Cardiac receptors in ducks: the effect of their stimulation and blockade on diving bradycardia

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JONES, DAVID R., WILLIAM K. MILSON, AND NIGEL H. WEST. *Cardiac receptors in ducks: the effect of their stimulation and blockade on diving bradycardia.* Am. J. Physiol. 238 (Regulatory Integrative Comp. Physiol. 7): R50–R56, 1980.—It has been suggested that cardiac receptors, activated by the rise in cardiac filling pressure during diving, make a crucial contribution to diving bradycardia in the duck (Blix et al., *Acta Physiol. Scand.* 97: 13–19, 1976). The effects of receptor stimulation and blockade performed pharmacologically by injecting nicotine and lidocaine, respectively, into the pericardial sac were presented as supporting evidence. We have confirmed that nicotine activates epicardial receptors causing transient bradycardia and hypotension and shown that sudden increases in left ventricular diastolic pressure (LVDP) during diving can enhance bradycardia. However, intrapericardial injection of 0.1% lidocaine that was sufficient to block the response of epicardial receptors to nicotine had no effect on diving bradycardia. Preventing LVDP rising, by continuous withdrawal of blood from the great veins during diving, also had no effect on either the initiation or maintenance of bradycardia. A 0.5% solution of lidocaine injected intrapericardially caused an immediate increase in heart rate even during vagal stimulation in a bilaterally vagotomized duck and, in intact ducks, considerably reduced diving bradycardia. We have failed to confirm previous evidence linking cardiac receptors to diving bradycardia and suggest that cardiac receptors are not normally involved in the cardiac chronotropic response to diving.

Anas platyrhynchos; left ventricular pressure; vagolytic drugs; ventricular receptors; epicardial receptors

IN MAMMALS, LEFT VENTRICULAR RECEPTORS (LVR) with nonmedullated vagal afferent axons are activated by an increase in left ventricular diastolic pressure (LVDP) or volume (LVDV) causing bradycardia and hypotension (20–22, 24, 26). However, strong chemoreceptor activation suppresses the hypotensive effect of stimulating LVRs although the induced bradycardia is unchanged or even enhanced (27). Diving birds and diving mammals exhibit prominent bradycardia during forced submergence and, because it is assumed that LVDP will increase in the same manner as recorded increases in central venous pressure (14, 16), it is possible that LVRs will be activated and contribute to the intensification of diving bradycardia. The hypotensive effect of LVR activation will be overruled by the concomitant strong excitation of arterial chemoreceptors during diving (4, 27).

Recently it has been shown that, although myocardial contraction is essential for receptor response, mammalian LVRs respond mainly to changes in end-diastolic volume (26). Furthermore, it has been shown that depression of ventricular contractility impairs the receptor response to pressure and volume loading (26). Because, in submerged ducks, left ventricular volume only increases moderately (1) while the heart is exposed to a considerable negative vagal inotropic effect (11, 12), it is possible that LVRs will not be stimulated during diving. In the only study to date Blix et al. (5) have claimed that cardiac receptors are important in intensifying the bradycardia that accompanies forced submergence in the duck. The effects of receptor stimulation and blockade were studied pharmacologically by injecting nicotine and lidocaine, respectively, into the pericardial sac. No attempt was made using physiological means to expose the reflex caused by activation of LVRs either before or during diving and the time course of the vagolytic action of lidocaine was not studied in detail. Consequently, we felt that the role of LVRs in causing or intensifying diving bradycardia in ducks deserved further investigation.

METHODS

Experiments were performed on 13 mallard ducks (*Anas platyrhynchos*) varying in mass from 1 to 2 kg, and 14 White Pekin ducks (*A. platyrhynchos*) varying in mass from 2.8 to 3.4 kg. All experiments were carried out at the temperatures at which the ducks were maintained (20–22°C). The adjectives "control" or "initial" when referring to any of the measured variables describes them before drug treatment or submergence. In the text and Figs. 1–3 numerical values, when referring to determinations of variables in a group of animals, are given as means ± SE of the mean of n observations on N animals. Groupings of data were compared using a t test and 5% was considered to be the fiducial limit of significant difference.

In the majority of experiments the animals were restrained, ventral side down, on an operating table with the head held pointing downwards into a large filter funnel. Submergence was effected by filling the funnel from a beaker of room temperature water after first clamping a piece of tubing attached to the spout. Removal of the clamp drained the funnel for emersion. All operative procedures for implantation of cannulas were of a superficial nature and were performed under local anesthesia (2% lidocaine). Furthermore, the area of any wounds was periodically infiltrated with local anesthetic

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throughout the course of an experiment.

