are low, the second stage of systole in the right ventricle gives rise to pressures that are higher than those in the left aorta, the left aortic valves open and blood is ejected to the systemic circulation, giving a right-to-left shunt. This can occur with no changes in pulmonary pressures or flows. Left aortic flow is not dependent on increased constriction of the pulmonary outflow tract, which continues to function as an on–off active valve. Constriction within the lung vasculature may, on some occasions, be significant in establishing left aortic flow, but it is clear from the present work that low systemic blood pressure is a factor of crucial importance.

Key words: alligator, heart, cardiac cycle, left aorta, active valve, blood shunt, blood pressures, blood flows, *Alligator mississipiensis*.

Introduction

Consideration of the evolution of structure and function in the reptile heart reminds us of the advice given to the enquiring traveller that his destination is easy to find provided he does not start from his present position. In the anapsid and diapsid reptiles, the anatomical relationships between the partially divided ventricle and its three output vessels (the right and left aortae and the pulmonary artery) evolve in directions that seem to make complete separation of lung and body circuits difficult to achieve (Webb *et al.* 1971, 1974; Webb, 1979). It may be that such separation would not be adaptive in these animals. It has been argued, with some justification, that because of central shunts the ability to regulate blood flow to the lungs independently of that to the body persists as an adaptive feature in intermittently breathing animals such as amphibians and the vast majority of reptiles (Shelton, 1985; Burggren, 1987; Shelton and Croghan, 1988). However, the fact that intermittent breathing can develop in spectacular form in diving birds and mammals (Butler and Jones, 1982) without central shunts undermines the argument a little. In addition, the selective advantages of the variable lung perfusion that is allowed by central shunts in intermittent breathers are hypothetical and largely untested. Finally, in varanid lizards, there is complete division of the ventricle during systole (Millard and Johansen, 1973; Burggren and Johansen, 1982) and almost certainly during diastole too (Webb *et al.* 1971; Heisler *et al.* 1983; G. Shelton, D. R. Jones and W. K. Milsom, unpublished work). The anatomical arrangements are extraordinary. An interventricular partition formed by the atrioventricular valves operates during diastole, whereas the muscular ridge within the ventricle divides it at a quite different site during systole. The varanids seem to have achieved an almost completely divided double circulation from a rather unpromising starting position, even though some species are well-adapted divers that breathe intermittently. Though a great deal of the physiology is still rather sketchily worked out and evolutionary trends difficult to see, there can be no doubt that the cardiovascular systems in living reptiles are evidence that a lot of evolutionary experiment has gone on in the group.

The heart and arterial arches in the Crocodilia show further evidence of that experiment and add to the confusion. These animals breathe intermittently and have a complete interventricular septum but the left aorta opens, together with the pulmonary artery, from the right ventricle (Fig. 1), a fact that has puzzled anatomists since the 17th century (see Grigg, 1989). More recently, White (1956, 1969) showed that, during normal cardiac cycles, oxygen levels in the left and right aortae were similar and that high systemic blood pressures kept the left aortic valve permanently closed, thus preserving complete separation of lung and body circuits. He suggested that, during systole in these circumstances, blood flowed into the left aorta from the right *via* the foramen of Panizza, a small connection between the left and right aortae just outside the aortic valves (Fig. 1). Greenfield and Morrow (1961), in contrast, thought that the foramen was closed during systole by the medial cusp of the right aortic valve, but that blood flow from right to left occurred during diastole. Grigg and Johansen (1987) suggested that flo[redacted]

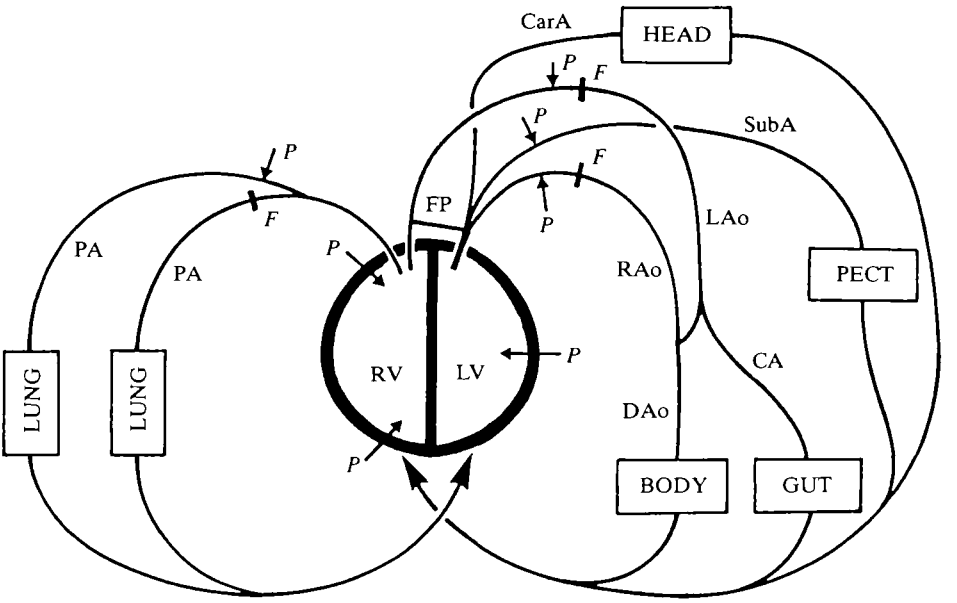

Fig. 1. Diagram to show details of connections between ventricles and central arteries in alligator circulation and to indicate sites where pressures (*P*) and flows (*F*) were measured. CA, coeliac artery; CarA, carotid artery; DAo, dorsal aorta; FP, foramen of Panizza; LAo, left aorta; LV, left ventricle; PA, pulmonary artery; PECT, pectoral region; RAo, right aorta; RV, right ventricle; SubA, subclavian artery.

could occur at both times in different circumstances and that the variability of different investigators' results could be attributed largely to changes in diameter of the foramen of Panizza. Direct measurement of flow in the left aorta of the caiman (Axelsson *et al.* 1989) showed that, in the undisturbed animal, flow rates were very low and entirely dependent on movement of blood through the foramen during most of the cardiac cycle.

However, this controversy seems rather peripheral to the fundamental problem of the connection between right ventricle and left aorta. In his important papers of 1968 and 1969, White suggested that the resistance of the pulmonary outflow tract from the right ventricle was variable and regulated by a cholinergic mechanism. During forced dives, with accompanying bradycardia, he found that the resistance of the outflow tract increased so that pressures in the right ventricle also increased and ultimately blood was ejected into the left aorta. In spite of the complete interventricular septum, therefore, the Crocodilia had retained the ability to shunt blood away from the lungs (right-to-left shunt) during certain types of apnoea. White (1970, 1976) developed these views further and the right-to-left shunt has since been confirmed by the pressure measurements and oxygen determinations of Grigg and Johansen (1987), though only in unforced, aerobic dives. Axelsson *et al.* (1989), however, were unable to confirm flow into the left aorta from the right

ventricle in their animals, even during voluntary dives. Dives with bradycardia and hypoxia could not be elicited in their calm and contented caimans and injections of acetylcholine were necessary to produce a right-to-left shunt *via* the left aorta.

There are still many aspects of the cardiac physiology of crocodiles that are largely conjectural and, until the details have been worked out, it will not be possible to understand the significance of the anatomical arrangements in the physiology of the whole animal and in the context of evolutionary change. The present paper attempts to describe some of the basic relationships between pressures and flows in the heart and arterial arches. Later papers will attempt to answer the more difficult questions on the broader issues of whole-animal physiology and adaptive significance of the relationships.

Materials and methods

The experiments were carried out on eight alligators (*Alligator mississipiensis*) purchased from a commercial alligator farm in South Carolina and shipped by air to the UK. Their mean body mass was 4.01 kg with a range from 2.79 to 5.02 kg. They were kept in a temperature-controlled room at 25 °C. Infrared lamps were positioned 1 m above the floor so that the crocodiles could bask and a trough of water was provided in which they could submerge at will. They fed aggressively and remained in good condition throughout the holding period.

All the experiments described in this paper were performed on anaesthetised animals with open chests. Anaesthesia was initially induced by placing an animal in a closed box containing a pad soaked in halothane. This procedure took some time because the animal breathed infrequently. When the alligator became unconscious it was placed on an operating table and a tracheal cannula was introduced through the glottis, which had previously been sprayed with xylocaine. The cannula was then connected to an intermediate animal ventilator (Harvard Apparatus, South Natick, MA) and the lungs were ventilated continuously at 2.5–4.0 breaths min^{-1} with a tidal volume of 15–20 ml kg^{-1}. These values were based on data given by Naifeh *et al.* (1970) for caiman and alligator. The ventilation was sufficient to keep arterial blood fully oxygenated and reduce the animal's own ventilation movements to a minimum. If the pump was stopped, however, as it was quite frequently in order to judge the level of anaesthesia, the animal began making the intermittent breathing movements characteristic of the species. The gas used for ventilation was usually a 50 % oxygen/50 % nitrous oxide mixture, though on occasions when very light anaesthesia was required this was changed to 80 % oxygen/20 % nitrous oxide. During the surgery, halothane up to a concentration of 4 %, but more usually of 2 %, was added to the oxygen/nitrous oxide mixture by means of a Fluotec 3 vaporiser (Cyprane Ltd, Keighley, UK). The halothane concentration was reduced to approximately 0.5 % after surgery had been completed, the level being adjusted to give appropriate levels of anaesthesia throughout the experiments. The animals were maintained at 25–30 °C

during the operations and experiments by means of a heated pad on the operating table and an infrared lamp above it. Rectal temperature was monitored continuously.

