

THE GENESIS OF CERTAIN CARDIAC ARRHYTHMIAS IN FISH

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(Received 1 March 1968)

The hearts of both elasmobranch and teleost fish are known at times to beat at a particular phase of the respiratory cycle and thus to exhibit a regular rhythm locked to that of respiration (Lyon, 1926; Lutz, 1930; Satchell, 1960; Shelton & Randall, 1962). In the laboratory, the hearts of *Squalus acanthias* and *Mustelus antarcticus* usually exhibit a 1:2, 1:3 or 1:4 rhythm, the P wave of the electrocardiogram occurring as the mouth opens in the appropriate respiratory cycle. Lyon (1926) reported a 1:1 co-ordination in *Carcharias sp.* In *Squalus acanthias* it is known that respiration stimulates receptors in the pharynx, the afferent fibres of which pass up the branchial branches of the glossopharyngeal and vagus nerves to the medulla; the efferent limb of the reflex is the cardiac vagus nerve (Satchell, 1960).

In addition to this locked rhythm, electrocardiograms exhibit a variety of arrhythmias. Both the locking with respiration and these arrhythmias are abolished by atropine which blocks the vagal control of the heart, and by partial curarization which enfeebls respiratory movements to a level at which the pharyngeal receptors no longer fire. These observations suggest that the locked rhythm and these arrhythmias are related phenomena. This hypothesis has been investigated by constructing a mathematical model based upon established data of branchiocardiac co-ordination in fish. In this model varying proportions of the output of a 'respiratory' oscillator were allowed to modulate the output of a 'cardiac' oscillator. It will be shown that certain common arrhythmias of the fish heart beat can be explained as stages of incomplete coupling between the respiratory centre in the medulla and the cardiac pacemaker in the sinus venosus. Fish hearts are known to undergo some of the well-known arrhythmias of mammals; A-V block was reported by McWilliam (1885) and paroxysmal ventricular tachycardia has been noted in experiments involving manipulation of the heart. The arrhythmias described in this paper are of a different type as they do not involve any impairment in the co-ordination between the atrium and the ventricle but are arrhythmias of whole cardiac cycles.

MATERIAL AND METHODS

Trawled specimens of *Squalus acanthias* and *Mustelus antarcticus* were kept in a swimming bath until required, and fed weekly. They were secured in circulating sea water and restrained by two clamps to the dorsal fins. Sea-water temperatures varied from 8° to 18° C. but did not differ by more than 2° C. during any one experiment. The fish were not anaesthetized. The electrocardiogram was monitored from a single precordial lead consisting of an insulated needle inserted so as to lie with its tip within the pericardium close to the ventricle; signals were amplified and displayed on a

four-channel polygraph. Respiration was recorded from two fine brass contacts clipped into the upper and lower jaws. When the jaws closed, a 10 mV. pulse deflected the pen and registered that phase of the respiratory cycle. The data are drawn from thirty-five sets of traces, of which twenty-seven were from *Squalus acanthias*, seven were from *Mustelus antarcticus*, and one was from *Cephaloscyllium isobella*. The thirty-five traces were derived from a study of thirty-eight fish. Three *Squalus acanthias* showed no recognizable arrhythmia or locking and their traces have been excluded from the study.

THE MODEL

The heart of a fish is known to be subject to a powerful tonic vagal inhibition; it will be assumed that the cyclic modulation of this tonic activity exerted by respiration is a sinusoidal one. The assumption rests on the approximately sinusoidal wave form derived from strain-gauge records of the movement of the pharyngeal skeleton and of tensions developed in respiratory muscles (Satchell, 1960). The receptors that initiate the branchiocardiac reflex have not yet been identified but they are presumed to be fired by these cyclic respiratory movements.

No records have yet been published of the pacemaker potentials in the sinus venosus of a fish. However, such potentials in other vertebrates are alike in showing a steady rise towards a threshold level leading to the discharge of an action potential when this is achieved (Hoffman & Crane-field, 1960). The waveform of the pacemaker potential may be regarded as a positive-going sawtooth. Onto this, respiration, it is suggested, injects a sinusoidal increase and decrease of excitation that hastens or prolongs the instant at which the threshold is intersected. The site of this modulation, on this assumption, is the cardiac pace-maker.

The following formula enables the level of excitation achieved at any instant to be calculated:

$$Vt = At + B \sin(\omega t + \alpha),$$

where Vt = level of excitation at time t , At = level of sawtooth excitation at time t , A = units of excitation/sec., B = amplitude of injected sine wave (units of excitation), ωt = phase angle in radians of respiratory oscillator at time t , α = phase angle in radians of respiratory oscillator at zero time, $\omega t = (6.28 \times t)/P$, where P = period of respiratory oscillator in seconds.