To monitor arterial and venous blood pressures a sciatic artery and ulnar vein were exposed under local anesthesia and cannulated with polyethylene cannulas attached to Hewlett-Packard 267-BC or Bio-Tec BT-70 pressure transducers. The venous cannula was advanced from its position of insertion in the wing until its opening was close to the heart. In some experiments a second venous cannula was inserted into the other ulnar vein and advanced until its tip reached the vena cava. This cannula was used for injecting and withdrawing blood to alter central venous pressure. Left ventricular diastolic pressure was measured by advancing a cannula from the right brachial artery until, as judged from the pressure trace, its tip lay in the ventricle. In a few experiments the pulmonary vein was exposed by a small incision between two anterior ribs and cannulated to record pulmonary venous pressure. The cannulas, of various sizes depending on the size of the vessel, were filled with heparinized avian saline (40 IU·ml^{-1}). The frequency response of the manometric system with the smallest cannula used was 40 Hz with damping of 0.7 of critical.

Breathing was monitored either with a pneumotachograph attached to a tracheal cannula and the flow signal integrated to give tidal volume or by measuring the CO_2 and O_2 contents of tracheal gas with a respiratory mass spectrometer (MGA 200, Twentieth Century Electronics, Croydon, UK). The mass spectrometer sampled gas at about 10 ml·min^{-1} and was calibrated periodically with gases of accurately known composition. The tracheal cannula was inserted high in the neck after exposing the trachea under local anesthesia. Heart rate was obtained from the ECG, the latter being recorded with bipolar copper wire electrodes (7) and after amplification the signal was fed into an instantaneous heart rate meter to give pulse frequency.

All signals were amplified by conventional means and arterial, venous, and left ventricular diastolic blood pressures, breathing frequency or integrated tracheal air flow, the O_2 and CO_2 composition of the tracheal air, and the ECG or instantaneous heart rate, were displayed on a Techni-Rite TR888 eight-channel thermal pen writer, writing on rectilinear coordinates.

Catheters were implanted into the pericardial sac by a ventral approach. The animal was placed on its back and a small incision was made in the abdominal wall at the end of the sternum. The sternum was raised and a small hole was pierced in the pericardial wall with a hypodermic needle. Soft silicone catheters were passed through this hole and advanced so that the tip of one catheter came to lie dorsal, and the other ventral, to the ventricles. In some animals only the ventral catheter was inserted. The hole in the pericardium, around the catheters, was closed with tissue cement. The catheters were sewn to the body wall and the animal closed. One catheter was used for drug injection and the other to drain the pericardial sac. All solutions injected into the animals were warmed to 41°C before injection. Immediately before injection of any drug the pericardial cavity was drained of fluid to avoid dilution of the test solution. Nicotine tartrate, in concentrations of 10–200 μg·ml^{-1}, was injected intrapericardially to stimulate epicardial receptors and lidocaine solution (0.1–0.5%) was used to block the response to nicotine tartrate. The systemic arterial baroreceptors in birds are located at the root of the aorta (16) and to ensure that they were unaffected by blockade of the nicotine response with 0.1% lidocaine intrapericardially, the cardiac chronotropic response during an elevation in arterial blood pressure was monitored before and after lidocaine treatment. The increase in arterial blood pressure was achieved by intra-arterial injection of epinephrine (2 mg·kg^{-1}).

For vagal stimulation, section, or blockade, both vagi were exposed high in the neck using a dorsal approach. The vagi were cooled by placing them on silver plates through which iced water flowed. Vagal section, when performed, was done distal to the cooling plate. There was no difference between the cardiovascular and respiratory responses to bilateral vagal cooling or bilateral vagal section. The distal side of a cooled or distal end of a cut vagus was stimulated using silver wire stimulating electrodes connected to a stimulator (Grass Instruments, Quincy, MA). During bilateral cooling or after bilateral section of vagi the animal was artificially ventilated using a Harvard positive-pressure respirator (Harvard Apparatus, Millis, MA).

