J. Exp. Biol. (1966), **45**, 31–44
With 11 *text-figures*
Printed in Great Britain

THE ACOUSTIC BEHAVIOUR OF THE BUSH CRICKET *PHOLIDOPTERA GRISEOAPTERA*

2. INTERACTION WITH ARTIFICIAL SOUND SIGNALS

By M. D. R. JONES

Department of Biology, Brunel College, London W. 3, and Department of Botany and Zoology, Sir John Cass College, London

(*Received* 25 *January* 1966)

INTRODUCTION

Analysis of recordings of alternation singing and synchronism in *Pholidoptera griseoaptera* (DeGeer) (Jones, 1966) has shown that mutual inhibition between singing males plays a major part in determining the timing of the chirps by each male. In addition it appeared possible that the interaction could have an excitatory effect in increasing the chirp rate of each male.

In this paper experiments with artificial signals are described, which give further evidence of the part played by the auditory input in the control of stridulation. A preliminary account of some of this work has been given by Jones (1963, 1964). Dumortier (1964) has reviewed previous work in this field.

MATERIALS AND METHODS

Male *Ph. griseoaptera* were caught, reared, housed and recorded as described previously (Jones, 1966). They were allowed to sing together except during experiments with single insects.

Production of artificial sound signals

Lorenz electrodynamic treble loudspeakers (LPH 65) were used. These were calibrated in an anechoic chamber using a previously calibrated Bruel and Kjaer condenser microphone (type 4131), cathode follower (type 2612) and audio frequency spectrometer (type 2112). The R.M.S. voltage across the loudspeaker terminals was measured with a Marconi valve voltmeter (TF 1100) and the output was calibrated in db. (rel. to 0·0002 dynes per sq. cm.), at a known distance (7 ft.) from the loudspeaker, for each volt across the loudspeaker terminals. The output was best in the range 2–20 kcyc./sec. although a detectable output was obtained up to 40–50 kcyc./sec.

In the first experiments the loudspeaker was driven directly by an Advance signal generator, and a morse key was used to make and break the signal. If a more gradual rise and decay of the signal was required, the signal was controlled with the volume control of the signal generator or by covering the loudspeaker with a soft cushion.

Fig. 1 is a diagram of the apparatus used in the later experiments to produce automatically, at regular intervals, signals of known length and with known rise and

decay times. The diode is biased so that it will conduct the signal only when the square-wave generator is producing a pulse of d.c. The modulation envelope of the signal is determined by the shape of the pulse from the square-wave generator and this can be modified by using an RC network as shown. The rise and decay times of the signal were controlled by altering the value of C. A tuned circuit was connected in parallel with the amplifier input. This reduced distortion of the signal and by-passed any unwanted frequencies.

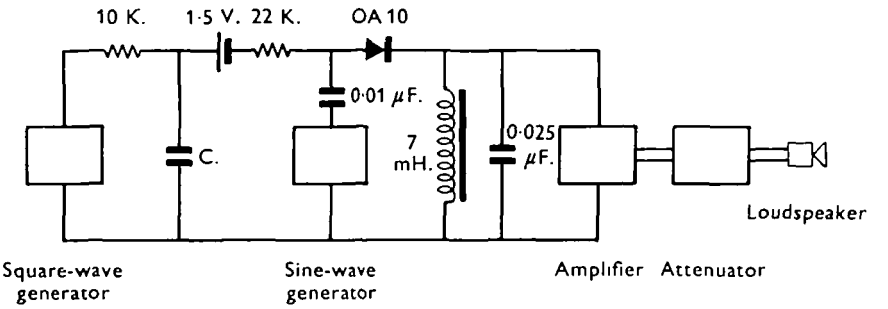

Fig. 1. Diagram of circuit used to produce short artificial signals at regular intervals. For details see text.

The square-wave generator could be set so that signals of known length of up to 100 msec. were produced at selected regular intervals. Longer signals were made manually by replacing the square-wave generator with a battery and a switch. The amplifier was a 3 W. transistor amplifier built from a Mullard circuit, and the output was connected through a variable attenuator so that the speaker output could be decreased 30 db. in 10 db. steps. Before any experiment the amplifier output was adjusted to give the required R.M.S. voltage across the speaker when the signal was switched on. If a signal below 70 db. was to be used, the apparatus was set up for 70 db. and the signal was attenuated to the required level. With this apparatus, using a frequency of 12 kcyc./sec, the maximum output was 80 db. at 7 ft. For greater output a more powerful amplifier would be needed.

Experimental technique

Most of the earlier experiments and all of the later ones were monitored using a Brenell Mk 5 2-channel tape recorder. The signal could be recorded directly on the second channel of the recorder as well as being monitored by the microphone which was placed near the insect. In all the later experiments the insect, microphone and loudspeaker were placed in one room, and the signal-producing equipment and recorder in another. The insect was placed 7 ft. from the loudspeaker and was allowed several hours to settle down in the darkened room before the experiment was started Before the first signals were played to the insect, 5–10 min. of 'basal' chirping was recorded. An experiment continued usually for about an hour, which comprised a number of periods with signals separated by 'rest' periods of silence.

The tape recordings were used to make paper chronograph records as previously described (Jones, 1966). It was simple to discriminate between chirps and signals in the record; often the tape recorder outputs could be adjusted so that only chirps

appeared in the trace from channel 1 and signals in the trace from channel 2. Measurements of the chirp rate and of the intervals between the signals and the chirps were made from these records. The intervals were measured to the nearest 0·1 sec.