A Fortran 1600 computer was programmed to calculate the instantaneous level of excitation at 0.1 sec. intervals, and to print out the time that had elapsed when an arbitrary level (90 units) of excitation had been achieved. This was regarded as the threshold. The phase angle of respiration at that instant was also printed and the term retained and used as α for the next cycle. Values of A were selected to span heart rates of from 8 to 30/min. and of P to span respiratory rates from 20 to 70/min. These correspond to rates encountered in *Squalus* and *Mustelus* under laboratory conditions. The value of B is unknown; it was assumed to have a range of values extending from zero up to one third of the threshold value (0-30 units of excitation). It may be supposed that it is related to the depth of respiration and that stronger respirations cause the receptors to fire more vigorously; this would increase the amplitude of B . The computer was allowed to run until it had 'set itself up' and the pattern of arrhythmia was clearly established.

ARRHYTHMIAS GENERATED BY THE COMPUTER

The arrhythmias generated by the computer were of three types and they represented stages in a transition from complete coupling, i.e. the locked rhythm, to the minimal degree of coupling that exerted any influence at all. Moderate coupling resulted in two arrhythmias which will be termed 'alternation' and 'arrhythmia of longer period, type 1'. Weak coupling caused a long-period arrhythmia which will be designated type 2.

THE LOCKED RHYTHM

If the natural period of the cardiac oscillator, set by the value of the threshold and of *A*, was slightly greater than or equal to some whole multiple of respiratory cycles, locking occurred and successive cardiac cycles were of the same length and terminated

Table 1. *The calculated duration and phase of successive cardiac cycles with increase of inherent heart rate (= A) and respiration held constant in depth (B = 10 units or 2 units) and rate (P = 1.1 sec.)*

(H.P. = Period of cardiac oscillator in seconds. H.R. = Heart rate/min. H.P./R.P. = ratio of period of cardiac oscillator to period of respiratory oscillator. C.L. = Cycle length in seconds. Ph = Phase angle in radians.)

A	Cardiac cycles														Type of coupling
	H.P.	H.R.	H.P./R.P.	1		2		3		4		5			
				C.L.	Ph	C.L.	Ph	C.L.	Ph	C.L.	Ph	C.L.	Ph		
<i>B = 10 units</i>															
18	5.0	12.0	4.5	5.17	6.14	4.60	0.99	4.52	1.71	5.17	6.14	4.60	0.99	Cycle of 3	
19	4.7	12.7	4.3	4.40	0.85	4.40	0.85	4.40	0.85	4.40	0.85	4.40	0.85	Lock 1:4	
20	4.5	13.3	4.1	4.40	0.28	4.40	0.28	4.40	0.28	4.40	0.28	4.40	0.28	Lock 1:4	
23	3.9	15.3	3.6	4.12	5.85	3.57	1.14	4.12	5.85	3.57	1.14	4.12	5.85	Alternation	
25	3.6	16.7	3.3	3.30	0.85	3.30	0.85	3.30	0.85	3.30	0.85	3.30	0.85	Lock 1:3	
30	3.0	20.0	2.7	2.75	2.28	2.95	0.28	3.17	5.85	3.25	5.56	3.27	5.42	Cycle of 5	
33	2.7	22.0	2.5	2.50	0.85	3.0	5.42	2.50	0.85	3.0	5.42	2.50	0.85	Alternation	
38	2.4	25.3	2.2	2.20	0.71	2.20	0.71	2.20	0.71	2.20	0.71	2.20	0.71	Lock 1:2	
40	2.25	26.7	2.0	2.20	0.28	2.20	0.28	2.20	0.28	2.20	0.28	2.20	0.28	Lock 1:2	
43	2.1	28.6	1.9	2.20	5.99	2.20	5.99	2.20	5.99	2.20	5.99	2.20	5.99	Lock 1:2	
<i>B = 2 units</i>															
30	3.0	20.0	2.7	2.95	0.85	3.05	5.71	3.10	4.56	3.0	2.85	2.95	0.85	Cycle of 4	

in the same phase of the respiratory cycle (Fig. 1). A decrease in the value of *A* from 25 to 19 units (Fig. 1, Table 1, *A* = 25, 19) was sufficient to prevent the uprising phase of the injected sine wave intersecting the threshold and the cycle was prolonged by the duration of one whole respiratory cycle. The locked condition was a relatively stable one; the stability was enhanced by increasing the amplitude of *B*. Small increases or decreases of respiratory period resulted in longer or shorter cardiac cycles terminating at the same phase. Small increases in the value of *A* also did not disturb the lock and (Table 1, *A* = 38, 40, 43) resulted in cycles which were the same length despite the inherent increase in rate but which terminated at earlier phases of the respiratory cycle. Larger changes in the value of *A*, and in particular a decrease of *A* such that it was less than some whole multiple of respiratory cycles, resulted in some arrhythmia. As this occurred an increase of *B* would restore the locked rhythm.