The whole heart and the arterial arches were exposed, in the initial experiments, by a ventral incision through the midline of the sternum, beginning midway between the front and rear legs and continuing forward to the level of the front legs. Incisions of this length proved to be unnecessary after the initial exploration, even in animals in which simultaneous pressure recordings were made from the ventricles and arteries. The major arteries are contained within a single, large connective tissue sheath as they emerge from the ventricle. This truncus is about the same length as the ventricles and is enclosed within the pericardium. In it the pulmonary artery and right aorta expand into chambers of substantial volume (called sinuses by Webb, 1979). The left aorta, in contrast, remains small and may even decrease in diameter in the truncus. The arterial arches were separated and freed of connective tissue, over lengths sufficient for flow probe and pressure catheter implantation, as they emerged from the anterior ends of the truncus and pericardium. There was little blood loss during surgery.

Several pressure values, up to a maximum of six, were measured simultaneously by inserting polythene catheters (PP60; i.d. 0.8 mm, o.d. 1.2 mm) into the left aorta, either the left or right pulmonary artery, and either the right aorta or the left subclavian artery (Fig. 1). The arteries were clamped off and a small hole was made in the wall with a hypodermic needle, and the catheter inserted. It was held in place with a purse string of surgical thread tied round the catheter and taking in a small amount of the arterial wall all the way round the aperture. This technique was subsequently modified by flaring the end of the catheter before insertion and then pulling the flared end back against the arterial wall before tightening the fixing loop. Pressures were also measured in different regions of the right and left ventricles (Fig. 1). Holes were made in the myocardium with a hypodermic needle and catheters, with their ends cut to sharply angled points, were then inserted through the holes and tied in place with a loop of surgical thread taking in the outer connective tissue coat of the ventricle. The pericardium, which had to be opened for the placement of ventricular catheters, was closed by a number of sutures. Again, blood loss was minimal.

The pressures were measured with BioTec BT 70 and Elcomatic EM 750 pressure transducers, both types being strain gauge instruments with variable resistances in a bridge configuration. They were connected to Hewlett Packard 8805A carrier preamplifiers or strain gauge bridge amplifiers. The pressure signals were displayed on a Hewlett Packard six-channel pen recorder writing on rectangular coordinates, and on a Medelec FOR 4.2 recording oscilloscope. The latter was used to produce superimposed records such as those of Figs 2 and 7. The transducers could be connected to columns of saline so that zero and calibration pressures could be applied to them at any time. The pressure-measuring apparatus was filled with 0.8 % NaCl containing 50 i.u. heparin ml^{-1} and care was taken to exclude air bubbles from transducers and catheters. The frequency characteristics

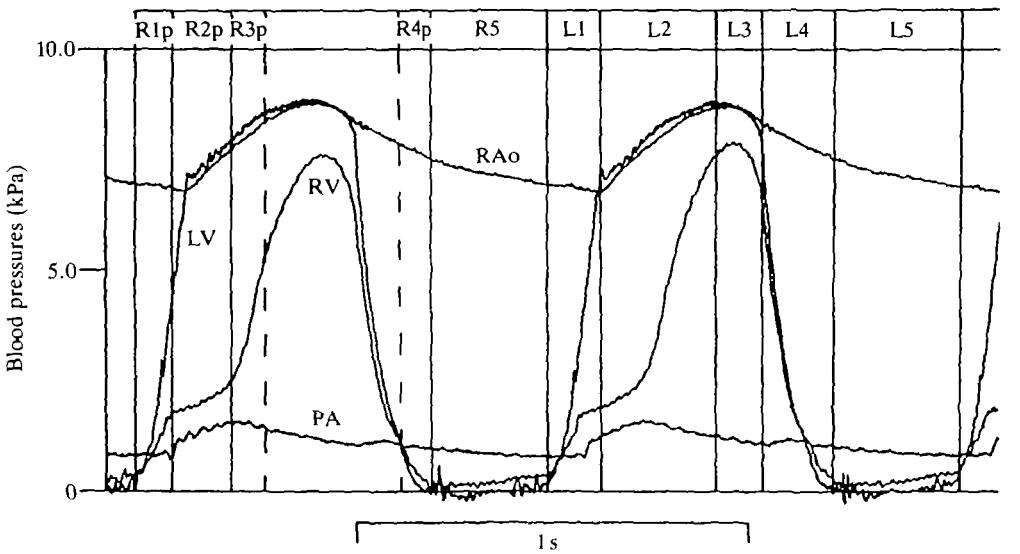

Fig. 2. Simultaneous pressure recordings from left ventricle (LV), right ventricle (RV), pulmonary artery (PA) and right aorta (RAo) in a 3.6 kg alligator. The letters above the recordings indicate phases of the cardiac cycle on the left (L) and right (R) sides of the heart, as described fully in the text. Mean durations (S.E.) of the phases in seconds, determined from three animals, are as follows for the left heart: L1, 0.121 (0.006); L2, 0.336 (0.010); L3, 0.109 (0.007); L4, 0.198 (0.008); L5, 0.380 (0.013); and for the right heart: R1p, 0.082 (0.007); R2p, 0.218 (0.012); R3p+R4p, 0.465 (0.022); R5, 0.375 (0.013). The mean heart rate to which these durations apply was 52.5 beats min^{-1}.

of the overall system were adequate to record the pressures found without distortion.

Blood flow was measured in the right aorta, the left aorta, and either the right or left pulmonary artery, whichever was not being used for pressure measurements (Fig. 1). Biotronix electromagnetic flow probes were used in all cases, with a 3 mm probe being fixed around the right aorta and 2.5 mm probes around the other two vessels. The probes were driven from Biotronix 410 and 610 flowmeters. The flow-measuring apparatus was calibrated after the experiments using a piece of excised right aorta perfused with saline from a reservoir at the correct height to give mean arterial pressure. Flow rates were regulated by a screw clamp on the outflow tube and the amount of saline collected in a measured time was determined by weighing. Five or six flow rates were used for each probe and the best calibration line was established by linear regression. Zero flow levels were determined regularly throughout the experiments by mechanical occlusion of the vessels, downstream of the probes, using miniature artery clamps.

A number of experiments were carried out in attempts to modify the relationships between the right and left ventricles and their respective arterial output vessels. The animal was arranged on the operating table so that its hea

could be tilted backwards and submerged in a beaker of water. In this way forced dives could be simulated for periods up to 10 min. The temperature of the animal could be changed by resetting the temperature-controlled pad and by augmenting the infrared heating above the operating table. Cooling was achieved by surrounding the alligator with ice-filled bags.

Results

The main features of the cardiac cycle are summarised in Fig. 2 which shows the timing of events, Table 1 which gives details of blood pressure and Table 2 which gives data on blood flow. The experiments were so arranged that these data summaries could be obtained from at least three animals; thus, blood flows were, in the main, measured in a group of three animals and ventricular blood pressures were measured in a different group, also of three animals. Sets of data were collected on at least three occasions in each animal, at times when the cardiovascular system was judged to be stable, there had been no recent experimental interventions and anaesthesia was at a light level. Arterial blood pressure and heart rate were measured in all animals and so provided the basis for establishing comparabilities between groups.

The cardiac cycle on the left side of the heart can be divided into five phases as shown in Fig. 2, based on significant components in the pressure records. The occurrence of the following events separates each phase of the cycle from its predecessor: (L1) a pressure rise marks the beginning of isovolumic ventricular contraction with no blood leaving to the right aorta; (L2) a pressure rise in the right aorta marks the beginning of ejection of blood from the left ventricle; (L3) peak pressure occurs in the left ventricle and outflow vessels and ejection slows;

Table 1. *Systolic (S) and diastolic (D) blood pressures from the right aorta, left aorta and pulmonary artery, the pressure at the incisura (I) marking valve closure between left ventricle and right aorta, and the systolic pressure in the right ventricle during the second stage of contraction*

		Blood pressure (kPa)							
		Right aorta			Left aorta		Pulmonary artery		Right ventricle
	Heart rate (beats min^{-1})	S	I	D	S	D	S	D	S
Mean	50.3	10.16	9.57	7.92	9.62	7.85	2.09	0.90	6.18
S.E.	2.4	0.43	0.41	0.36	0.39	0.35	0.17	0.07	0.54

There was no ejection from right ventricle to left aorta during any of the sampling periods.

Systolic pressure in the left ventricle is not measurably different from that given for the right aorta.

The data are derived from measurements on six alligators.

(L4) ejection stops and an inflection marks the closure of the aortic valves followed by isovolumic ventricular relaxation; (L5) pressure reaches zero in the left ventricle and filling begins. Identification of equivalent phases in the right heart is complicated by there being two components in its cycle. However, it is convenient to adopt the same criteria, as far as possible, to establish five phases for the right ventricle as it relates first to the pulmonary and then to the systemic circulations. Clearly there is a common filling phase (R5), which is not substantially different in timing from phase L5. However, the isovolumic contraction, ejection and isovolumic relaxation phases on the right side are different in relation to the pulmonary (R1p–R4p) and systemic (R1s–R4s) circulations, because of the sequential nature of ejection to the pulmonary artery and left aorta, and they are dealt with separately in the descriptions that follow. Only pulmonary phases are labelled in Fig. 2 because right ventricular ejection to the left aorta does not occur. Some systemic components can always be seen, even in the absence of ejection, with systemic isovolumic contraction (R1s) being immediately followed by isovolumic relaxation (R4s), but they are not analysed in Fig. 2. Systemic ejection phases from the right ventricle will be described in a later section. To make comparisons easier, vertical lines, showing pulmonary phases in the right ventricle together with those in the left ventricle, have been drawn where possible on Figs 3 and 4. Similar lines to show systemic phases in the right ventricle have been drawn on Figs 7, 8 and 9.

The durations of the five phases shown in Fig. 2 are given in the caption, the values being means derived from three animals with heart rates close to the mean value of $52.5+0.97$ beats min^{-1}. When the rate was made to decrease by the intravenous injection of acetylcholine, only the filling phase (5) changed substantially in duration. The remaining phases increased slightly, though not significantly, so the times given in Fig. 2 are not unduly sensitive to changes in the level of vagal tone such as would be involved, for example, in the development of a mild diving bradycardia.