RESULTS

Pharmacologic exposure and blockade of epicardial receptors and the effect of blockade on the cardiac chronotropic response to diving. Nicotine tartrate, injected into the pericardial cavity, caused immediate bradycardia and hypotension (Fig. 1*F*). In mallard ducks bradycardia occurred with doses as low as 5 μg (concn, 10 μg·ml^{-1}), although in some White Pekin ducks doses as high as 100 μg (concn, 100 μg·ml^{-1}) were necessary for an obvious response. However, in some animals no response could be elicited by intrapericardial nicotine and in two instances this was positively attributed to loculation of the pericardium, confining delivery to a small area of the heart. In ducks, as in dogs (24), the response to nicotine declined rapidly if it was elicited repeatedly with only a short time (1–5 min) between injections. The diminution in response occurred despite washing out the pericardial sac with saline between injections. Recovery periods of 10–30 min between injections gave consistent responses to nicotine injections. Injection of similar volumes of saline alone caused slight bradycardia in the majority of mallards (Fig. 1*E*), but only in 2 of 14 White Pekin ducks. The effect of nicotine injection, after bilateral vagotomy, was assessed in mallards. Bilateral section or bilateral cooling of the vagi did not prevent the bradycardia and hypotension following nicotine injection regardless of the initial heart rate and blood pressure (Fig. 1, *G* and *H*) although the response to the same dose differed both qualitatively and quantitatively from that obtained when vagi were intact (Fig. 1*F*).

The characteristic reflexogenic response to intrapericardial nicotine was blocked 1 min after application of a 0.1% solution of lidocaine hydrochloride intrapericardially. This was the case even when the dose of nicotine injected after blockade was four times that used before blockade (in White Pekins up to 200 μg). However, injec-

tion of lidocaine into the pericardial cavity also caused an immediate increase in heart rate (within 5 s) in intact, and unilaterally vagotomized ducks, in all concentrations used (0.1–0.5%; Table 1). The rate of increase in heart rate and maximum heart rate attained were positively correlated to the concentration applied (Table 1). One minute after the introduction of intrapericardial lidocaine the response to nicotine was lost although unilateral vagal stimulation still caused a fall in heart rate. Before treatment with lidocaine, in both intact and unilaterally vagotomized ducks, and in one bilaterally vagotomized duck, heart rate fell to the same level for a given level of unilateral vagal stimulation. However, 1 min after lidocaine injection the amount by which heart rate fell during vagal stimulation was unchanged, but resting heart rates had increased so that heart rates during vagal stimulation could be as much as 30–90 beats·min^{-1} above those seen in the ducks before treatment. By 2–4 min, however, the vagolytic action of the highest lidocaine concentration (0.5%) was often complete so that vagal stimulation no longer reduced heart rate. This effect could be reversed by washing out the pericardial cavity with warm saline. Applying either 0.1 or 0.5% lidocaine to unilaterally vagotomized ducks during vagal stimulation caused similar changes in heart rate as in intact ducks so they have been grouped together for presentation in Table 1. The decrease in heart rate in response to an elevation in arterial blood pressure, caused by intra-arterial injection of epinephrine (2 mg·kg^{-1}), was unaffected by blockade of the nicotine response with 0.1% lidocaine intrapericardially (30 tests on four White Pekin ducks).