THE ALTERNATION ARRHYTHMIA

When there was a considerable disproportion between the periods of the two oscillators, Table 1, H.P./R.P. = 2.5, 3.6 (Fig. 2), cycles starting from a point on the positive half of the ascending slope of the sine wave ($\alpha = 0.157$ radians) terminated in the negative half of a later sine wave so that the cycle was prolonged. This in turn

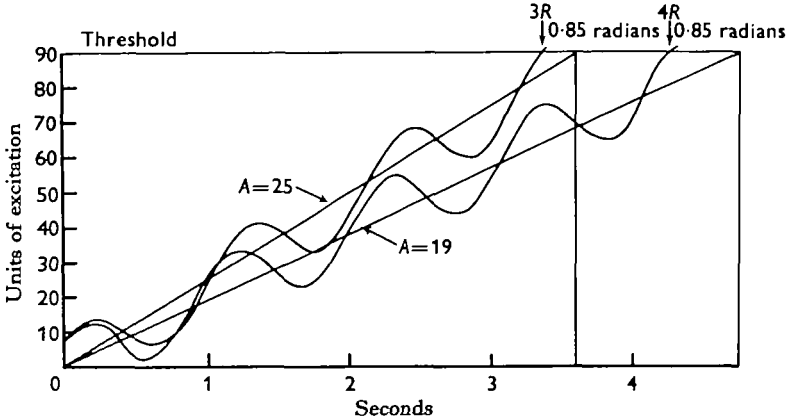


Fig. 1. The genesis of a 1:3 and a 1:4 locked rhythm. Threshold = 90 units of excitation; $B = 10$ units of excitation; $P = 1.1$ sec.; $A = 25$ units/sec. (upper trace), 19 units/sec. (lower trace); $R =$ respirations.

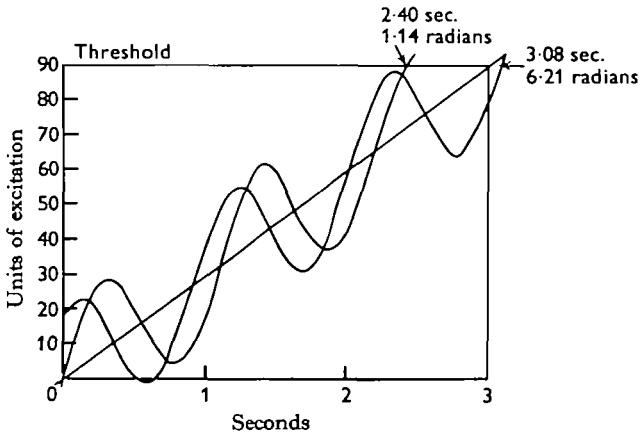


Fig. 2. The genesis of the alternation arrhythmia. Threshold = 90 units of excitation; $B = 20$ units of excitation; $P = 1.1$ sec.; $A = 30$ units/sec.

resulted in a cardiac cycle in which $\alpha = 4.51-6.28$ radians and which therefore terminated on the positive half of an ascending slope. There was thus an alteration of cycle length and of the phase angle of respiration at which the beat occurred. The short and long cardiac cycles both comprised the same whole number of respiratory cycles but differed by an interval less than one whole respiratory cycle, e.g. Table 1, $A = 23$, where the short and long cycles were equal to 3.2 and 3.7 respiratory cycles. Increasing B caused the alternation arrhythmia to be replaced by the locked rhythm.

ARRHYTHMIAS OF LONGER PERIOD, TYPE 1

With less disproportion between the periods of the two oscillators, and smaller values of B , the computer generated arrhythmias characterized by the regular occurrence of cycles that were either markedly longer or shorter than the others in the train. Short cycles were followed by cycles of increasing length; most of the increase occurred in the second cycle following the short one (Table 1, $A = 30$). Long cycles were followed by cycles of decreasing length; most of the decrease occurred in the second cycle following the long one. The pattern in which the periodic short cycle was followed by cycles of increasing length was observed when the length of the unmodulated cardiac cycle was slightly less than some whole multiple of respiratory cycles. The pattern in which the periodic beat was a long one occurred when the cardiac cycle was slightly longer than some whole multiple of respiratory cycles. Increasing the size of B tended to diminish the number of beats in the trains and ultimately to cause locking. In Table 1, $A = 30$, increasing B from 10 to 15 reduced the train from 5 to 3; when $B = 25$ a 1:2 lock occurred.