Left ventricular output

The pressure and flow relationships between the left ventricle and its output vessels, the right aorta and the carotid and subclavian arteries, were of a conventional pattern (Figs 2, 3 and 4). The pressure in the left ventricle increased rapidly during the isovolumic contraction in phase L1 to 7.92 kPa (Table 1), at which time the right aortic valves opened passively and blood was ejected with increasing velocity into the aorta and its associated vessels (Fig. 4, phase L2). Pressures in the ventricle and arteries continued to rise during phase L2 to mean systolic values of 10.16 kPa (Figs 2 and 3; Table 1), though flow rate eventually began to decline. This decrease in flow continued through phase L3 and the aortic valves closed passively. Pressures also fell, rapidly approaching zero in the ventricle during the isovolumic relaxation of phase L4. Diastolic pressure run off was identical in the subclavian and carotid arteries and the right aorta during phases 4 and 5. A substantial flow was maintained in these vessels throughou

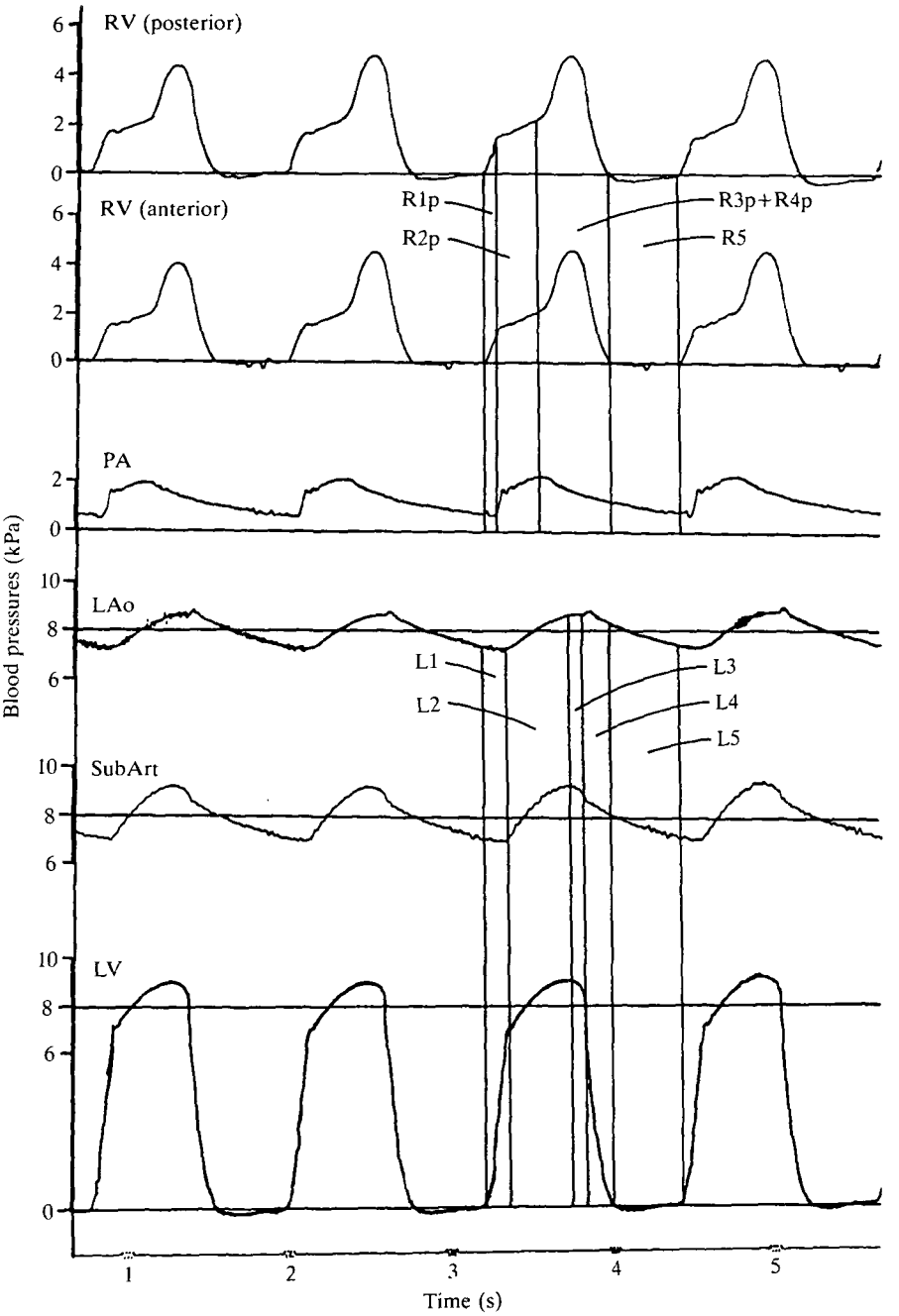

Fig. 3. Pressures recorded from the left ventricle (LV), right ventricle (RV), subclavian artery (SubArt), left aorta (LAo) and pulmonary artery (PA) of a 3.8kg alligator. The vertical lines and numbered sections of trace refer to left ventricular phases (L1–L5) and right ventricular phases (R1p–R5) as described in the text.

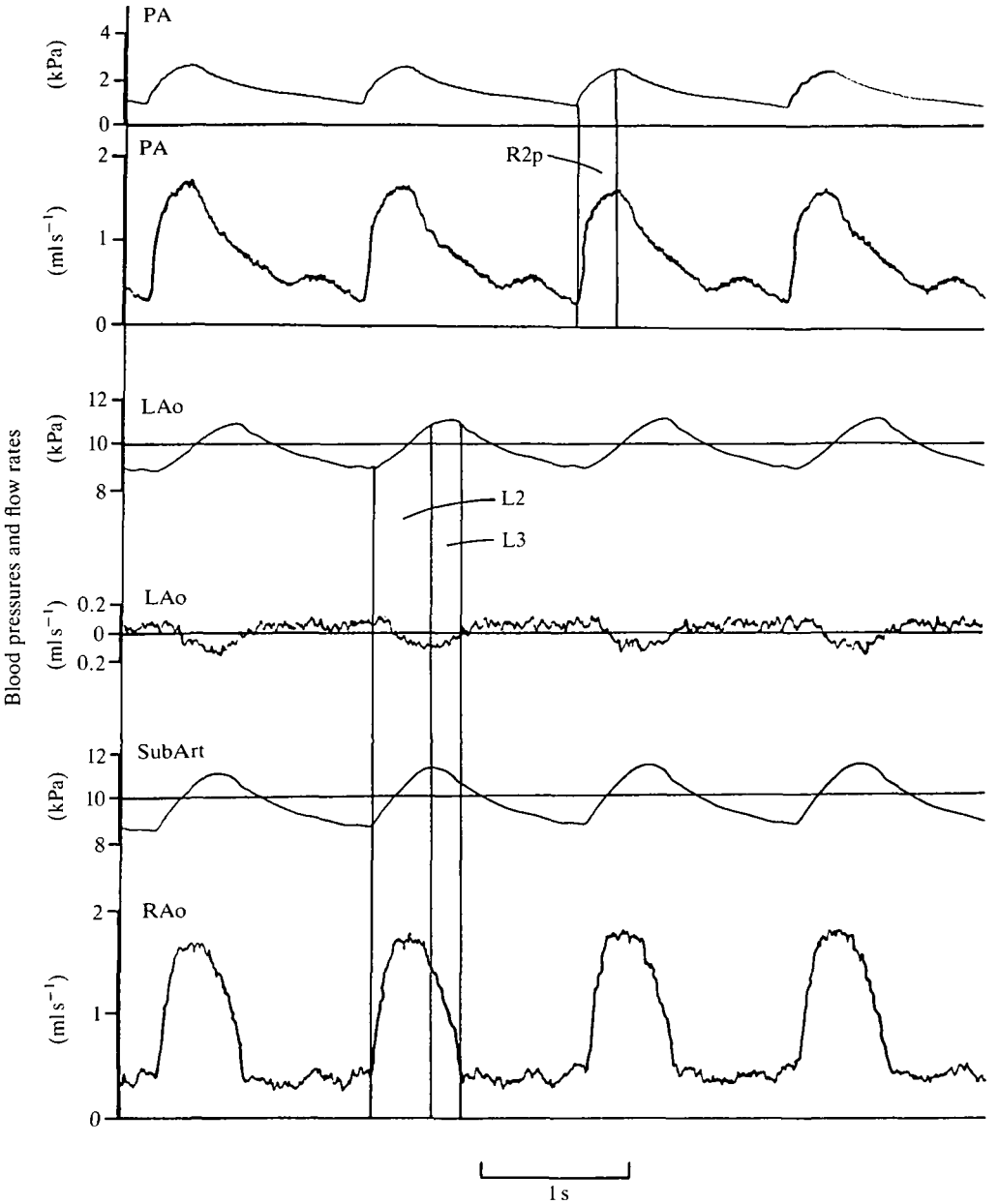

Fig. 4. Pressures and flows recorded from the right aorta (RAo) and subclavian artery (SubArt), the left aorta (LAo) and the pulmonary artery (PA) of a 4.6 kg alligator. The right aorta and the pulmonary artery flow rates were 44.1 ml min^{-1} and 52.5 ml min^{-1}, respectively, and the stroke flows were 1.05 ml $cycle^{-1}$ and 1.25 ml $cycle^{-1}$, respectively. Forward flow in the left aorta was 3.4 ml min^{-1} and reversed flow was 3.0 ml min^{-1}. The forward stroke flow was 0.08 ml $cycle^{-1}$ and reversed stroke flow was 0.07 ml $cycle^{-1}$. The vertical lines and numbered sections of trace refer to phases of the cardiac cycle in the right and left hearts.

diastole. Flow zero was checked on numerous occasions and the maintained flow confirmed; the level changed very little during diastole in spite of the continuously falling arterial pressures.