Signal frequencies of 12 or 15 kcyc./sec were used since preliminary experiments had shown that the insects responded to frequencies in this range. In most of the later experiments 12 kcyc./sec. was used because the signals could be more easily monitored at this frequency. In the bush crickets tested by Wever & Vernon (1959) the maximum auditory sensitivity is to frequencies in the 10–50 kcyc./sec. range, and all other observations are in agreement with this (Autrum, 1964).

All experiments were performed during the evening or night when the insects were chirping actively.

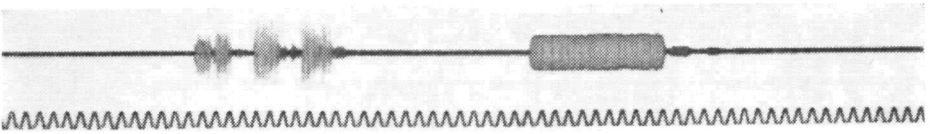

Fig. 2. A short chirp by a male followed by a 100 msec., 12 kcyc./sec., 70 db. signal with a rise and decay time of 1–2 msec. Time marker 100/sec.

Quality of the signal

Monitoring of the signal showed that the methods used gave a crude but effective control of the various parameters of the signal. The signal was often followed by a series of faint sounds, lasting a total time of less than 0·1 sec., which appeared to be produced by the loudspeaker when the signal decayed rapidly. Fig. 2 is an example of a 100 msec. signal with a rise and decay time of 1–2 msec. monitored by a microphone placed near the insect. It is preceded by a short chirp by the insect. Signal rise and decay times of less than 1–2 msec. were not possible with this type of loudspeaker.

RESULTS

Preliminary experiments

Ph. griseoaptera males were easily induced to sing in alternation with such varied sounds as the note of a Galton whistle, hissing sounds made between the teeth, and pure tones from a signal generator and loudspeaker. Actively chirping insects would alternate on a one-to-one basis with signals at repetition rates of 60 per min. or more. Less active insects would sing after every second or third signal if the rate was increased beyond the insect's chirp rate. It was very noticeable that the insects avoided chirping at the same time as the signal and that this led to alternation. In some of the experiments, using a signal generator, the signal was not switched off at all, but the intensity was varied by means of the volume control, between 30–40 and 70–80 db. The insects tended to chirp whenever the intensity was decreased. Silent males occasionally started to chirp when artificial signals were played, but the best results were obtained with males which were already chirping actively.

Experiments with long signals

When males were subjected to alternate equal periods of signal and silence of 5–30 sec. in length, (signals 10–30 kcyc./sec., 60–90 db.) they often sang during the signals, but less often than in the silent periods. More chirp emissions occurred in the first second after the end of the signal than in any other second (Jones, 1963, 1964). Although there were always more chirps in the silent periods, the percentage of chirps in the signal period increased with higher chirp rates. With 5 sec. periods of signal and silence there were hardly any chirps during the signals when the chirp rate was below 20 per min. Increasing the intensity made the signal more effective.

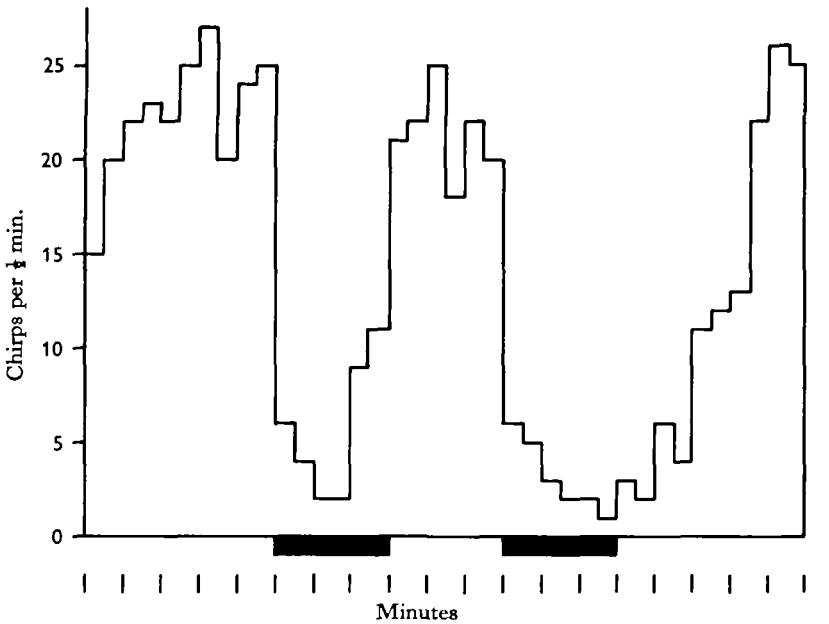

Fig. 3. Chirp rate (per ½ min.) of an isolated male (at 18° C). Effect of two 3 min. continuous signals (15 kcyc./sec., 70 db.).

In spite of this inhibitory effect it was found, when 5 sec. periods were used (signals 10–15 kcyc./sec., 65–80 db.), that the artificial signals tended to increase the overall chirp rate, except when the initial chirp rate was very high.

When 3 min. signals were used, the chirp rate was, in some experiments, decreased during the signal, and there was a 'rebound' increase at the end of the signal. Fig. 3 shows the effect on one male's chirp rate (at 18° C.) of two 3 min., 15 kcyc./sec., 70 db. signals. It is noticeable that the chirp rate takes longer to recover after the second signal. In some experiments the chirp rate was depressed for a long time after the signals. Fig. 4 shows the total chirp rate of two males in an experiment in which long signals caused a large increase in chirp rate during the signal. The long signals were preceded by an experiment with alternating periods of 5 sec. signal and 5 sec silence (signal 15 kcyc./sec., 80 db.) in which a similar increase in chirp rate was observed, but in this case most chirps were emitted in the periods of silence between the signals. Increases in chirp rate during the signal have been observed several times

but so far only in experiments with pairs of males. It has not yet been established whether this is significant.