The characteristic feature of the type 1 arrhythmia was the repeated sudden transition in the length of the cardiac cycle. If the natural period of the cardiac oscillator was slightly less than some whole multiple of respiratory cycles, the terminal respiration in each successive cardiac cycle occurred progressively later. But, as can be seen from Fig. 2, the uprising part of the terminal sine wave cannot be indefinitely shifted to the right, thereby producing successively longer cardiac cycles, without the preceding sine wave intersecting the threshold. When this happens the sequence is ended by a shorter beat. The degree of the disproportion between the periods of the two oscillators decided the number of beats in the sequence; as the disproportion increased the sequence shortened. The extreme condition was thus that of the alternation arrhythmia in which only one long beat followed each short beat. Each successive long beat was initiated at an earlier phase of the respiratory cycle. In Table 1, $A = 30$, the last three beats fell back through the phases of the cycle behind. The occurrence of the short beat permitted the phase to advance once more as a portion of a respiratory cycle was omitted. This reset the oscillators relationship and a further sequence of long beats was initiated.

It can also be seen in Fig. 1 that there can be no cardiac cycle terminating or having its origin on the descending slope of the sine wave. There will be a region in each respiratory cycle between phase angles, 2.3–5.4 radians which cannot ever intersect the threshold without the corresponding ascending half of the sine wave having intersected it first. The precise phase angles that are sheltered in this way vary with the steepness of the slope of the sawtooth waveform.

ARRHYTHMIAS OF LONGER PERIOD, TYPE 2

When the value of B was reduced to a level such that no segment of the modulated waveform had a negative slope, a third type of arrhythmia developed. It consisted of sequences in which the length of the cardiac cycles successively increased and decreased. This occurred as regions of greater and of lesser positive slope in turn intersected the threshold. In Table 1, $B = 2$, a type 2 sequence containing four beats is

shown but longer sequences with more beats were generated if the disproportion between the periods of the two oscillators was less. The type 2 arrhythmia differed from type 1 in that the longest and the shortest cycles in a sequence were not adjacent but separated from each other by half a sequence. Only in this arrhythmia was it possible for cardiac cycles to be initiated in every phase of a respiratory cycle. As the value of B was increased there was an abrupt change from type 2 to type 1 arrhythmia as the modulated waveform acquired regions of negative slope where the rate at which the cardiac oscillator was accumulating excitation was less than the rate at which the decreasing half of the injected sine wave was subtracting it.

CARDIAC ARRHYTHMIAS RECORDED IN FISH
THE LOCKED RHYTHM

Amongst the thirty-five electrocardiograms recorded from three species of elasmobranch fish, ten (*Mustelus* 4, *Squalus* 6) exhibited a locked rhythm for more than fifty consecutive beats. The locked rhythm has also been frequently observed in the Port Jackson shark, *Heterodontus portusjacksoni*. The heart beat occurred at every second, third or fourth respiratory cycle (Fig. 3A) apart from the transient disturbances caused by coughing. In such traces a single beat at the next highest multiple of respiratory cycles was often included, suggesting a transient increase in vagal tone, comparable to the sudden change of A from 25 to 19 (Fig. 1). Conversely small changes in the respiratory period did not disturb the lock, the cardiac cycles changing their lengths

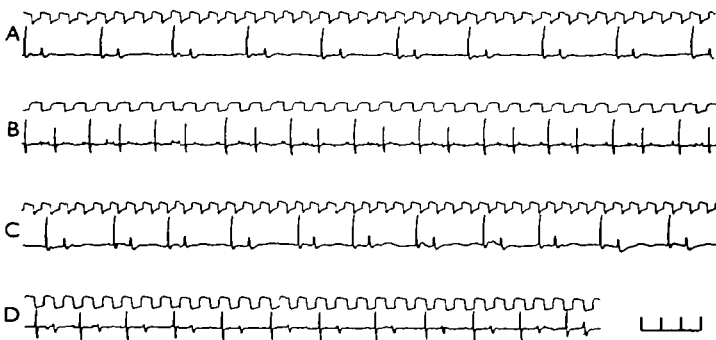


Fig. 3. A, a 1:4 locked rhythm. B, the alternation arrhythmia. C, arrhythmia of longer period, type 1; the trace shows three sequences of short, long, longer beats; the first sequence starts at the second QRS complex from the left. D, arrhythmia of longer period, type 2; the trace shows one sequence of beats with the shortest at each end and the longest in the middle; in each trace the upper pen recorded respiration with the downstroke signalling the closing of the mouth; the lower pen recorded the E.C.G. Time = seconds. A, B, C, *Mustelus antarcticus*; D, *Squalus acanthias*.

accordingly. The locked beats were initiated during the phase of the respiratory cycle just preceding and following mouth opening. In the analysis of these traces the respiratory cycle has been arbitrarily divided into ten equal phases numbered 1-10, commencing with the closing of the mouth. No attempt has been made to ascribe phase angles to these, related to those used in the model, as there is no precise knowledge of how the vagal tone changes with the respiratory cycle.