In 10 White Pekin ducks we investigated the effect of

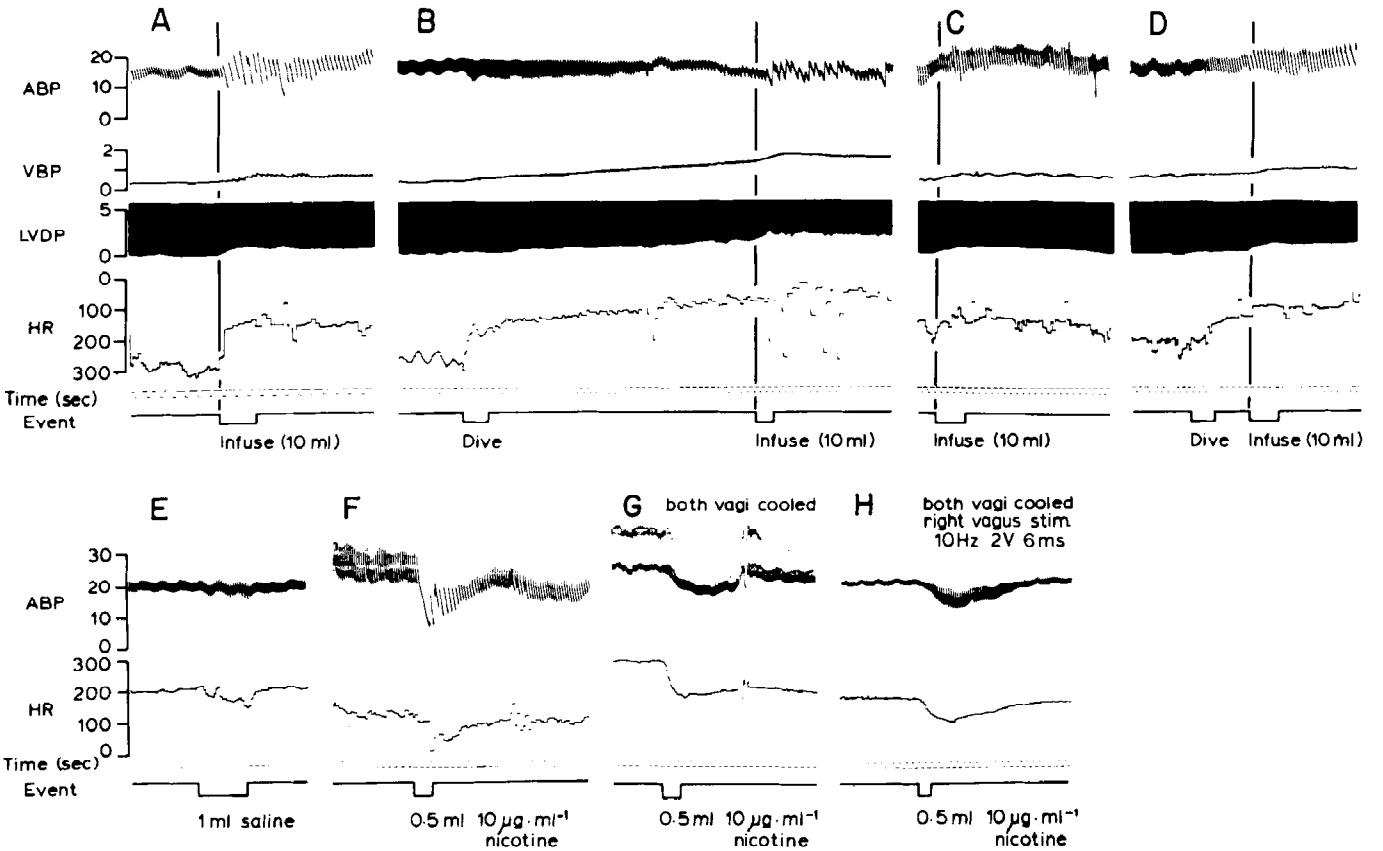

FIG. 1. Arterial blood pressure (ABP), venous blood pressure (VBP), left ventricular diastolic pressure (LVDP), and heart rate before, during, and after infusion of blood into central veins (event markers *A*-*D*) or saline or nicotine into pericardium (event markers *E*–*H*) under various conditions indicated. In *A*, *C*, *E*, and *F* animals are resting under control conditions. All pressures are expressed in kPa, heart rate in beats·min^{-1}.

TABLE 1. *Effect of intrapericardial injection of lidocaine solution on heart rate in mallard and White Pekin ducks*

Preinjection Heart Rate, beats·min^{-1}	*N*	Concn of Lidocaine, %	Dose of Lidocaine, mg	Heart Rate after Injection, beats·min^{-1}				
				5 s	10 s	20 s	30 s	50 s
184 ± 9 (11)	10*	0.1%	1.9 ± 0.09 (11)	176 ± 7.5 (9)	204 ± 8 (9)	214 ± 8 (9)	224 ± 11 (10)	236 ± 8.4 (11)
172 ± 15 (10)	9†	0.25–0.5%	7.3 ± 0.5 (10)	212 ± 15 (10)	242 ± 13 (10)	249 ± 14 (9)	256 ± 16 (8)	264 ± 23 (8)
200 (1)	1‡	0.5%	10 (1)	210 (1)	220 (1)	345 (1)	370 (1)	395 (1)

Values in parentheses record the number of trials on *N* number of animals contributing to the mean values ± SE presented. In unilateral and bilateral vagotomized ducks the lidocaine solution was injected during electrical stimulation of the distal end of the cut vagus. * Three unilaterally vagotomized. † Five unilaterally vagotomized. ‡ Bilaterally vagotomized.