Flow values for the pulses shown are given in the figure captions and mean values from three animals are summarised in Table 2.

Right ventricular output

Pressure and flow relationships between the right ventricle and its output vessels, the pulmonary arteries and the left aorta, are much more complex. The pulmonary stage of right ventricular systole began synchronously with that in the left ventricle, though the rate of rise in pressure was lower on the right side (phase R1p, Figs 2 and 3). The rate diminished even more at a well-defined inflection, coinciding with the sudden increase in pulmonary artery pressure (phase R2p) or following soon after. Right ventricular pressure then began to increase at progressively greater rates during phase R3p, marking the beginning of phase R1 s and ultimately reaching peak values with a mean of 6.18 kPa. Peak systolic value varied over a much wider range (S.D. = 1.33 kPa) than the equivalent value in the left ventricle (S.D. = 1.06 kPa) and occurred approximately 25 ms later. In trabeculate hearts it is possible to record two component pressures of the type shown here if the catheter tip is enclosed within the trabeculae and squeezed by the ventricular muscle as it contracts. Though the alligator heart has fewer trabeculae than most reptile hearts, the multi-chambered nature of the ventricle suggested that such an artefact could not be discounted. Pressures were therefore recorded from catheters located in two different parts of the right ventricle (Fig. 1). Identical pressure outlines were always found, even in regions of the ventricle as far away from one another as possible (Fig. 3).

Pressures in the pulmonary arteries were related to, but not the same as, the initial components found in the right ventricle during systole. The lung circulation in alligators is clearly a high-flow, low-resistance system in which the arterial pressures are very low, having a mean systolic value of only 2.09 kPa. In contrast, the flow rates were higher in both the right and the left pulmonary artery than those measured in the right aorta with the flow probe located downstream of the branches to the carotid and subclavian arteries (Table 2). Slightly less than half the resistance of the whole pulmonary system was located in the pulmonary outflow tract, as judged by the pressure gradients between ventricle and arteries (Figs 2 and 3). Moreover, that part of the resistance possessed some non-linear characteristics. The increase in arterial pressure and the beginning of blood flow in the pulmonary vessels (phase R2p) did not occur until 80 ms after right ventricular contraction had started. Usually, an abrupt increase in both pulmonary pressure and flow was found following this delay, as though some part of the outflow region had suddenly opened, but, even so, arterial pressure did not rise to the levels found in the right ventricle (Figs 2 and 3). The inflection in the ventricular pressure trace occurred as pulmonary arterial pressure increased abruptly and high flow rates were established (phase R2p, Figs 3 and 4). As pressure and flow in the

Table 2. *Heart rate and measured blood flow rates in the right aorta, left aorta and pulmonary artery*

	Heart rate (beats min^{-1})	Flow rates (ml min^{-1})						
		Right aorta	Left aorta		Pulmonary artery	Subclavian artery	Carotid artery	Cardiac output (ml min^{-1})
			F	R				
Alligator 6, 4.6 kg; occluded subclavian arteries								
Mean	39.3	44.6	5.5	2.6	49.5	0	51.7	98.9
S.E.	1.5	1.7	2.3	0.3	1.5		6.3	3.1
Alligator 7, 4.7 kg; no occlusions								
Mean	59.7	22.7	3.3	0	32.4	10.7	28.2	64.9
S.E.	2.2	3.6	0.2	0	1.6	–	–	3.2
Flow distribution (%)		35	5		–	16	44	100
Alligator 8, 3.3 kg; no occlusions								
Mean	45.5	53.7	7.3	2.3	67.8	13.3	63.6	135.6
S.E.	2.6	3.1	0.7	0.4	4.3	–	–	8.6
Flow distribution (%)		39	4		–	10	47	100

Left aortic values are given as forward flow (F) and reversed flow (R).

Cardiac output (pulmonary flow×2) in all alligators, and carotid artery flow (carotid flow=cardiac output−right aorta flow − left aorta flow) in alligator 6 with occluded subclavian arteries, are estimated.

Subclavian artery flow and percentage flow distributions are also estimated, assuming that the carotid:(right aorta+left aorta) ratio is the same in alligators 7 and 8 as in alligator 6.

pulmonary artery reached peak values at the end of phase R2p (Figs 2, 3 and 4), the systemic stage of right ventricular contraction began with the pressure rising rapidly. Resistance of the pulmonary outflow tract must have increased substantially at this point because pulmonary arterial flow began to fall exponentially through phases R3p and R4p.

It is difficult to decide at what point ventricular ejection stopped, or even whether it continued at a low level over much of the systemic stage of right ventricular contraction. The boundary between phases R3p and R4p cannot therefore be shown with certainty on Fig. 2. Eventually, pressure in the chamber fell below that in the pulmonary artery and from this stage, at least, relaxation was isovolumic (phase R4p) and the pulmonary arterial valves closed.

During the later stages of phase R3p, through R4p into phase R5, two small additional pulses were sometimes seen to distort the smooth exponential decline in both flow and pressure traces from the pulmonary arteries. The first of these pulses (Figs 2 and 5) occurred just as the ventricles of both sides began to relax, coinciding more or less with phase L4. The second (Figs 4, 5, 8 and 9) began as pressures fell to zero in both ventricles and filling started (phases L5 and R5).

For most of the time in these acute preparations the pressures in the right ventricle, though variable, never reached the levels found in the left aorta and the left aortic valves remained closed throughout the cardiac cycle. All the blood leaving the right ventricle was conveyed to the lungs. Cardiac output from one side of the heart could be estimated as twice the flow measured in a pulmonary artery (Table 2), assuming equal flow in the two vessels.

Left aortic patterns: with no right ventricular components

When the left aortic valves were closed throughout the cycle, pressures in the left aorta resembled those found in the right in their timing but differed importantly in value. The systolic pressures in the left aorta were lower, by a mean value of 0.54 kPa, than those in the right, a difference that is significant at the 0.1% level (using paired comparisons and Student's *t*-test). The differences between the two diastolic pressures were much smaller, with a mean of 0.07 kPa, but they were still significant at the 2% level.

At the end of phase L3, when the right aortic valves closed, a notch was frequently observed in the trace from the left aorta (Figs 3 and 6). Its size depended on the difference that had developed between the two pressures during the preceding systole. The most important determinant of this pressure difference was the resistance to flow offered by the left aorta below the pressure catheter (i.e. in the direction of the dorsal aorta). In preparations in which a flared catheter was inserted through the arterial wall and then pulled back so that the flare abutted the wall itself, no obstruction was caused, left and right pressure differences were small, and the notch was small or non-existent (Fig. 4). In earlier experiments, a short length of catheter was inserted into the lumen of the vessel before being anchored to the wall, so that resistance to flow was increased by an unknown amount. In some of these cases a very large notch or spike was seen (Fig. 6).

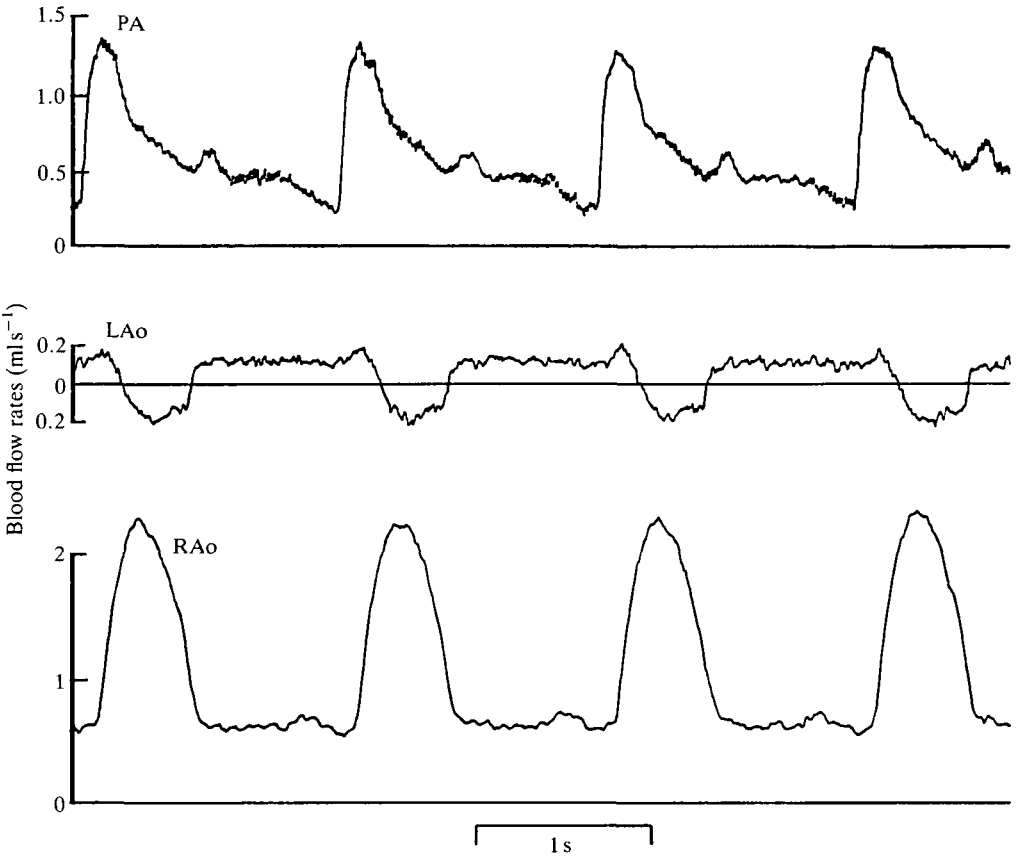

Fig. 5. Blood flows recorded from the right aorta (RAo), left aorta (LAo) and pulmonary artery (PA) of a 3.3 kg alligator. The right aorta and pulmonary artery flow rates were 63.2 ml min^{-1} and 39.2 ml min^{-1}, respectively, and the stroke flows were 1.59 ml $cycle^{-1}$ and 0.96 ml $cycle^{-1}$, respectively. In the left aorta, forward flow rate was 6.0 ml min^{-1} and reversed flow 2.6 ml min^{-1}. Left aortic stroke flows were 0.15 ml $cycle^{-1}$ forward flow and 0.06 ml $cycle^{-1}$ reversed flow.