THE ALTERNATION ARRHYTHMIA

Alternation of long and short cardiac cycles that differed by less than the duration of one whole respiratory cycle occurred as a sporadic incident in many of the traces. Records from two specimens of *Mustelus* and one of *Squalus* exhibited unbroken runs of this arrhythmia (Fig. 3 B). There was an alternation of phase as well as of cardiac cycle length. Long cycles had their inception as the mouth opened; short cycles had their inception as the mouth closed. In Fig. 3 B the alternately tall and short recording of the QRS complex was due to this alternation of phase; the contraction of the pharynx when the mouth was closed pressed the electrode closer against the epicardial surface and caused a larger signal.

ARRHYTHMIAS OF LONGER PERIOD, TYPE I

Twenty-three of the thirty-five electrocardiograms exhibited sequences of this arrhythmia. They were derived from twenty specimens of *Squalus*, two of *Mustelus* and one of *Cephaloscyllium*. As with the arrhythmias generated by the computer, the longest

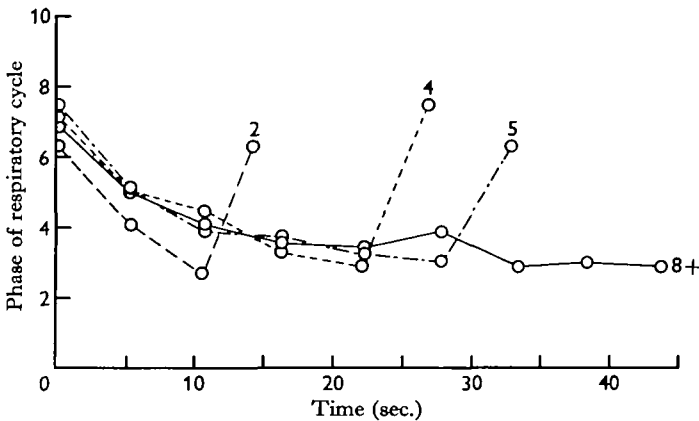


Fig. 4. *Squalus acanthias*. The change in phase and cycle length of the long cardiac cycles preceding a short cycle in sequences from a type I arrhythmia. Each set of points has been plotted from the averaged phase and length of ten sequences. The numerals indicate the number of long beats in the sequences. The 8+ trace includes some containing more than eight long beats.

and shortest cycles were adjacent to each other in the trace (Fig. 3 C). Fish electrocardiograms differed from the arrhythmias generated by the computer in that the number of beats in each sequence was seldom constant for more than four repetitions of it. The computer studies showed that small changes in the periods of either oscillator would alter the number of beats in a sequence. The failure of the fish arrhythmia to be as regular as that of the computer reflects perhaps the constantly fluctuating level of vagal tone in the fish. By grouping similar sequences together it was possible to analyse such traces. Sequences of the pattern, short, long, longer, longer, longest may be designated as S , L_1 , L_2 , L_3 and L_4 . By averaging the corresponding beats of ten such sequences it was possible to show that, as in the computer rhythms (Table 1, $A = 30$), the long beats of a sequence involve a loss of phase, successive beats falling back through the respiratory cycle, and the short beat advanced the phase by omitting

part of a respiratory cycle, (Fig. 4). Moreover, most of the change of phase occurred in the two beats following the short beat. Sequences containing more than four long beats were close to locking.

Statistical analysis of the lengths of cardiac cycles in these collected sequences demonstrated that S was shorter than L_4 ($P < 0.001$), that L_2 was longer than L_1 ($P < 0.01$) and that L_3 was longer than L_2 ($P < 0.05$). It was not possible to demonstrate that subsequent beats were significantly lengthened. The arrhythmias generated by the computer (Table 1, $A = 30$) showed that the increase between the last two beats was less than 1% of the cycle length and thus was presumably swamped in the fish arrhythmia by spontaneous changes in vagal tone.