In these experiments the same results were obtained regardless of whether the signals were controlled with a morse key, which gave 'transients' at make and break, or with the volume control of the signal generator or a cushion over the loudspeaker, which gave slow rise and decay times.

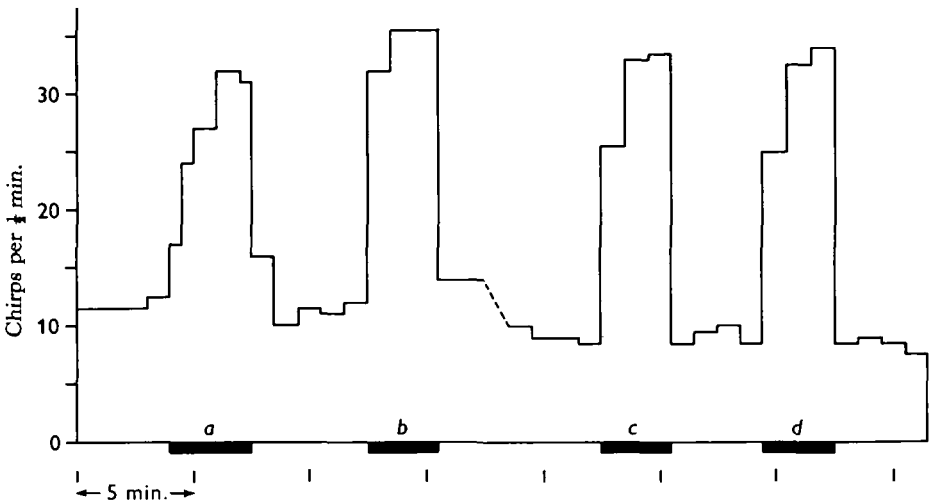

Fig. 4. Total chirp rate (per ¼ min.) of two males (at 18° C). Effect of (*a*) alternating periods of 5 sec. signal and 5 sec. silence (signal 15 kcyc./sec., 80 db.). (*b*) 3 min. continuous signal (15 kcyc./sec., 70 db.). (*c*) 3 min. continuous signal (15 kcyc./sec., 80 db.). (*d*). 3 min. continuous signal (15 kcyc./sec., 65 db.). Time marker 1 per 5 min. The dotted line indicates 1 min. in which no readings were taken.

Experiments with signals 0·01–1·0 *sec. in length*

In a series of experiments with four males (at 18·5–20° C.) 12 kcyc./sec., 70 db. signals 0·01, 0·02, 0·1 and 1 sec. in length were used. The rise or decay time of the signal was 1–2 msec. After its basal activity had been recorded, the insect was subjected to 2 min periods with signals, interspersed with 3 min. periods of silence. Each experiment was continued for about an hour and different lengths of signal were used, each for several signal periods. 1 sec. signals were made manually at a rate of 1 per 5 sec.; the others were made automatically at 1 per 5·7 sec.

Fig. 5 is a record of the chirp rate during one of these experiments. Fig. 6 shows for all these experiments the mean number of chirps in each half minute, before, during and after periods using each length of signal. The results for 0·01 and 0·02 sec. signals were added together, because each of these signals was used for only two periods of 2 min. Signals of 0·1 sec. duration were used for twelve periods and 1 sec. signals for fifteen. The increase in chirp rate during the periods with signals is significant (χ^2 test; $P < 0{\cdot}01$) for the 0·1 sec. and the 1 sec. signals, but not for the shorter signals, but this may well be due to the smaller number of results. The 1 sec. signals have a significantly greater effect in raising the chirp rate than the 0·1 sec. signals (χ^2 test; $P < 0{\cdot}01$).

Fig. 7 shows the distribution of the intervals between signal and chirp in this series

of experiments. In most of these experiments the insects chirped several times between successive signals. With a 1 sec. signal chirping is inhibited during the signal and the first chirp appears usually 0·1–0·3 sec. after the end of the signal. The timing of subsequent chirps between signals depends on the insect's chirp rate. With the shorter signals the intervals between the signal and the first chirp are much longer, usually

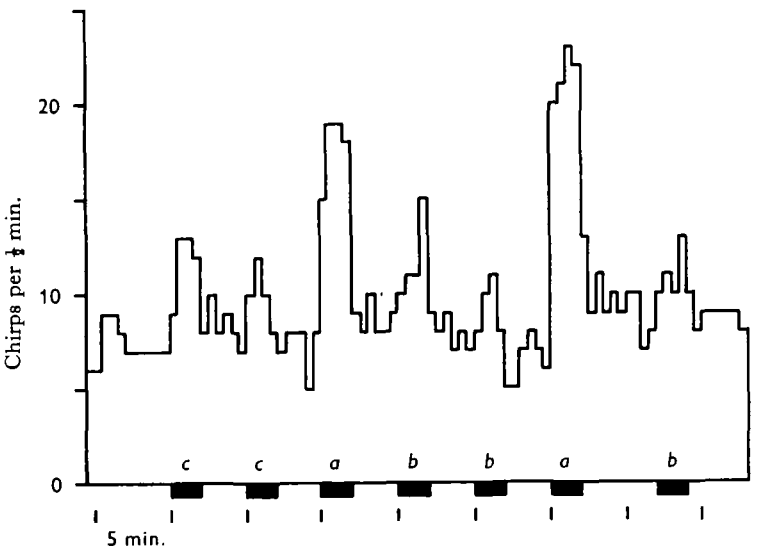

Fig. 5. Chirp rate (per ½ min.) of an isolated male. Effect of (*a*) 1 sec. signals repeated every 5 sec. (*b*) 0·1 sec. signals repeated every 5·7 sec. (*c*) 0·01 sec. signals repeated every 5·7 sec. (signals 12 kcyc./sec., 70 db.). Time marker 1 per 5 min.