blockade of epicardial receptors with either 0.1 or 0.5% lidocaine intrapericardially on the cardiac chronotropic response to forced submergence. The protocol of these experiments was similar to that described by Blix et al. (5). First, the response to intrapericardial nicotine was elicited by slow infusion of a nicotine tartrate solution of 100 $\mu g \cdot ml^{-1}$ concentration. Infusion was stopped as soon as the characteristic response to nicotine was obtained and the dose delivered noted (making an allowance for catheter dead space). Up to 2 ml of lidocaine solution was then injected intrapericardially, and removed again after 50 s or so when two to four times the previously effective dose of nicotine was injected into the pericardium. If there was no response to nicotine it was removed and replaced by the same lidocaine solution. The animal was submerged no more than 90 s after the first intrapericardial lidocaine injection. A 0.1% lidocaine solution, although successful in preventing the response to nicotine, had no effect on the average heart rate attained during the last 30 s of the dive (Table 2). However, after 0.5% lidocaine solution, the average heart rate during the last 30 s of the 1-min dive was significantly above the rate in normal dives or dives following 0.1% lidocaine treatment (Table 2).

Stimulation of cardiac receptors by changes in LVDP and the effects of changes in LVDP on the cardiac chronotropic response to diving. Bolus injection of quantities of blood from 5 to 30 ml into the central venous system caused sudden elevations in LVDP both before and during diving although the same volume injected in the dive gave three to four times the increase in LVDP as injection before submergence. Fourteen blood injections in three ducks at the surface resulted in bradycardia in nine (Fig. 1*A*) and no change in heart rate in five (Fig. 1*C*). However, on the average, the injections only increased LVDP by 0.34 ± 0.06 kPa which was only slightly more than a doubling of the preinjection level. In the 14 trials heart rate fell by 50 ± 23 beats·min^{-1} from the control rate of 295 ± 19 beats·min^{-1}. In all tests in which bradycardia was evoked systolic pressure rose by some 4 ± 1.4 kPa (n = 9) from a control value of 16.2 ± 1.3 kPa (n = 9). Diastolic pressure rose in seven of nine trials by, on average, 2.2 ± 1.4 kPa (n = 9) from a control level of 12.8 ± 1.1 kPa. Because, in one animal, the bradycardia accompanied hypertension when there was no change in LVDP whereas on three occasions in another duck there was no bradycardia when arterial blood pressure remained constant after blood injection, we felt that systemic arterial baroreceptors could be playing an important role in this response. During diving the absolute rise in LVDP after blood injection was 1.4 ± 0.25 kPa (n = 23), which caused bradycardia in 17 of 23 trials on four ducks (Fig. 1*B*). In the 17 positive trials heart rate fell by 34 ± 4.7 beats·min^{-1}, from an initial rate of 131 ± 9, while diastolic pressure fell by 1.1 ± 0.4 kPa and systolic pressure rose only slightly (by 1.3 ± 0.4 from 15.0 ± 1.0 kPa). In the six trials where bradycardia did not occur the changes in blood pressure were not significantly different from those in which bradycardia occurred, although the initial heart rate (71 ± 16 beats·min^{-1}) was significantly lower than in those animals displaying bradycardia (Fig. 1*D*).

TABLE 2. *Effect of intrapericardial lidocaine on heart rate immediately before diving and during last 30 s of a 60-s dive*

	Normal Dive	Dive after 0.1% Lidocaine	Dive after 0.5% Lidocaine
Initial heart rate, beats·min^{-1}	224 ± 12	193 ± 9.5	208 ± 14
Postlidocaine heart rate, beats·min^{-1}		232 ± 13	244 ± 14.5
Average heart rate during last 30 s of dive, beats·min^{-1}	44 ± 4	44 ± 5	152 ± 34
n	22	16	9
N	10	10	6

The dives commenced no later than 90 s after intrapericardial lidocaine. All values are means ± SE of n observations on N White Pekin ducks.

In a series of dives on mallard ducks, heart rate fell from 257 ± 12.6 to 47 ± 6.5 beats·min^{-1} after 60 s of submergence whereas mean arterial blood pressure remained near predive levels, falling from 28 ± 2.9 to 27 ± 3.1 kPa after 60 s submergence (26 dives on five ducks). In these experiments LVDP rose to a maximum value, after 60 s submergence, of 1.3 ± 0.1 kPa in 26 dives on five ducks, which was identical to the rise in central venous or pulmonary vein pressure (1.3 ± 0.1 kPa in six dives on four ducks). Continuous withdrawal of blood from the great veins during the first 20–30 s of submergence virtually eliminated the rise in LVDP although the onset of bradycardia was unaffected (Fig. 2, *B* and *C*) despite a more marked fall in mean arterial blood pressure of some 6.7 kPa in this dive (Fig. 2, *A* and *B*). The intensification of bradycardia in the later stages of the dive was totally independent of the level of LVDP (Fig. 2).