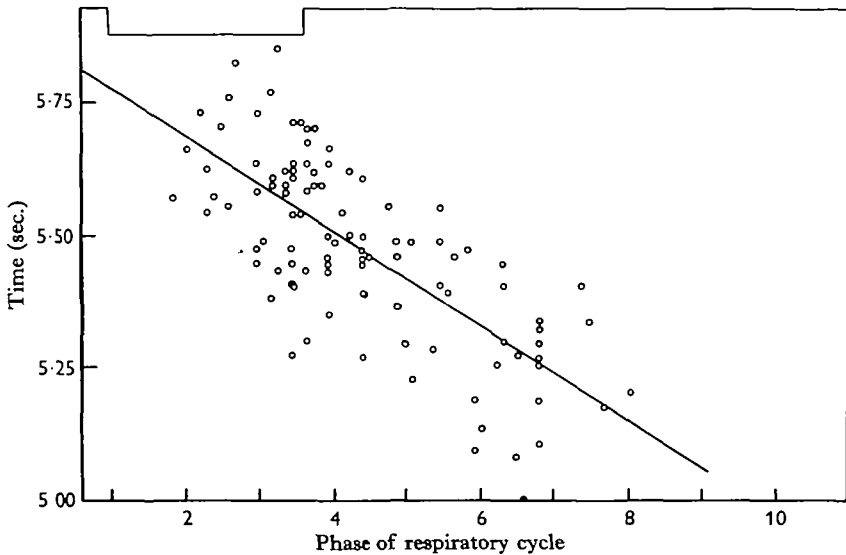


Fig. 5. *Squalus acanthias*. The regression of cardiac cycle length on the phase of a respiratory cycle in which each occurred for 202 long cardiac cycles from a trace of type 1 arrhythmia. The respiratory cycle is shown at the top of the graph.

The tendency for successive long beats in these type 1 sequences to be initiated progressively earlier in the respiratory cycle (Table 1, $A = 30$) implies that there should be a negative regression of cardiac cycle length on the phase of the respiratory cycle in which each beat has its origin. In Fig. 5 this regression has been plotted for 202 cardiac cycles that constituted the long beats of successive sequences in a type 1 arrhythmia. Though there is a considerable scatter the regression is significant ($P < 0.001$). It shows that the longest cardiac cycles were initiated close to the time of closing of the mouth and that the duration of a cardiac cycle can be predicted if the phase angle of respiration in which it was initiated is known.

ARRHYTHMIAS OF LONGER PERIOD, TYPE 2

Electrocardiograms from three specimens of *Squalus* exhibited sequences in which the duration of the cardiac cycle successively increased and decreased (Fig. 3 D). The longest cycle in the sequence was thus remote from the shortest cycle. Such sequences exhibited a cyclic waxing and waning of phase change as well as of cycle length.

In Fig. 6 the phase change of successive beats in a sequence is plotted and compared with that in a fish in which all coupling had been abolished by an injection of atropine. The pattern of phase change manifested by a fish with a locked rhythm is also shown. The forward march of the heart beat through the phases of the respiratory cycle was slowed as the cardiac cycles were transiently shortened, and speeded as they were prolonged. It is clear that the retardation of phase gain occurred at that phase of the respiratory cycle at which locking would have occurred if the amplitude of the injected sine wave had been increased.

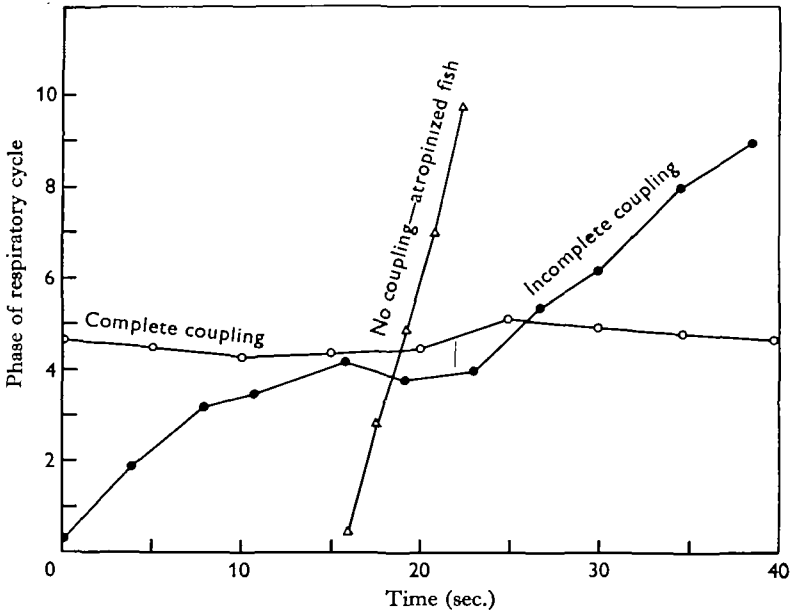


Fig. 6. The phase of the respiratory cycle in which successive heart beats occur. Each point has been derived by averaging the phase and duration of corresponding beats in three sequences. The trace labelled 'incomplete coupling' is derived from a trace showing type 2 arrhythmia. All three are from *Squalus acanthias*.