about 0·4–1·0 sec. The signal has the effect of preventing chirping, not only while it is on but also during this interval after it has finished. This interval is longer and more variable than the one following the 1 sec. signals, and so first chirps and subsequent chirps tend to overlap in the record. The shortest signal-to-chirp intervals were recorded when the chirp rate was high. Chirps continue to be emitted during the first 0·1 sec. of the signal. The 50 msec. inhibitory reaction time, calculated from records of the acoustic interaction between males (Jones, 1964, 1966), would make it possible for chirps to start up to 50 msec. after the beginning of the signal.

Similar increases in chirp rate during periods with signals were observed in another series of experiments with single insects, using two different males and 1 sec., 15 kcyc./sec., 70 db. signals repeated every 5 sec. In these experiments the signals were controlled with the volume control of the signal generator and so had slow rise and decay times. In another series with a pair of different males and 1 sec., 12 kcyc./sec., 70 db. signals repeated every 5 sec. the initial chirp rate was slightly less than the signal rate. The signals caused an increase in chirp rate, and more than 60% of the chirps occurred in the first second after the end of the signal. One insect was chirping more vigorously than the other, and nearly all the chirps in the first second after the signal were made by this insect. The other insect chirped later, presumably because it had been inhibited by the first insect as well as by the signal. In alternate signal periods the signals were controlled with the volume control or with a morse key. No

difference in response to the different rise and decay times of the signal could be detected.

Some preliminary experiments were made to determine the effect of the intensity of the signal. Fig. 8 shows the chirp rate of a male (at 19° C.) during an experiment with 12 kcyc./sec., 1 sec. signals repeated every 5 sec.; 70 and 40 db. signals were used in alternate signal periods. Fig. 9 gives the total chirp rate of two males (at 18° C.) during an experiment with 1 sec., 15 kcyc./sec. signals repeated every 5 sec.,

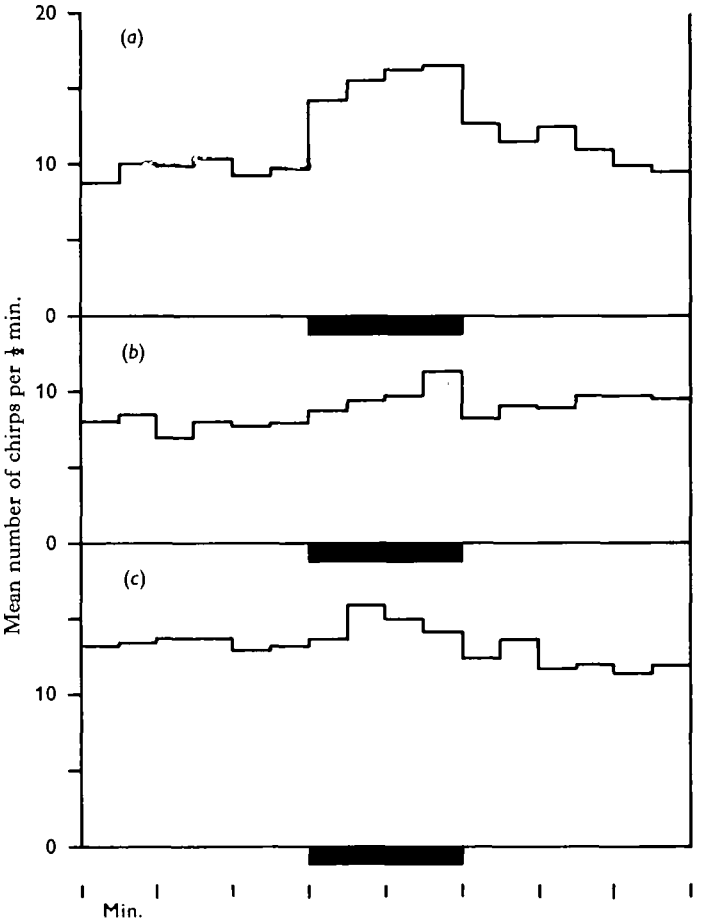

Fig. 6. Mean number of chirps per ½ min. before, during and after 2 min. periods of (*a*) 1 sec. signals repeated every 5 sec. (*b*) 0·1 sec. signals repeated every 5·7 sec. (*c*) 0·01 and 0·02 sec. signals repeated every 5·7 sec. signals 12 kcyc./sec., 70 db.).

in which periods of 70, 50 and 40 db. signals were used. It can be seen that the excitatory effect of the signals increases with the intensity. In the conditions of Fig. 9, 40 db. signals appear to cause a decrease in chirp rate.

Fig. 10 shows the distribution of chirps relative to the signals during the experiment graphed in Fig. 8. It can be seen that when 40 db. signals are used some chirps tend to break through the inhibition towards the end of the signal.