It was necessary to remove 30 to 40 ml of blood to prevent a rise in LVDP during a dive and it is possible that, due to the fall in blood pressure, the chemoreceptors may have been strongly stimulated causing bradycardia despite the constant LVDP. However, once bradycardia was established it was possible to reduce LVDP to zero or even to double or triple LVDP by withdrawal or injection respectively of no more than 5–10 ml of blood. Removing or injecting this volume of blood had no effect on arterial blood pressure (Fig. 3) and, in spite of extremely large changes in LVDP, there was no effect on heart rate (Fig. 3).

DISCUSSION

The present results show that, in ducks, activation of epicardial receptors by nicotine causes bradycardia and hypotension. In their reflexogenic effects avian epicardial receptors resemble those in mammals (24) and, as in mammals, are blocked by bathing the ventricles in 0.1% lidocaine. However, blockade of these receptors, with 0.1% lidocaine, has no effect on the level of bradycardia attained in the last 30 s of a 1-min dive. Stimulation of epicardial and, presumably, endocardial receptors by sudden increases in LVDP may also cause bradycardia

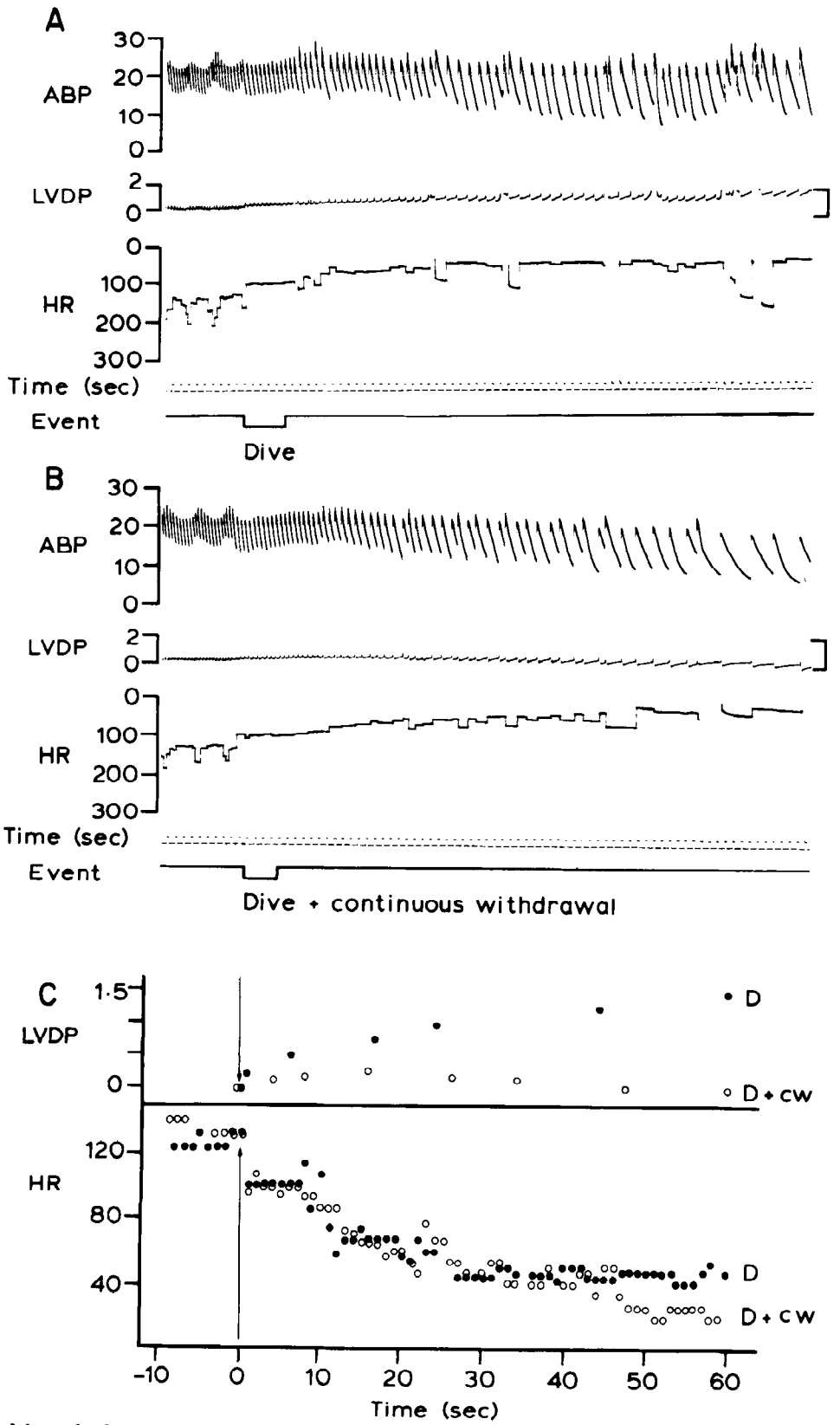

FIG. 2. *A*: changes in arterial blood pressure (ABP), left ventricular diastolic pressure (LVDP), and heart rate (HR) during normal diving. *B*: changes during diving accompanied by continuous withdrawal of blood from great veins to keep LVDP constant. *C*: graphic representation of changes in HR and LVDP with time following start of dive (*arrows*) in both *panel A*, (D), and *panel B*, (D + cw). All pressures are expressed in kPa, heart rate in beats·min^{-1}.