Orientation of the catheter was important. When the record in Fig. 6 was taken, the catheter opening was facing towards the heart. The additional resistance, caused by the catheter itself, was therefore located between its opening and the dorsal aorta. Immediately afterwards, the catheter was redirected towards the dorsal aorta so that the additional resistance was located between the heart and the catheter opening. The spike disappeared and records resembling those shown in Fig. 3 were obtained. In all cases, regardless of catheter type or orientation, or the size of the spike, differences in pressure between right and left aortae became very small after aortic valve closure and remained so throughout diastole (Table 1; Figs 3, 4 and 6).

Two types of flow pattern were seen in the left aorta when there was no outflow from the right ventricle. The usual one (Figs 4 and 5), found in five out of a total o

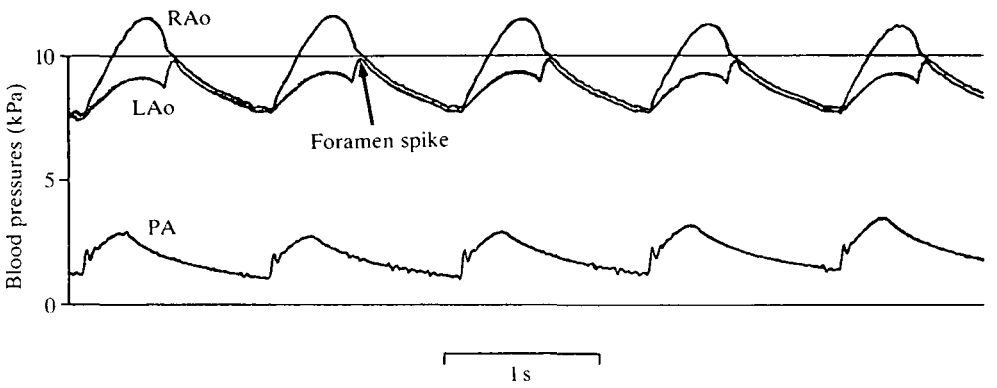

Fig. 6. Pressure records from the right aorta (RAo), left aorta (LAo) and pulmonary artery (PA) of a 3.8 kg alligator. The sudden jump in pressure (foramen spike) as the right aortic valves close and the foramen of Panizza opens is clear, as is the close relationship between the declining left and right aortic pressures following closure of the aortic valves.

six animals in which some flow measurements were made, was an alternation of flow towards the heart (reversed flow) during phases L2 and L3 and away from the heart (forward flow) during phases L4, L5 and L1. Reversed flow began during phase L2, some 100 ms after the aortic pressure pulses started to rise, and was usually preceded by a very small increase in forward flow which began synchronously with the start of phase L2 (Figs 4 and 5). The crossover from reversed flow to forward flow occurred exactly at the end of phase L3 as the right aortic valves closed and the notch in the left aortic trace was observed (Fig. 4). Flow rates in the left aorta in either direction were always very low compared to those in the right aorta and pulmonary arteries. Forward flow was greater than reversed flow (Table 2).

The second pattern, seen in only one of the six animals in which flows were recorded, was one in which forward flow was maintained in the left aorta throughout the cardiac cycle. In this animal there was a pulse of increased flow that began at the same time as phase L2. This pulse ended and flow fell briefly to zero as phase L3 ended. Forward flow at a very low level was then maintained in the left aorta throughout the rest of diastole.

Left aortic patterns: with right ventricular components

In four alligators fundamentally different relationships began to emerge spontaneously, as judged from pressures measured in three animals and pressures and flows recorded from one animal. The conditions under which the pattern appeared were not easily defined, although it was clear that the systolic pressures in the left ventricle and its associated vessels were significantly lower, at the 1% level, than the mean value given in Table 1. The systolic pressures in the right ventricle, however, were not significantly different from the mean value given in

Table 1. The pressures in Table 1 were determined when there was no blood flow to the left aorta.

The clearest indication of the appearance of this new pattern was a change in the shape of the left aortic pressure pulse. An increased slope developed in the rising pressure wave during phase L2 (Figs 7 and 8). It was produced when pressures in the right ventricle equalled or slightly exceeded those in the left aorta (Fig. 7). Blood was ejected through the open aortic valves, causing a transition in the left aortic pressure as it became determined by right ventricular rather than left ventricular activity. The transition marked the beginning of phase R2s and its timing was variable. When it came late in phase L2 (Figs 7 and 8), very small volumes of blood were pumped from the right ventricle into the left aorta. The effect of this was to bring reversed flow in the aorta to an end somewhat earlier than would be the case when there was no right ventricular output. The crossover to forward flow occurred early in phase L3, before closure of the aortic valves (compare Fig. 8 with Figs 4 and 5). When the transition appeared earlier in phase L2, a correspondingly greater change was seen in the left aortic flow record (Fig. 9). Reversed flow now occurred only momentarily, if at all, and a more substantial forward component began with the opening of the left aortic valves. Peak pressure and forward flow occurred more or less synchronously in the left aorta, marking the beginning of phase R3s, some 25 ms later than that of phase L3. Peak pressures occurred later in the right ventricle than in the left, whether or not ejection to the left aorta occurred. Ejection came to an end simultaneously on both sides of the heart as right and left aortic valves closed simultaneously and phases R3s and L3 ended. In spite of the increased forward component, the total flow in the left aorta was still small, being only about 5 % of the total output of the right ventricle.

Fig. 9 shows one of the more marked spontaneous changes in pattern. The ventilator was running throughout the record as the regular oscillations in the pressure traces show. There were no changes in the level of anaesthesia or any other known variable. The aortic blood pressures were seen to fall and the left aortic flow pattern to change during routine recording at low paper speed. It is clear that the falling aortic pressures brought the left aorta into the range of pressure generated in the second stage of right ventricular contraction, making ejection possible. The immediate cause of the fall in aortic pressures must have been a sudden decrease in systemic resistance, though the reason for the change was unknown. The recorder speed was increased for a short period until the pressures started to rise and the flow pattern to revert to its more usual state. Most significantly, there were no changes in pulmonary pressures and flow accompanying these modifications. Changes were found, however, in flow pulses recorded from the right aorta, with peak systolic flows increasing and diastolic flows falling to lower levels, and these were also attributable to a sudden fall in systemic resistance. Recovery from this short period of right ventricular ejection was as rapid as its onset.

A number of experiments were carried out in attempts to reproduce this

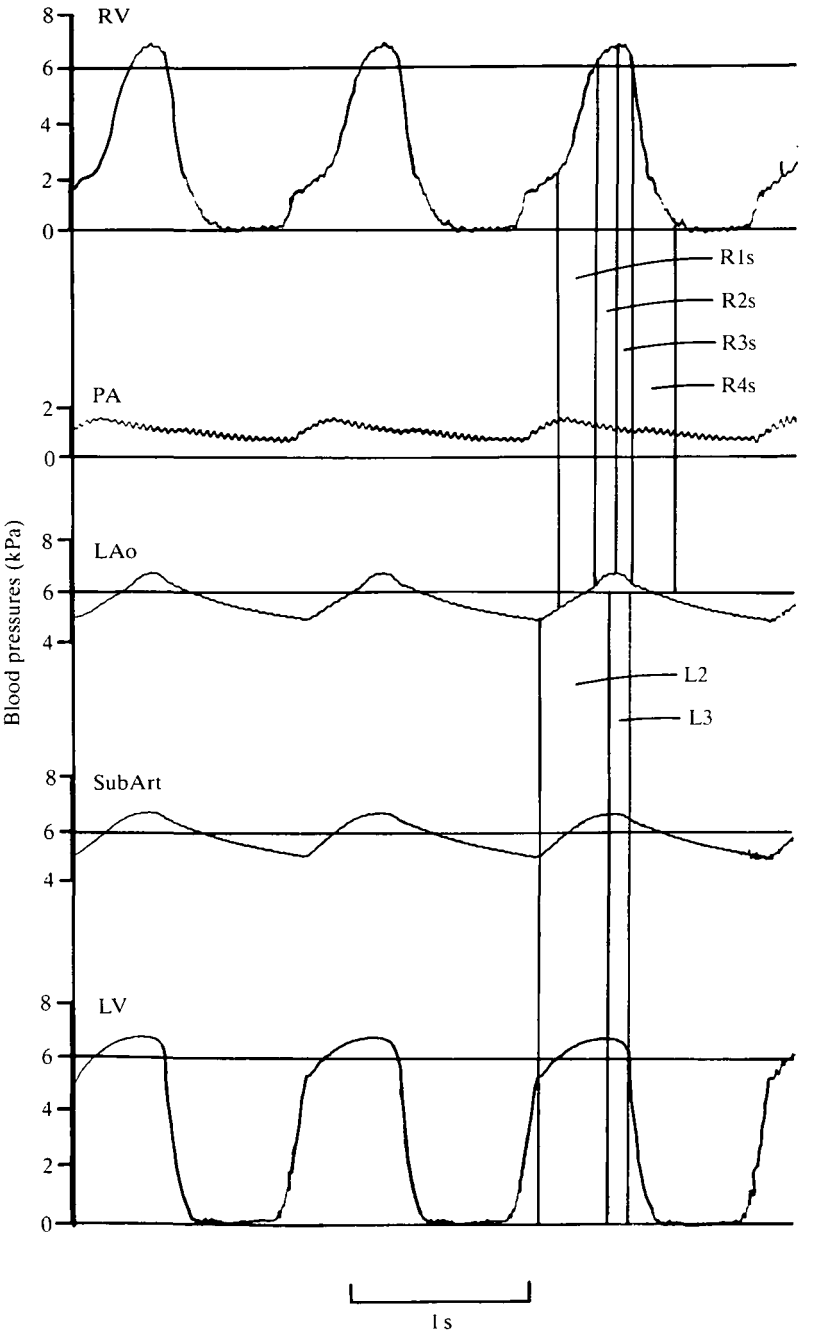

Fig. 7. Pressures recorded in the left ventricle (LV), right ventricle (RV), left aorta (LAo), subclavian artery (SubArt) and pulmonary artery (PA) of a 3.6 kg alligator. The vertical lines and numbered sections on the trace refer to phases in the right ventricle as they relate to the left aorta (R1s–R4s). Ejection into the left aorta occurs during phases R2s and R3s, as described in the text. L2 and L3 are phases in the left ventricle.