In experiments with 0·1 sec., 12 kcyc./sec. signals in which 70 db. signals were

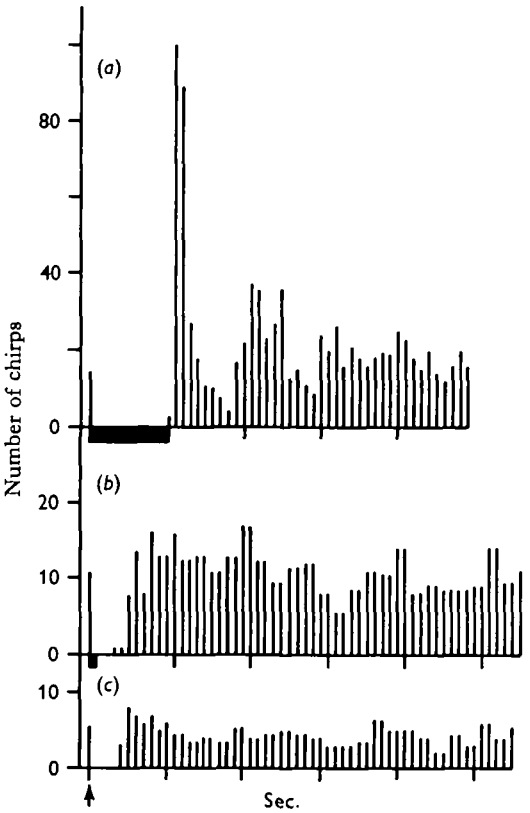

Fig. 7. Distribution of chirp emissions during the periods with signals. (*a*) (*b*) and (*c*) as for Fig. 6. Total times 30, 24 and 8 min. respectively. The arrow marks the beginning of the signal.

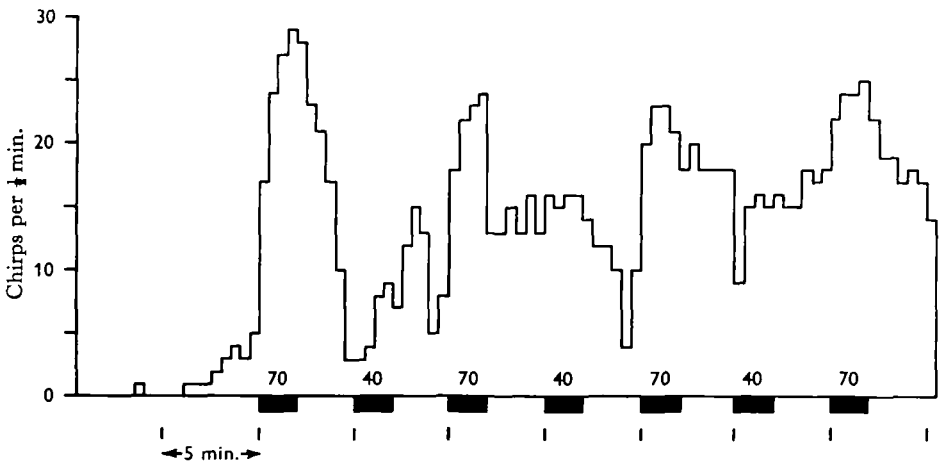

Fig. 8. Chirp rate (per ½ min.) of an isolated male (at 19° C). Effect of 2 min. periods of 1 sec signals repeated every 5 sec. 40 db. and 70 db. (12 kcyc/sec.) signals are used in alternate signal periods. Time marker 1 per 5 min.

compared with 40 db. signals, two different sorts of result were obtained. With one insect the 70 db. signals gave a moderate increase in chirp rate as in previous experiments, and 40 db. signals produced no change or possibly a slight decrease in chirp rate. The distribution of the chirps relative to the signal was similar to that in Fig. 7*b* for both 40 and 70 db. signals, but the signal-to-chirp intervals tended to be shorter

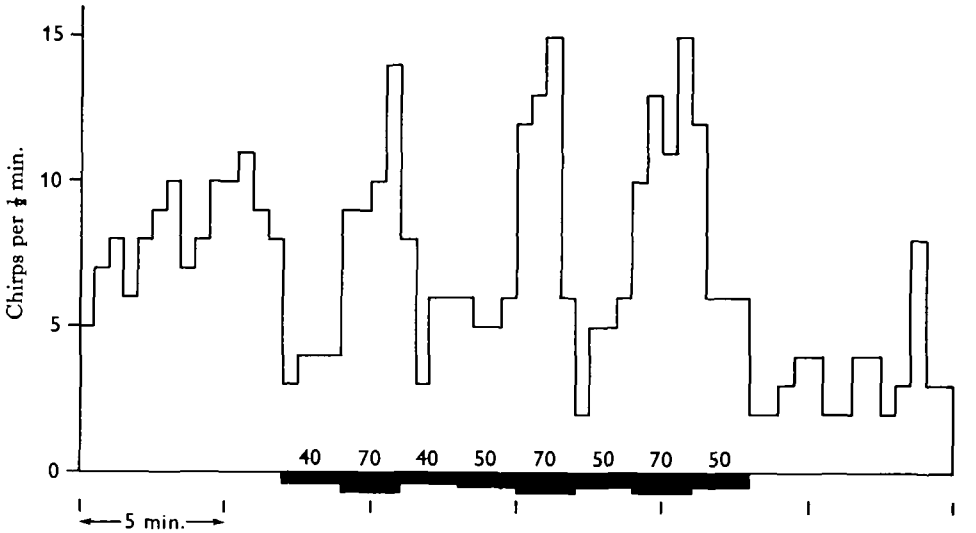

Fig. 9. Total chirp rate (per ½ min) of two males (at 18° C.). Effect of a continuous series of 1 sec. signals repeated every 5 sec. (Signals 15 kcyc./sec.; 40, 50 or 70 db.). Time marker 1 per 5 min.