DISCUSSION

The model put forward in this paper may well be inexact; in particular it is conjectural that the injected modulation of the cardiac pacemaker potential has a sinusoidal form. A more abruptly rising waveform would constitute a more precise trigger and would bring about a similar series of locked beats and arrhythmias. In the field of electronic engineering it is recognized that a narrow sawtooth is the ideal waveform for oscillator locking (Terman & Pettit, 1952). Until we know more of the discharge patterns of the receptors that effect branchiocardiac co-ordination it is pointless to speculate further on the waveform of the injected signal. What has been established is that a model that is constructed to match the known and conjectured data of the circulatory and respiratory systems of fish generates the locked rhythm, and that, if the extent of the coupling between the two systems is reduced, it generates three patterns of arrhythmia that resemble those so commonly recorded in elasmobranch fish. This suggests that in such fish these arrhythmias have their genesis in this way.

A consequence of incomplete coupling in fish should be that during a change of heart-rate, periods in which the heart is locked to one multiple of respiratory cycle length should be separated from a lock at the next multiple by a sequence of arrhythmias as the mutual proportions between the cardiac and respiratory oscillators change. A trace of such a period of change of heart rate should differ from that of a mammal in the extent to which the lengths of successive cycles differ from a running average of their lengths. In Fig. 7 a running average based on ten heart beats depicts the change in heart rate of a specimen of *Squalus*. During this period of 23 min. the heart slowed

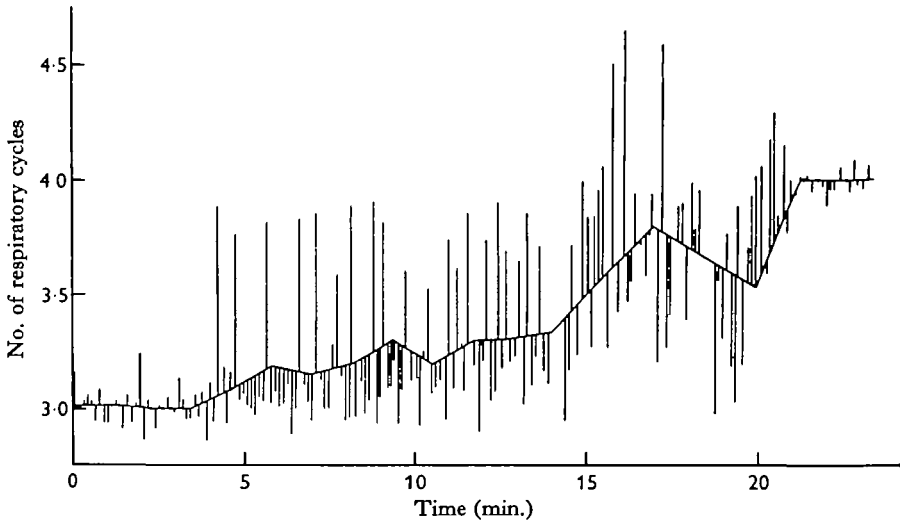


Fig. 7. The durations of successive cardiac cycles during a period of change of heart rate in *Squalus acanthias*. The graphed line is the average heart rate based on a running average of ten cycles. The vertical lines indicate the number of respiratory cycles spanned by each cardiac cycle.

from a 1:3 lock to a 1:4 lock. Each beat is indicated as a vertical line, the free end of which marks the number of respiratory cycles spanned. As the cardiac cycles became slightly longer than three respiratory cycles, after the 4th min., a type 1 arrhythmia with intercalated long beats was evident. Initially, when the disproportion was not great, some sequences with as many as seven short cycles occurred. As the cardiac cycle lengthened and approached 3.4-3.5 respiratory cycles, these sequences shortened and isolating alternating sequences occurred. Further lengthening of the cardiac cycle made it slightly shorter than four respiratory cycles, and after the 15th min. sequences of type 1 arrhythmia with intercalated short beats occurred. Following a transient increase of rate the heart slowed further and locked with a 1:4 rhythm. The model thus provides the means of interpreting what would otherwise seem a complex and irregular trace.

The study also showed that, in the model, diminishing the coupling increased the phase angle of respiration within which the heart could beat. Inspection of traces showed that this is true in fish but that even in the type 2 arrhythmia (Fig. 6) there was still a recognizable tendency for the heart to beat more frequently at the phase angle at which locking would occur if the coupling were increased. If it is true, as suggested

in an earlier paper (Satchell, 1960), that branchiocardiac co-ordination serves to ensure that the heart beats at the most effective part of the respiratory cycle, then even these partial couplings represent some increase in the efficiency of the haemorespiratory exchange system.

The cardiac arrhythmia of type 1 in fish shows certain resemblances to the Wenckebach type of arrhythmia in man, in which the P-R interval of the E.C.G. progressively lengthens in successive beats until the atrial impulse fails to initiate a ventricular contraction and a beat is missed. The next P-R interval is then a short one. The greatest increase in P-R interval occurs between the first and second beat of a train. The longest and shortest P-R intervals lie adjacent to each other in the series. The type 1 arrhythmia in fish shows a progressive increase in the length of whole cardiac cycles until the train is terminated by a short cycle, the equivalent of the dropped beat. The greatest increase in cardiac cycle length occurs between the two cycles following the short cycle.