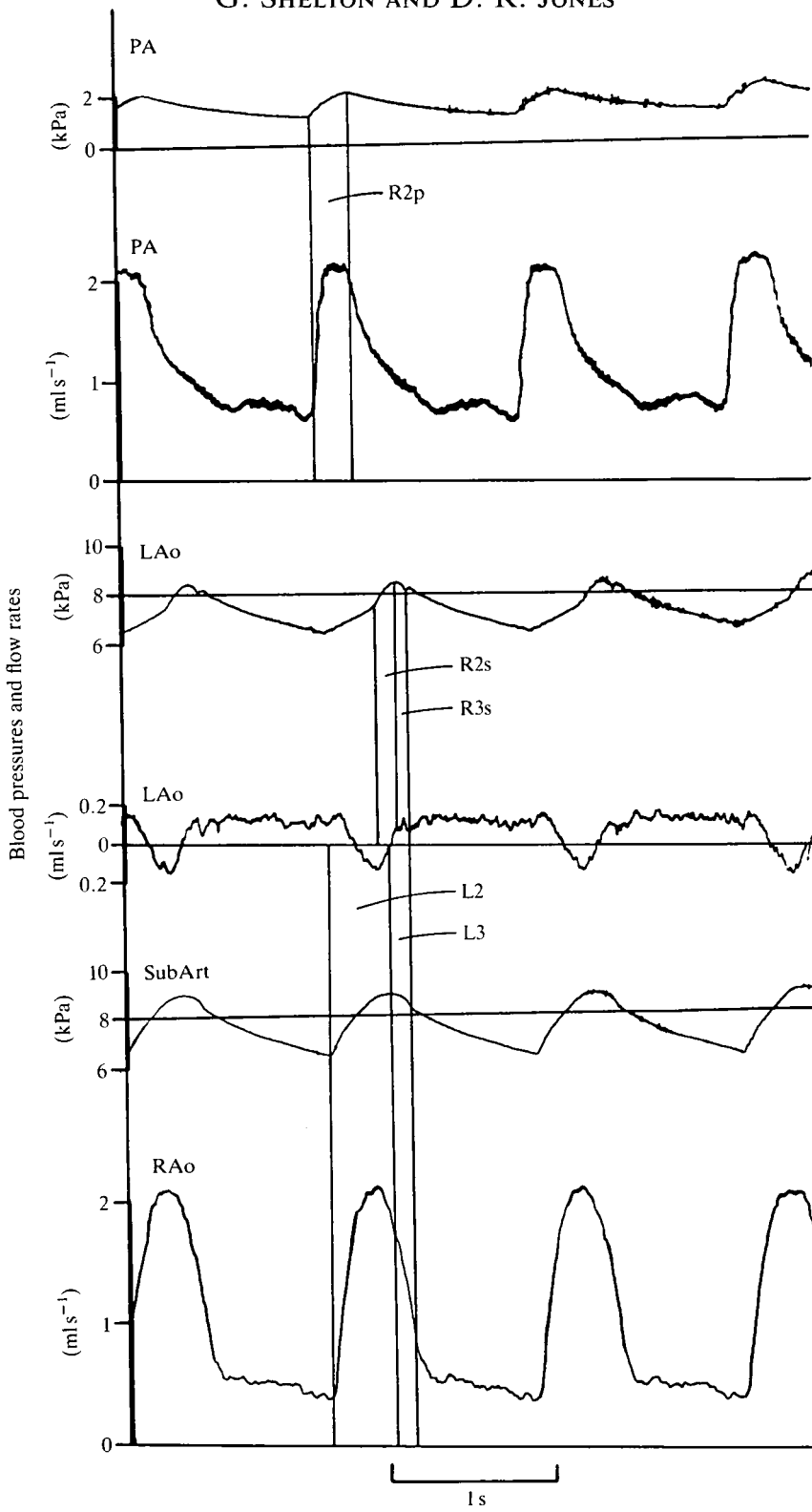
PA
(kPa)
2
0
R2p
PA
(ml s⁻¹)
2
1
0
LAo
(kPa)
10
8
6
R2s
R3s
LAo
(ml s⁻¹)
0.2
0
0.2
L2
L3
SubArt
(kPa)
10
8
6
RAo
(ml s⁻¹)
2
1
0
1 s
Blood pressures and flow rates

Fig. 8

Fig. 8. Pressures and flows recorded from the right aorta (RAo), subclavian artery (SubArt), left aorta (LAo) and pulmonary arteries (PA) of a 3.3 kg alligator. The right aorta and pulmonary flow rates were 56.8 ml min^{-1} and 72.2 ml min^{-1}, respectively, and the stroke flows were 1.19 ml $cycle^{-1}$ and 1.50 ml $cycle^{-1}$, respectively. Forward flow in the left aorta was 6.6 ml min^{-1} and reversed flow was 1.9 ml min^{-1}. Left aortic stroke flows were 0.14 ml $cycle^{-1}$ forward flow and 0.04 ml $cycle^{-1}$ reversed flow. The vertical lines and numbered sections show phases in the left ventricle (L2 and L3) and in the right ventricle as they relate to ejection into the left aorta (R2s and R3s) and the pulmonery arteries (R2p).

ejection to the left aorta. Immersion of the nostrils and head in water, to simulate the forced dives that White (1969) suggested were effective in causing left aortic flow and left-to-right shunt, did not result in a change in pattern. Although bradycardia was induced in some of these experiments, the systemic blood pressures were always higher than those generated in the second stage of right ventricular contraction. Switching the ventilator off for periods of up to 5 min was equally ineffective in causing ejection to the left aorta. Grigg (1989) has suggested, in a slightly different context, that low water temperatures give rise to low systemic blood pressures. We found that cooling the alligators from 30 to 22 °C had no effect on blood pressure and did not trigger ejection to the left aorta. The only experimental intervention that would reliably cause left aortic flow was the intravenous injection of acetylcholine. These experiments will be described in a later paper.

Discussion

The data presented in Table 2 are the first estimates of cardiac output in crocodiles. The values range from 14 to 41 ml min^{-1} kg^{-1} and are slightly, but not significantly, lower than the mean value of 49 ml min^{-1} kg^{-1} determined by Tucker (1966) for *Iguana iguana* between 20 and 30 °C, and of 44 ml min^{-1} kg^{-1} also for *Iguana* and measured over the same temperature range by Baker and White (1970). Both *Iguana* values were determined from oxygen consumption and arteriovenous differences in oxygen content. The blood pressures in Table 1 are not significantly different from those recorded by Grigg and Johansen (1987) in air-breathing *Crocodylus porosus*, but the systolic values are significantly higher than those found in the femoral artery of *Caiman crocodylus* by Axelsson *et al.* (1989).

The systemic circulation

The simultaneous recording of a number of pressure and flow traces makes it possible to interpret the events of the cardiac cycle in greater detail than has been done before. Pressure relationships between the left ventricle and right aorta are similar to those seen in mammals (McDonald, 1974; Noble, 1968), with a forward pressure gradient during phase L2 causing the rapid acceleration of blood flow to its peak value midway through the phase. Reversal of the gradient in phase L3 leads to flow deceleration and probably to passive valve closure due to local flow