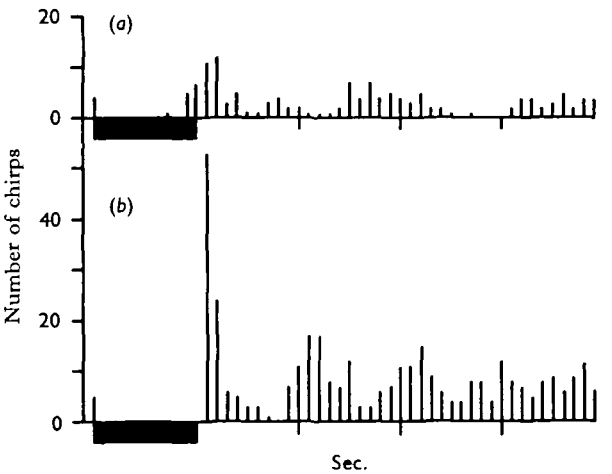

Fig. 10. Distribution of chirp emissions during experiment graphed in Fig. 8. (*a*) 40 db. signals (*b*) 70 db. signals.

after the 40 db. signals. With another male, however, the 70 db. signals caused a marked decrease in the chirp rate which was repeated in each period in which the 70 db. signals were used. The 40 db. signals had little effect on chirp rate. Fig. 11 gives the distribution of chirps relative to signals for this series. The interval between

the signal and the first chirp is shorter for the 40 db. signals. Both males were mature adults which had stridulated actively in the company of other males.

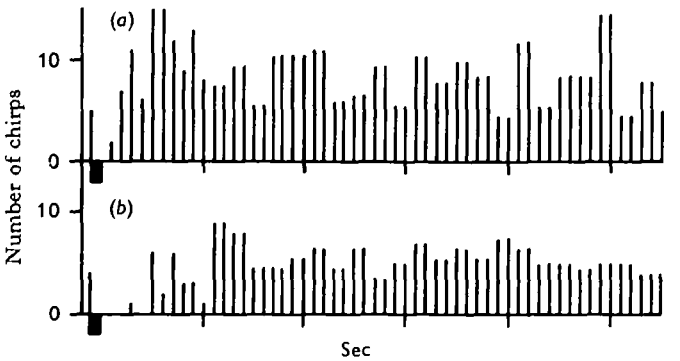

Fig. 11. Distribution of chirp emissions of an isolated male (at 22·5° C.) during an experiment in which the signals depressed the chirp rate. (*a*) during periods with 0·1 sec., 12 kcyc./sec., 40 db. signals repeated every 5·7 sec. (*b*) during periods with otherwise identical 70 db. signals.

DISCUSSION

Artificial signals which are audible to the insect appear to have an inhibitory action on chirping which is similar to the action of the stridulation of another male. A three- (or four-) syllable short chirp seems to be an all-or-nothing phenomenon and the inhibition takes effect by stopping or delaying whole chirps. The inhibitory effect may be broken down into reaction time, inhibition during the signal, and 'build-up' time necessary after the end of the signal before the next chirp can be emitted. For 70 db., 12–15 kcyc./sec. signals, 0·1 sec. or less in length, this 'build-up' time appears to be of the order of 0·4–1·0 sec. (at 18–20° C.), that is, considerably longer than the signal. This is of the same order as the interval by which one insect follows another in alternation singing. Similarly signal-to-chirp intervals and alternation intervals are shortest if the chirp rate is high. If the length of the signal is increased to 1 sec. the interval decreases, usually to 0·1–0·3 sec. Busnel, Dumortier & Busnel (1956) have noticed a similar effect with *Ephippiger* males. With longer signals chirps may start to 'break through' the signal before the end, but even when this occurs the chirp rate during the signal is usually less than that between signals (Jones, 1963, 1964). There appears to be a conflict between the 'spontaneous' tendency of the insect to chirp and the inhibitory effect of the signal. The inhibitory effect is increased by intensity but is possibly decreased by adaptation if the signal is prolonged. 'Break-through' can occur towards the end of 1 sec., 40 db. signals.

The increase in chirp rate that usually takes place during a period in which signals are being produced indicates that the signals have an excitatory as well as an inhibitory effect. The excitatory effect is increased by an increase in length of the signals from 0·1 to 1 sec. or more, and, if the signals are excitatory, by an increase in intensity from 40 to 70 db. On occasions, the signals may have a depressant rather than an excitatory effect, and in this case an increase in intensity increases the depressant effect. Both these effects may last for several minutes after the signals have ended. All the experiments were made at much the same time of day, so this factor does not seem to have determined whether the signals were excitatory or depressant.

The high probability of a chirp being emitted 0·1–0·3 sec. after the end of a 1 sec. signal may be due only to the delaying of chirps which would have been emitted during the signal. When the chirp rate is initially slightly less than the signal repetition rate, however, the signals appear to cause a retiming of the chirps as well as a small increase in chirp rate, so that most are emitted within 1 sec. of the end of the signals. This is similar to the 'paradoxical driving' of follower neurones in the cardiac ganglion of decapod Crustacea by inhibitory stimulation (Maynard, 1961). Weih (1951) found that with *Chorthippus brunneus* the most 'responses' to artificial signals were obtained when the signal repetition rate did not depart too far from the natural emission rate.

Busnel & Loher (1961) have shown that for the signal to evoke stridulation in a male *C. brunneus* the rise and decay times must be short. In their experiments the insect was silent before the signals were produced, and so this may be a case in which signals have a mainly excitatory effect; or it may be an extreme case of 'paradoxical driving'. In this insect the chirp of another male appears to have a large excitatory effect; alternation between males causes a marked acceleration of emissions by both partners (Weih 1951). It is not known whether transients are particularly important to *Pholidoptera griseoaptera* males. Signals of 1 sec. or more with long rise and decay times have similar effects to those made with a morse key which appeared to have 'transients'. The insect chirps when the signal intensity has decreased to a sufficiently low level. However, transients may be more important in short signals which are comparable in length with the insect's short chirp. In all the experiments with these signals, and in comparisons with signals of different length, a rise and decay time of 1–2 msec. was used. It would be interesting to investigate the effect of altering the rise and decay times of such short signals; transients may possibly have more effect on excitation than inhibition. The present apparatus, particularly the loudspeaker, is not suitable for producing really sharp transients where the signal rises or decays within 1–2 cycles.