Reversed Wenckebach periods have been described by Berman (1955) in which a train of beats exhibiting a 2:1 A-V block showed a progressive shortening of the P-R interval and terminated in a single 1:1 normal beat. Most of the decrease of the P-R interval was achieved between the first and second beats of the train. This recalls the type 1 arrhythmia of fish in which a train of cardiac cycles exhibit a successive shortening terminated by a single longer beat.

The Wenckebach arrhythmias have their genesis in the impairment of coupling between two oscillators, the sinoatrial and atrio-ventricular nodes. The nature of the cyclic change at the A-V node remains in dispute. Grant (1956) postulates a change in the excitability of the nodal pacemaker. Hoffman & Cranefield (1960), on the basis of microelectrode recordings of nodal fibres, postulate cyclic changes in the amplitude of the action potentials generated at the atrionodal junction. Decherd & Ruskin (1946) discussed cycles of recovery of excitability following the previous discharge. Whatever the nature of the second oscillator, the interaction is believed to be between two parts of the heart that normally undergo cyclic changes in excitability.

A less well known arrhythmia in man offers a further parallel. Scherf & Schott (1953) describe an unusual type of *bigeminus* rhythm in which an ectopic pacemaker in the ventricle initiated a ventricular beat that alternated with a normal beat initiated at the s.a. node. The interval between the normal and the ectopic beat lengthened progressively until an ectopic beat was dropped. They also described a variant of this arrhythmia in which the intervals shortened successively. Again the two oscillators both lay within the heart, one being the s.a. node and the other the ventricular ectopic focus.

In these human cardiac arrhythmias the period that undergoes the cyclic change of length is the time that elapses between the discharge of the first oscillator and the discharge of the second. The Wenckebach arrhythmia is thus primarily an arrhythmia of the P-R interval and only secondly an arrhythmia of cardiac cycles. The *bigeminus* arrhythmia described above is an arrhythmia of the interval between the normal and the ectopic beat. When the fish arrhythmias are compared with these they are seen to differ in two important respects. They are arrhythmias of whole cardiac cycles, because the oscillator interaction is between the respiratory rhythm generator in the medulla and the cardiac pacemaker. The link between the two oscillators is not therefore within the heart but outside it. It is a reflex involving receptors that are fired by

respiration and that relay in vagal afferent fibres to the medulla. The cardiac vagus is the efferent limb. In these anatomical relationships the fish arrhythmias resemble the normal sinus arrhythmia of mammals in which the heart beat speeds on inspiration and slows on expiration. Because the mammalian respiratory cycle is as long as three or four cardiac cycles it cannot add excitation to or subtract it from any particular heart beat but exerts its effect as a cyclic change in heart rate. In fish, where the respiratory cycle is shorter than or equal to the cardiac cycle, single respirations may trigger particular cardiac cycles. The respiratory rate is often approximately twice the heart rate, a proportion similar to that between the inherent rates of the S.A. and A-V nodes in the mammalian heart. The patterns of partial coupling between the beating of the pectoral and tail fins of teleost fish, described by Von Holst (1937), offer another example in which the coupling is mediated through the central nervous system. That the type 1 arrhythmias of fish resemble the Wenckebach arrhythmias rather than sinus arrhythmia of mammals emphasizes that it is the mutual proportions between the periods of the free and the dependent oscillator and the extent of their coupling that decides the pattern of the arrhythmia rather than the anatomical features of the coupling between them.

SUMMARY

1. The co-ordination of the heart beat with respiration in fish is treated as a problem in coupled oscillators and a mathematical model is developed in terms of the modulation of a sawtoothed waveform by an injected sine wave.
2. It is shown that reducing the coupling between the two oscillators replaces the locked rhythm by various arrhythmias that can be interpreted in terms of the mutual proportions of the periods of the two oscillators and the amplitude of the injected signal.
3. Electrocardiograms from three species of elasmobranch fish, *Mustelus antarcticus*, *Squalus acanthias* and *Cephaloscyllium isobella* are presented and the arrhythmias they show are compared with those generated by the model.
4. It is concluded that the resemblances between the cardiac arrhythmias in fish and in the model suggest that they have their genesis in partial coupling of the cardiac pacemaker with the respiratory rhythm generator.
5. The relation of the fish arrhythmias to the Wenckebach form of A-V block, and to certain variants of the *bigeminus* rhythm in man is discussed.

It is a pleasure to acknowledge my indebtedness to the National Heart Foundation of Australia who supported this project.

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