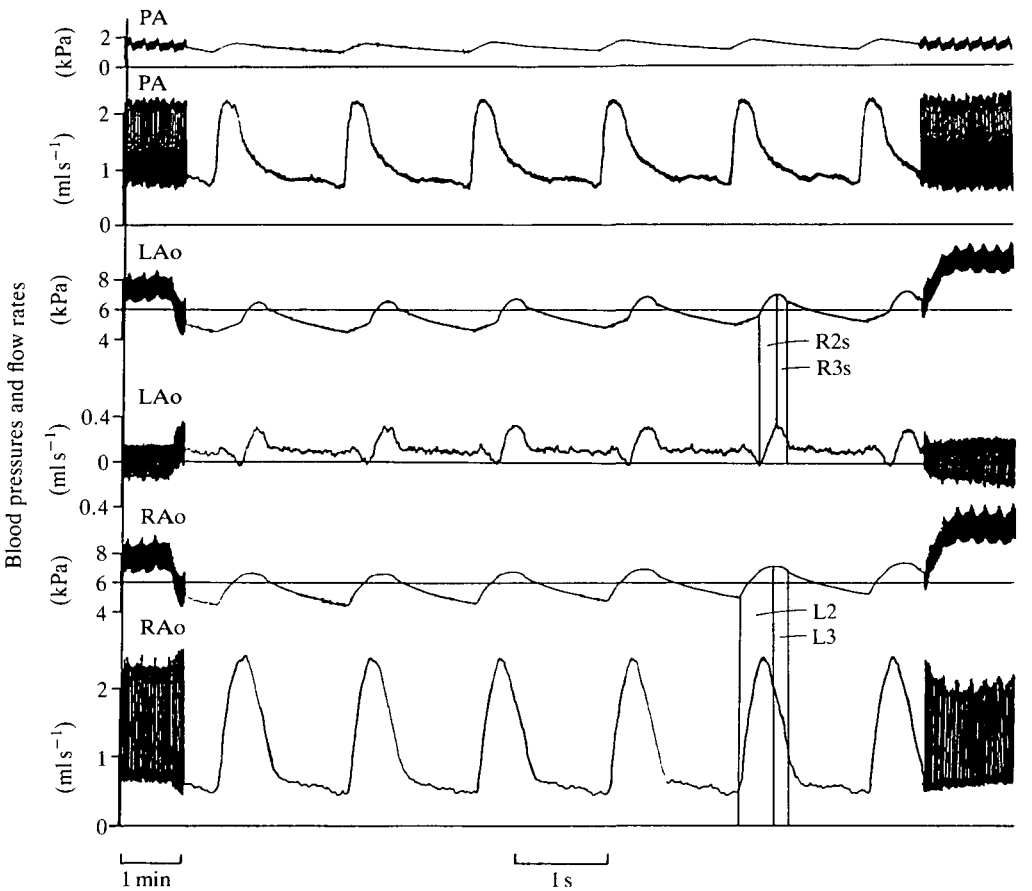

Fig. 9. Pressures and flows recorded in the right aorta (RAo), left aorta (LAo) and pulmonary artery of a 3.3 kg alligator, showing the development of right ventricular ejection to the left aorta. The right aorta and pulmonary flow rates were 62.0 ml min^{-1} and 68.2 ml min^{-1}, respectively and stroke flows were 1.38 ml $cycle^{-1}$ and 1.52 ml $cycle^{-1}$, respectively. Forward flow in the left aorta was 7.2 ml min^{-1} after right ventricular ejection had begun and stroke flows were 0.16 ml $cycle^{-1}$ in the forward direction. There were no measurable reversed flows when right ventricular ejection was well established.

reversal at the end of the phase. At the flow recording site some distance down the right aorta, continuous flow occurs throughout diastole because of the stored energy in the substantial central elastic reservoir. The right systemic artery expands into a large aortic sinus within the connective tissue truncus that binds all the aortic trunks together, so that the central reservoir is relatively large in volume. The left aorta does not expand into a large sinus in this way.

The pulmonary circulation

The relationships between the right ventricle, pulmonary arteries and left aor

are unique, as Greenfield and Morrow (1961) and White (1969, 1970) first revealed with their pressure measurements on *Alligator mississipiensis*. Pulmonary flow begins early in the cardiac cycle, about 80 ms before flow from the left ventricle to the right aorta (Figs 4 and 5). This is due, as it is in mammals, to the fact that ejection starts at very low pressure in the pulmonary circuit. However, the delay between the beginning of ventricular systole and the appearance of flow and pressure pulses in the pulmonary artery is clearly greater than might be expected (Figs 2, 3 and 4). This delay, together with the substantial pressure gradient that continues to be apparent during the early stages of pulmonary ejection (phase R2p), must be due to the remarkable anatomy of the pulmonary outflow tract. A subpulmonary chamber is marked off from the rest of the right ventricle, extending as far as the pulmonary valves. It probably represents an evolutionary remnant of the conus arteriosus. It has a contractile wall with a lining of dense fibrous nodules projecting into and largely occluding the outflow tract (Webb, 1979; van Mierop and Kutsche, 1985). These nodules appear to be able to fit together closely and may intermesh when the myocardium beneath them contracts. The evidence from pressure measurements suggests that some part of this outflow tract has to open suddenly after right ventricular systole has been in progress for about 80 ms (Fig. 2). Phase R2p then begins and flow accelerates in the pulmonary arteries. Even after opening in this way, the tract still contributes an appreciable component to the total resistance of the low-resistance lung circuit, judged by the persisting pressure gradient between right ventricle and pulmonary artery.

As phase R2p ends, the myocardium of the outflow tract begins to contract. Closure of this active valve causes the pressure gradient from ventricle to artery to increase rapidly. Pulmonary outflow must stop or slow substantially soon after peak arterial pressure is reached because, at this time, arterial flow begins a slow exponential decline in spite of the rapidly increasing pressure gradient. This fall in flow is much less abrupt than that leading to aortic valve closure in the right aorta, reflecting significant differences in the length and compliance of systemic and pulmonary circulations. There is an extensive pulmonary sinus distal to the outflow tract and to the arterial valves which acts as a substantial elastic reservoir.

White (1969) suggested that the resistance of the pulmonary outflow tract could be changed by a cholinergic mechanism so that the second stage of right ventricular systole produced higher pressures and ultimately outflow through the left aorta. If, as we believe, the active valve functions as an on–off mechanism so that pulmonary outflow stops or becomes extremely low during this second stage, such control of outflow resistance is impossible and cannot be important in regulating ventricular pressures and left aortic flow. The cholinergic mechanism could induce earlier closure of the pulmonary outflow tract or a tonic constriction of some sort, both of which would restrict pulmonary blood flow earlier in right ventricular systole and so leave more blood in the ventricle for the second stage. We have no evidence that either of these occurs. The relative timing of right and left ventricular events remains constant during systole (Figs 2, 3 and 7) and is not

affected by acetylcholine. In addition, there is no evidence of large changes in resistance of the open pathway from right ventricle to pulmonary artery as a necessary condition for left aortic outflow (Figs 7 and 9). Further experiments are being carried out to test the hypothesis that the outflow tract acts as an active on–off valve and not as a variable resistance controlling pulmonary flow over a wide range.

The additional small flow pulses that sometimes occur in the pulmonary artery are of some interest in this context. The earlier, more discrete, pulse (Fig. 5) is seen during isovolumic relaxation (phases L4, R4s) and is probably attributable to the beginning of relaxation in the cardiac muscle of the outflow tract. Right ventricular pressure is still high during the early stages of this phase (Fig. 2) and is sufficient to cause an outflow of blood into the pulmonary artery before closure of the pulmonary valves proper. The pulse, when it occurs, is always brief (10–15 % of the cardiac cycle) and shorter than phase 4. The second pulse (Figs 4, 5, 8 and 9) is much more difficult to explain since it occurs during phases L5 and R5 when ventricular pressure is below that in the pulmonary artery so that the passive pulmonary valves must be closed. It may be due to the transmural transfer of energy between compartments or vessels within the confines of the relatively inextensible pericardium and/or truncus.

Flow in the left aorta

Pressures in the left aorta are necessarily related fairly closely to those in the rest of the systemic circulation because of the connections *via* the foramen of Panizza and the dorsal aorta (Fig. 1). When pressures in the systemic circulation are high and left aortic valves closed, blood in the aorta moves slowly away from the heart (forward flow) during diastole and towards it (reversed flow) during systole. As diastole begins, closure of the right aortic valve causes the medial cusp to move away from the foramen of Panizza and so open it up for restricted flow from right to left aorta. Forward flow continues, with both the foramen and elastic recoil in the left aorta contributing, through to the next systole. This flow persists briefly as the foramen is closed by the opening right aortic valves, but ultimately reverses after the flow pulse completes the circuit from right to left aorta *via* the dorsal aortic connection.

During systole, reversed flow can only charge up the elastic reservoir of the left aorta since flow through the foramen is prevented by the cusp of the right aortic valve. This stored energy runs down during diastole. However, forward flow is greater than reversed flow and the additional flow must be due to movement of blood through the foramen. Mean foramen flow calculated on this basis is 0.022 ± 0.009 ml kg^{-1} cardiac $cycle^{-1}$ or approximately 0.95 ml min^{-1} kg^{-1} moving through the foramen. This amounts to only 2–6 % of the blood being pumped into the right aorta. The restricted aperture and obscured position of the foramen, together with the small pressure gradients from right to left sides during diastole, are consistent with this conclusion on the minor nature of foramen flow. It has very little significance in the overall cardiac cycle, except as a mechanism to preve

total stagnation of blood in the left aorta at times when the left aortic valves are closed. The slow flow of blood prevents clot formation and damage to the artery.

In one animal, forward flow in the left aorta occurred throughout the cardiac cycle, reaching a maximum during systole and a minimum with a momentary reversal as the aortic valves closed. The pattern must be due either to a failure of the cusp of the semilunar valve to close the foramen or to a transmural transmission of energy between aortic trunks during systole. The former seems more likely.

The pressure differences that develop between right and left aortae when the foramen is closed by the right aortic cusp can be quite large under some circumstances (Fig. 6). The beginning of diastole is marked then by a jump in pressure in the left aorta as the foramen opens and the pressure differences between the two aortae become very small. This was called the foramen spike by Grigg and Johansen (1987), who suggested that the variability in the spike size depended on fairly rapid, controlled changes in the diameter of the foramen. They dismissed the possibility of pressure wave transmission around the loop from right to left aorta *via* the dorsal aortic connection. Our evidence suggests that these hypotheses are incorrect. The size of the spike depends on the pressure difference that develops between right and left aortae during systole, since diastolic pressures are always close to one another through the open foramen. In turn, when there is no ejection from the right ventricle to the left aorta, the difference in systolic pressures in the two aortae depends on the leakiness of the cusp closure of the foramen and on the overall resistance to flow in the right-to-left loop. In most cases the foramen closes completely so that, after the initial forward movement of blood accompanying the cusp closure (Figs 4 and 5), the further increase in pressure in the left aorta is due entirely to reversed flow round the loop. If there is an appreciable resistance to this flow, then the rate of pressure increase at the left aortic cannula will be low and a large spike will ensue (Fig. 6) when the right aortic valve closes and the foramen opens. Conversely, with low loop resistance, only a small pressure difference, due principally to time delays in the loop, will be developed and the spike will be non-existent (Fig. 4) or seen as a small notch (Fig. 3).