although the typical hypotension seen in mammals during this bradycardia is not often seen in ducks (20–22, 27). However, preventing LVDP from rising in a dive by continuous blood withdrawal from the great veins also has no effect on the cardiac chronotropic response to submergence. Obviously, the increase in LVDP during diving is the necessary consequence of bradycardia and not its cause. In the present experiments we have been unable to show a role for cardiac receptors in generating or maintaining bradycardia and believe that diving bradycardia in birds, at least in the later stages of a dive, is largely under the control of arterial chemoreceptors (13, 17, 18). Systemic arterial baroreceptors may also affect heart rate in a dive, especially when the barostatic reflex

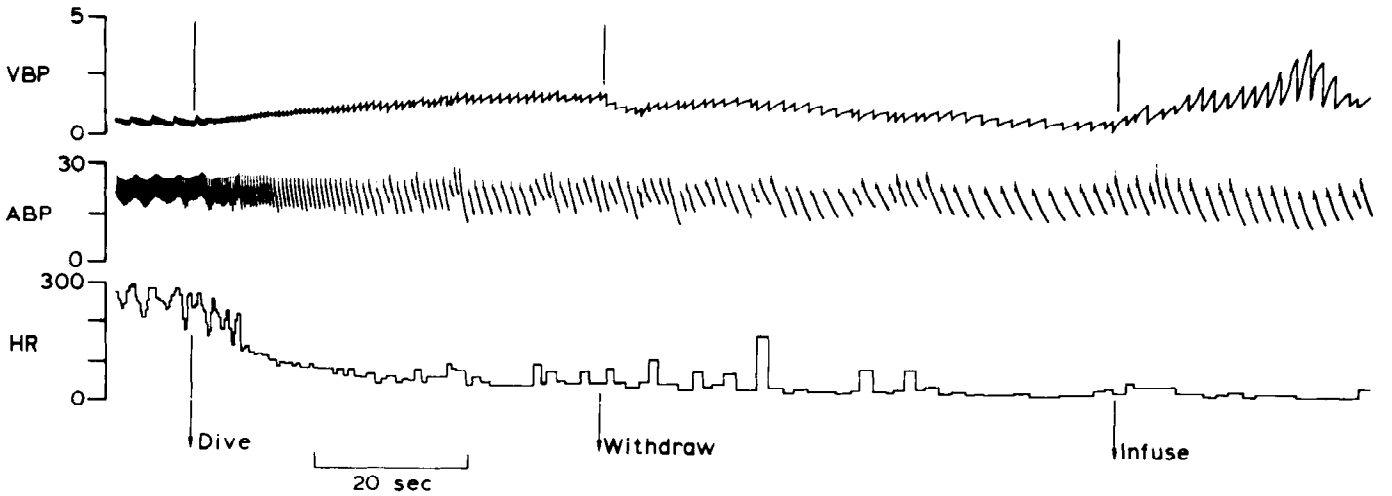

FIG. 3. Effect of changing central venous pressure, by withdrawing or injecting blood into central venous system, on heart rate pertaining during diving. Animal was submerged at *first arrow* and blood was withdrawn starting at *second arrow*, withdrawn blood was reinfused starting at *third arrow*. Traces from top to bottom: top, central venous pressure (kPa); middle, arterial blood pressure (kPa); bottom, heart rate in beats·min^{-1} (down on trace indicates fall in heart rate). *Time bar*, 20 s and refers to all traces.

is artificially activated (3), although their effect under normal circumstances appears to be much less than the effect of chemoreceptors (16, 18).