It appears that artificial signals always have an inhibitory effect on chirping in *Ph. griseoaptera* and that this takes effect rapidly, with a reaction time of the order of 50 msec. Such signals may also have excitatory or depressant effects which are delayed; these may only become apparent when the signal has ended, and may last for several minutes. These effects may be purely after-effects of inhibition. J. S. Kennedy (1958, 1965) and Kennedy & Booth (1963, 1964) have postulated such 'antagonistic induction' and 'antagonistic depression' between mutually inhibitory activities in the behaviour of aphids—a theory which draws upon the work of Sherrington (1947) on successive induction between antagonistic spinal reflexes in mammals. The work of Maynard (1961) on the cardiac ganglion of decapod Crustacea has shown that rebound activity increases with the strength of inhibition and also with the duration of inhibition for the first 5–10 sec. in such a way as to suggest that the rebound activity and adaptation to the inhibitory stimulus are connected. Similarly, an increase in length or intensity appears to increase the after-effect of the signals on the chirp rate of *Ph. griseoaptera*.

Although many of the experiments with very long signals give a typical picture of inhibition and 'rebound', the large increases in chirp rate in some of these experiments (Fig. 4) are difficult to explain on a 'rebound' theory. These increases appear to occur

at times when other signals are also having a particularly excitatory effect, but in the case of the very long signals the effect is manifested during the signal. When the signal ends the chirp rate decreases until it reaches the basal level. The signal may interfere with the insect's auditory feedback system, if such a system exists, and cause 'runaway' activity, but it seems more likely that the signal has parallel inhibitory and excitatory effects in the C.N.S. It appears that the inhibitory effect is fast, but may be reduced by adaptation, and the excitatory effect is slow and longer lasting, and may well account for the adaptation. D. Kennedy (1960) has proposed such a parallel system of excitation and inhibition in the visual system of the mollusc, *Spisula*; and the work of Sherrington (1947) on successive induction does not rule out this possibility.

It would be most interesting to know more about the path of the auditory input from the tympanal organ. Suga & Katsuki (1961) have discovered auditory 'T' large fibres in the tettigoniid, *Gampsocleis*, by means of which it appears that the auditory input is transmitted equally to the brain and to the thoracic ganglia. Nothing is known about the possible direct effect of the auditory input on the thoracic system which by comparison with Huber (1964, 1965) would appear to control the production of syllables but not of chirps. As acoustic inhibition acts at the chirp level rather than the syllable level, it seems likely that both inhibition and excitation are mediated via the brain. In *Gampsocleis* the auditory input takes about 12 msec. to reach the brain (Suga & Katsuki, 1961). This would leave time for it to inhibit the centre which controls chirping within a total reaction time of 50 msec. If the syllables are myogenic, however, inhibition could act directly on the thoracic nerve centres without affecting the all-or-nothing nature of the chirp. The excitatory (or depressant) effect of the auditory input may depend on more complex interactions with higher centres and with other inputs. Hormonal levels may be important in determining whether this secondary effect is excitatory or depressant.

Another aspect to be considered is that of recognition of the signal. Walker (1957) has shown that the pattern of amplitude modulation in the song of male crickets and in artificial imitations of the song is very important in evoking the phonotaxis of the females. Busnel *et al.* (1956) have shown that an artificial signal will evoke this response in *Ephippiger* females if it possesses one sufficiently sharp transient. The work of Regen (1926) on *Pholidoptera* (*Thamnotrizon*) *aptera* (F.) has often been misquoted in the literature to the effect that he could only induce alternation with artificial signals in newly moulted males which had not learnt to alternate with other males (Pumphrey 1940; Haskell, 1961;; Thorpe, 1963; Dumortier, 1964). In fact, before he began his experiments, Regen allowed the young males to sing together until they had begun to alternate for fairly long periods. He usually waited until one of a pair of males which had been alternating began to sing less frequently, and he then alternated with the other using artificial signals. He was interested in testing the frequency range of the hearing organ and did not re-examine the reasons for his earlier failures with more mature adults. *Ph. griseoaptera* males appear to respond to short artificial signals in much the same way as they would to the song of another male. A longer signal is even better for evoking a response and perhaps corresponds to the long 'rivalry' chirp of another male. Recognition of the finer points of a chirp by another male does not seem to be particularly important.

SUMMARY

1. Artificial signals have an inhibitory effect on chirping which affects whole chirps. With signals 1 sec. or less in length, the insects hardly ever chirp during the signal. If the signal is longer, chirps 'break through', but the chirp rate is usually less during the signals than in the periods of silence between the signals.

2. With signals of 0·1 sec. or less, the insect does not chirp until 0·4–1·0 sec. after the end of the signal. With longer signals this interval is reduced until the insect chirps during the signal.

3. Artificial signals may have an excitatory effect in increasing the total chirp rate in the period during which signals are being produced, and this effect may last for several minutes after the signals have ended. This may be due to rebound from inhibition or to a parallel excitatory effect of the signal. The excitatory effect is increased if the length of the signal is increased from 0·1 to 1 sec.

4. The excitatory effect is not so certain as the inhibitory effect and sometimes may be reversed, giving a reduction in chirp rate.