Care is needed to avoid introducing resistance into the loop inadvertently. A short length of 1.22 mm tubing inserted into the left aorta, with its aperture facing towards the heart, was sufficient to produce the spike in Fig. 6. The possibility that the resistance of the loop may be controlled in some way cannot be eliminated. An obvious region for such control is the connection between the left aorta and the dorsal aorta (Fig. 1), which most authors refer to as small or minor (Webb, 1979; van Mierop and Kutsche, 1985). The vessel is inaccessible and we have not seen it in a living animal. In dead animals it is undoubtedly smaller than the vessels it connects but, nevertheless, appears to be a substantial connection that would not constitute a high resistance to flow. There is no direct evidence of mechanisms that might control its diameter, but the possibility is worth examining.

Flow from the right ventricle into the left aorta changes relationships in a major

way but, before discussing the new patterns, it will be useful to consider the conditions that could give rise to this type of right ventricular output. Usually in these preparations, the total output of the right ventricle is pumped into the pulmonary circuit in the first stage of systole. When the active valve closes and the second stage of systole begins, the pressures that can then be generated are not as high as those in the left ventricle. This is not due to the right ventricular myocardium being less powerful than the left, as it is in the varanid lizards; both left and right chambers have thick walls in alligators. The relatively low pressures reached must be the result of low ventricular volume and short muscle fibre length, together with declining levels of activation.

There are four ways in which the pressures in the right ventricle could be made to equal or exceed those in the left: (1) increasing the contractility of the right myocardium selectively, for example *via* the sympathetic innervation; (2) increasing the length of the right ventricular fibres by increased filling; (3) decreasing blood flow to the lungs by pulmonary vasoconstriction; (4) reducing left ventricular and systemic pressures by systemic vasodilation. The first hypothesis has never been investigated in alligators. Nor has the second, though it must have some importance because, other things being equal, consistent right ventricular output to the systemic circulation must increase venous return to the right side of the heart. The third mechanism is of known importance. The resistance of the pulmonary circuit has been shown to increase during diving (White, 1969) and after intravenous injections of acetylcholine (White, 1969; Axelsson *et al.* 1989), causing a right-to-left shunt. The precise site of the resistance change is not clear, though White (1969) suggested that the outflow tract was important. We think that there are difficulties with this hypothesis, as outlined earlier, and we shall be describing the properties of the pulmonary circulation in a later paper.

The fourth possibility, reduction of systemic blood pressure, is clearly of major importance in giving rise to left aortic flow. It is the most significant factor established in the present experiments and involves no obvious changes in right ventricular contractility or modifications in resistance of the pulmonary circuit. The lower the systemic pressure, the earlier in the cycle does the second-stage pressure in the right ventricle reach levels high enough to open the left aortic valve (phase R2s) and establish flow (Figs 7, 8 and 9). However, no matter how low the systemic pressure, flow cannot be set up before the active closure of the pulmonary outflow tract has occurred and the second stage of right ventricular systole has become well established. Consequently, the early stages of left aortic flow are as before, with a short pulse of forward flow as the right aortic cusp moves to close the foramen, followed by an equally short period of reversed flow round the right-to-left loop (Figs 8 and 9). The reversed flow is quickly ended by the ejection of blood through the left aortic valves (Fig. 9) when right ventricular pressure reaches that in the left aorta. This pressure is often lower than that in the right aorta because of losses round the loop. The pressure difference can be sustained at the central ends of the aortae, as blood flows down them from their respective ventricles, because the foramen is now closed by both right and left aortic valve

There will be some interesting consequences of such differences at the dorsal anastomosis. In fact, we have evidence that pressure gradients can be set up in both directions. This suggests that flow can occur from right aorta to coeliac artery and from left aorta to dorsal aorta. Dynamic effects would favour the latter because of the oblique connections of the anastomosis (Fig. 1), but there can be no doubt that adjustments occur in both directions. The evidence does not support the suggestion that the left aorta supplies almost all of the blood to the stomach and intestines or that blood from that vessel does not get into the systemic circuit (Webb, 1979).

The experiments described in this paper provide the basis for several new hypotheses about the relationships between the right and left sides of the alligator heart and their respective arterial arches. The conditions for right ventricular ejection to the left aorta are defined and one, the reduction of systemic blood pressure, is shown to be of considerable importance. It leads to increased cardiac output as the right ventricle begins to pump more blood than the left one, whose output, in turn, may stay constant or may increase as well. The other well-established condition for ejection to the left aorta is that of vasoconstriction in the pulmonary circuit. This will lead to reduced cardiac output, since flow round the pulmonary circuit, and therefore venous return to the left heart, must be reduced. We shall describe these relationships in a subsequent paper. In both cases the right side of the heart pumps more blood than the left, but the physiological consequences in terms of lung perfusion and relationships in the systemic circulation are different. The alligator heart is a most versatile and intriguing organ and there are many questions yet to be answered about its physiology.

We are grateful to the Royal Society, London, to NATO and to NSERCC for financial support.

References

AXELSSON, M., HOLM, S. AND NILSSON, S. (1989). Flow dynamics of the crocodilian heart. *Am. J. Physiol.* **256**, R875–R879.

BAKER, L. A. AND WHITE, F. N. (1970). Redistribution of cardiac output in response to heating in *Iguana iguana*. *Comp. Biochem. Physiol.* **35**, 253–262.

BURGGREN, W. W. (1987). Form and function in reptilian circulations. *Am. Zool.* **27**, 2–19.

BURGGREN, W. W. AND JOHANSEN, K. (1982). Ventricular hemodynamics in the monitor lizard, *Varanus exanthematicus*: pulmonary and systemic pressure separation. *J. exp. Biol.* **96**, 343–354.

BUTLER, P. J. AND JONES, D. R. (1982). The comparative physiology of diving in vertebrates. In *Advances in Comparative Physiology and Biochemistry*, vol. 8 (ed. O. E. Lowenstein), pp. 179–364. London, New York: Academic Press.

GREENFIELD, L. J. AND MORROW, A. G. (1961). The cardiovascular hemodynamics of Crocodilia. *J. surg. Res.* **1**, 97–103.

GRIGG, G. (1989). The heart and patterns of cardiac outflow in Crocodilia. *Proc. Aust. physiol. pharmacol. Soc.* **20**, 43–57.

GRIGG, G. C. AND JOHANSEN, K. (1987). Cardiovascular dynamics in *Crocodylus porosus* breathing air and during voluntary aerobic dives. *J. comp. Physiol.* B **157**, 381–392.

HEISLER, N., NEUMANN, P. AND MALOIY, G. M. O. (1983). The mechanism of intracardiac shunting in the lizard *Varanus exanthematicus*. *J. exp. Biol.* **105**, 15–32.

McDONALD, D. A. (1974). *Blood Flow in Arteries.* London: Edward Arnold.

MILLARD, R. W. AND JOHANSEN, K. (1973). Ventricular outflow dynamics in the lizard, *Varanus niloticus*: responses to hypoxia, hypercarbia and diving. *J. exp. Biol.* **60**, 871–880.

NAIFEH, K. H., HUGGINS, S. E., HOFF, H. E., HUGG, T. W. AND NORTON, R. E. (1970). Respiratory patterns in crocodilian reptiles. *Respir. Physiol.* **9**, 31–42.

NOBLE, M. I. M. (1968). The contribution of blood momentum to left ventricular ejection in the dog. *Circulation Res.* **23**, 663–670.

SHELTON, G. (1985). Functional and evolutionary significance of cardiovascular shunts in the Amphibia. In *Cardiovascular Shunts: Phylogenetic, Ontogenetic and Clinical Aspects. Alfred Benzon Symp.* **21** (ed. K. Johansen and W. W. Burggren), pp. 100–120. Copenhagen: Munksgaard.

SHELTON, G. AND CROGHAN, P. C. (1988). Gas exchange and its control in non-steady-state systems: the consequences of evolution from water to air breathing in the vertebrates. *Can. J. Zool.* **66**, 109–123.

TUCKER, V. A. (1966). Oxygen transport by the circulatory system of the green iguana (*Iguana iguana*) at different body temperatures. *J. exp. Biol.* **44**, 77–92.

VAN MIEROP, L. H. S. AND KUTSCHE, L. M. (1985). Some aspects of comparative anatomy of the heart. In *Cardiovascular Shunts: Phylogenetic, Ontogenetic and Clinical Aspects. Alfred Benzon Symp.* **21**, (ed. K. Johansen and W. W. Burggren), pp. 38–56. Copenhagen: Munksgaard.

WEBB, G. J. W. (1979). Comparative cardiac anatomy of the Reptilia. III. The heart of crocodilians and an hypothesis on the completion of the interventricular septum of crocodilians and birds. *J. Morph.* **161**, 221–240.

WEBB, G. J. W., HEATWOLE, H. AND DE BAVAY, J. (1971). Comparative cardiac anatomy of the Reptilia. I. The chambers and septa of the varanid ventricle. *J. Morph.* **134**, 335–350.

WEBB, G. J. W., HEATWOLE, H. AND DE BAVAY, J. (1974). Comparative cardiac anatomy of the Reptilia. II. A critique of the literature on the Squamata and Rhynchocephalia. *J. Morph.* **142**, 1–20.

WHITE, F. N. (1956). Circulation in the reptilian heart (*Caiman sclerops*). *Anat. Rec.* **125**, 417–431.

WHITE, F. N. (1968). Functional anatomy of the heart of reptiles. *Am. Zool.* **8**, 211–219.

WHITE, F. N. (1969). Redistribution of cardiac output in the diving alligator. *Copeia* **3**, 567–570.

WHITE, F. N. (1970). Central vascular shunts and their control in reptiles. *Fedn Proc. Fedn Am. Socs exp. Biol.* **29**, 1149–1153.

WHITE, F. N. (1976). Circulation. In *Biology of the Reptilia*, vol. 5 (ed. C. Gans), pp. 275–334. London, New York: Academic Press.