There is controversy concerning the nature of the sensory innervation of the atria and ventricles in birds. Most investigations have failed to find the specialized nerve endings that are present in mammalian hearts (2, 9, 25) although Bogusch (6) and Mathur and Mathur (19) identify spirally wound nerve fibers and knoblike nerve terminations that may be mechanoreceptors. However, virtually all histological studies of the avian heart have identified an extensive nerve net which, from analogy with the skin, is probably capable of signaling many different modalities of sensation (24, 28). Inasmuch as reptilian hearts also lack specific receptor structures their absence in birds would not be surprising, making the specialized nerve endings in mammalian hearts an obvious phylogenetic curiosity (2). Two electrophysiological studies have also presented evidence for the existence of a sensory receptor system in the avian heart (10, 15). Receptors recorded from by Estavillo and Burger (10) were sensitive to intravenous veratridine. The discharge characteristics of these receptors showed similarities to those recorded in mammals (8, 23), but the precise relation of the recorded discharge to the ECG was not established since conduction delays were not measured. As Thorén (26) points out, his recent observation that LVRs are activated mainly in systole contradicts earlier work with Öberg (20) in which an incorrect estimate of intracardiac nerve conduction time led them to conclude that left ventricular C fibers were mainly activated in diastole.

We have shown that activation of cardiac receptors by sudden increases in LVDP causes bradycardia in the majority of tests both predive and during diving. The bradycardia induced predive, however, could equally well have been caused by a barostatic reflex. Changes in cardiac contractility, which were not monitored in these experiments, may be the reason for the inconsistency we observed during diving. During diving cardiac contractility may be depressed due to a vagal negative inotropic effect (11, 12) and a depression of this type would account for the reduction in the response of LVRs to pressure and volume loading (26) and presumably also their reflex cardiovascular effects. It is significant that failure to elicit bradycardia by blood injection occurred when the heart rate was lower, and therefore exposed to greater vagal restraint, than earlier in dives when heart rate was high.

The present study does not give any support to the claim by Blix et al. (5) that LVRs are important for intensification of diving bradycardia. Pharmacologic blockade of epicardial receptors with 0.1% lidocaine solution had no effect on the intensity of bradycardia developed in the last 30 s of a 1-min dive. Furthermore, preventing the stimulation of epicardial and other cardiac receptors by keeping LVDP constant in a dive had no effect on initiation or maintenance of diving bradycardia. On the other hand, the amelioration of bradycardia after topical application of 0.5% lidocaine solution to the heart is caused by the vagolytic action of the local anesthetic and not to blockade by LVRs. We have shown that the same concentration of lidocaine, applied in a similar manner as by Blix et al. (5), has a vagolytic effect within seconds of its application. It is not possible to argue that the immediate increase in heart rate after 0.5% lidocaine is due to blockade of LVRs since, applying lidocaine during vagal stimulation in a bivagotomized duck caused heart rate to increase in the same manner as it did in intact or unilaterally vagotomized ducks, confirming that the effect is caused by anesthesia of the vagal efferent pathway and not LVRs. Lidocaine concentrations (0.1%) that were effective in blocking the response to nicotine within 50–60 s in the present study were similar to those found effective in dogs by Sleight (24). The increase in heart rate after blockade by 0.1% lidocaine could be due to either anesthetization of LVRs or a slight vagolytic action. However, Sleight (24) concluded that, in dogs, similar cardiac effects were not due to motor blockade. As pointed out by Blix et al. (5), even after the highest concentrations of lidocaine (0.5%) intrapericardially, the immediate vagolytic effects are not total and resting heart rate is only elevated by some 30–90 beats·min^{-1}.

However, in our experiments the threshold for cardiac inhibition was increased 60 s after 0.5% lidocaine since the same intensity of vagal stimulation, as was given before blockade, did not reduce the heart rate to the same level. Given that the vagus is the efferent pathway for diving bradycardia one would expect bradycardia, after treatment with these concentrations of lidocaine, to be less pronounced. Certainly, all five of the ducks subjected to "endoanesthesia of LVRs" by Blix et al. (5) had higher predive heart rates after treatment. In one case heart rate increased by 200 beats·min^{-1}, which surely attests to the vagolytic action of lidocaine in their experiments.

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