5. With very long signals (3 min.) there may be (*a*) a decrease in chirp rate during the signal followed by an increase after the signal, (*b*) a decrease in chirp rate during the signal with a very slow recovery after the signal, or (*c*) an increase in chirp rate during the signal followed by a decrease to normal after the signal. These effects can be more easily explained by parallel excitatory and inhibitory effects than by rebound from inhibition.

6. An increase in intensity of the signal from 40 to 70 db. gives an increase in both the inhibitory and the excitatory (or depressant) effects of the signals.

7. The rapidity and certainty of the inhibitory effect make it seem probable that few synapses are involved. The greater flexibility of the excitatory effect indicates that this effect may be mediated by higher centres in the C.N.S.

8. Recognition of the characteristic chirp of the species does not appear to be particularly important in the acoustic interaction of *Ph. griseoaptera* males.

I am grateful to Mr W. B. Broughton, Department of Botany and Zoology, Sir John Cass College, and Prof. J. D. Gillett, Department of Biology, Brunel College, for their criticism and advice, and to Dr J. S. Kennedy, F.R.S., A.R.C. Unit of Insect Physiology, Cambridge, for his helpful discussion of some of this work. I should also like to thank the members of the Department of Physics and the Department of Electrical Engineering and Electronics, Brunel College, who have helped me.

REFERENCES

AUTRUM, H. (1964). Anatomy and physiology of sound receptors in invertebrates. In *Acoustic Behaviour of Animals*, ed. R. G. Busnel, pp. 412–33. Amsterdam: Elsevier.

BUSNEL, R. G., DUMORTIER, B. & BUSNEL, M. C. (1956). Recherches sur le comportement acoustique des Ephippigères, *Bull. biol. Fr. Belg.* **90**, 219–86.

BUSNEL, R. G. & LOHER, W. (1961). Déclenchement de phonoréponses chez *Chorthippus brunneus* (Thbg). Acridinae. *Acustica*, **11**, 65–70.

DUMORTIER, B. (1964). Ethological and physiological study of sound emissions in Arthropoda. In *Acoustic Behaviour of Animals*, ed. R. G. Busnel, pp. 583–654. Amsterdam: Elsevier.

HASKELL, P. T. (1961). *Insect Sounds*. London: Witherby.

HUBER, F. (1964). The role of the central nervous system in Orthoptera during the co-ordination and control of stridulation. In *Acoustic Behaviour of Animals* ed. R. G. Busnel pp. 440–88. Amsterdam: Elsevier.

HUBER, F. (1965). Brain controlled behaviour in orthopterans. In *The Physiology of the Insect Central Nervous System*, ed. J. E. Treherne and J. W. L. Beament pp. 233–46. London and New York: Academic Press.

JONES, M. D. R. (1963). Sound signals and alternation behaviour in *Pholidoptera*. *Nature, Lond.*, **199**, 928–9.

JONES, M. D. R. (1964). Inhibition and excitation in the acoustic behaviour of *Pholidoptera*. *Nature, Lond.*, **203**, 322–3.

JONES, M. D. R. (1966). The acoustic behaviour of the bush cricket. *Pholidoptera griseoaptera* 1. Alternation, synchronism and rivalry between males. *J. Exp. Biol.* **45**, 15-30.

KENNEDY, D. (1960). Neural photorception in a lamellibranch mollusc. *J. gen. Physiol.* **44**, 277–99.

KENNEDY, J. S. (1958). The experimental analysis of aphid behaviour and its bearing on current theories of instinct. *Proc. 10th Int. Congr. Ent., Montreal*, 1956, **2**, 397–404.

KENNEDY, J. S. (1965). Co-ordination of successive activities in an aphid. Reciprocal effects of settling on flight. *J. Exp. Biol.* **43**, 489–509.

KENNEDY, J. S. & BOOTH, C. O. (1963). Co-ordination of successive activities in an aphid. The effect of flight on the settling responses. *J. Exp. Biol.* **40**, 351–69.

KENNEDY, J. S. & BOOTH, C. O. (1964). Co-ordination of successive activities in an aphid. Depression after flight. *J. Exp. Biol.* **41**, 805–24.

MAYNARD, D. M. (1961). Cardiac inhibition in decapod Crustacea. In *Nervous Inhibition*, ed. E. Florey, pp. 144–78. New York: Pergamon Press.

PUMPHREY, R. J. (1940). Hearing in insects. *Biol. Rev.* **15**, 107–32.

REGEN, J. (1926). Über die Beeinflussung der Stridulation von *Thamnotrizon apterus* Fab. (Männchen) durch künstlich erzeugte Töne und verschiedenartige Geräusche. *Sber. Akad. Wiss. Wien*, **135**, 329–68.

SHERRINGTON, C. S. (1947). *The Integrative Action of the Nervous System*, 2nd edn. University Press, Cambridge.

SUGA, N. & KATSUKI, Y. (1961). Central mechanism of hearing in insects. *J. Exp. Biol.* **38**, 545–58.

THORPE, W. H. (1963). *Learning and Instinct in Animals*, 2nd edn. London: Methuen.

WALKER, T. J. (1957). Specificity in the response of female tree crickets (Orthoptera, Gryllidae, Oecanthinae) to calling songs of the males. *Ann. ent. Soc. Am.* **50**, 626–36.

WEIH, A. S. (1951). Untersuchungen über das Wechselsingen (Anaphonie) und über das angeborene Lautschema einiger Feldheuschrecken. *Z. Tierpsychol.* **8**, 1–41.

WEVER, E. G. & VERNON, J. A. (1959). The auditory sensitivity of Orthoptera. *Proc. Natn. Acad. Sci. U.S.A.* **45**, 413–19.