

'SPECIFIC' AND 'NON-SPECIFIC' STIMULI IN THE DRIVE OF RESPIRATION

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Abstract. In decerebrate and awake cats it was shown that central chemosensitivity can be blocked by cooling or coagulating bilaterally a formerly described area on the ventral medullary surface of cats, called area S. Such a procedure is followed by apnoea under the condition that vagi and sinus nerves are cut. With either vagi, sinus nerves, or central chemosensitivity intact respiration can be influenced by noise, touch, and electrical stimulation of dorsal roots, femoral nerves, and pontine reticular formation. With vagi, sinus nerves, and central chemosensitivity blocked, these 'non-specific' stimuli are not able to influence respiration. In the described experiments vagi, sinus nerves, and central chemosensitivity were essential for the activity of the respiratory centres and were therefore called 'specific'.

Central chemosensitivity was localized on the ventral medullary surface of cats by Mitchell et al. (1963) (Fig. 1M) and Loeschcke et al. (1970) (Fig. 1L). Schlaefke and Loeschcke (1967) have shown that lung ventilation and phrenic discharge (M. E. Schlaefke and W. R. See, unpublished data) vanish completely when vagi and sinus nerves are cut, and area S (Fig. 1S) is bilaterally cooled or coagulated. Area S is a little field between the areas M and L on the ventral medullary surface and was described in 1967 (Schlaefke and Loeschcke 1967). Its possible relation to areas M and L has been discussed (Schlaefke et al. 1969, 1970).

Figure 2 gives an example from such an experimental series with anaesthetized cats. Vagi and sinus nerves are cut, area S is coagulated on one side, and cooling of the intact area S causes complete respiratory arrest. The same observations were made in unanaesthetized cats, which were decerebrated. In awake cats with vagi and sinus nerves intact but

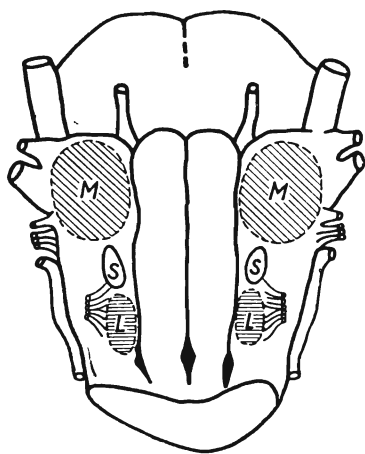


Fig. 1. Ventral medullary surface of the cat. M, areas of Mitchell et al. (1963); L, areas of Loeschke et al. (1970); S, areas of Schlaefke and Loeschke (1967).

with both areas S coagulated CO_2 -sensitivity was diminished by more than 65%. These and other formerly published observations have proved that the central chemosensitivity can be abolished by cooling or coagulating area S on both sides.

Encouraged by G. Moruzzi (personal communication) and the investi-

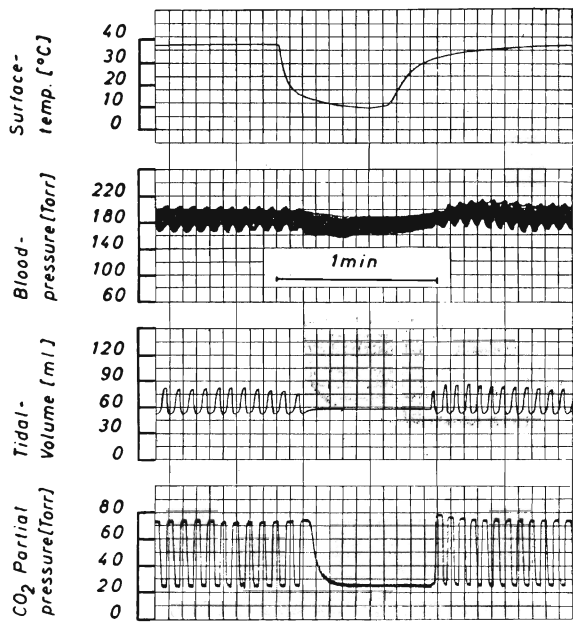


Fig. 2. Anaesthetized cat (chloralose-urethane), 12-9-1968. Vagi and sinus nerves cut, area S coagulated on the left side. Cold block of the intact area S. Inspiration upwards. (From Schlaefke et al. 1969.)

gations of Hugelin and Cohen (1963) another experimental series was performed. Hugelin and Cohen have shown that in vagotomized cats stimulation of the reticular formation was able to drive the respiratory centres, elicited 'non-specifically' by an arousal reaction mechanism.

Such a 'non-specific' stimulation caused by the ringing of a telephone bell (Fig. 3, arrow) could be studied in conscious cats, in which the central

Fig. 3. Conscious cat, 8-12-1970. Areas S coagulated bilaterally on 8-4-1970. Ringing of the telephone marked by the arrow. Vagi and sinus nerves intact.

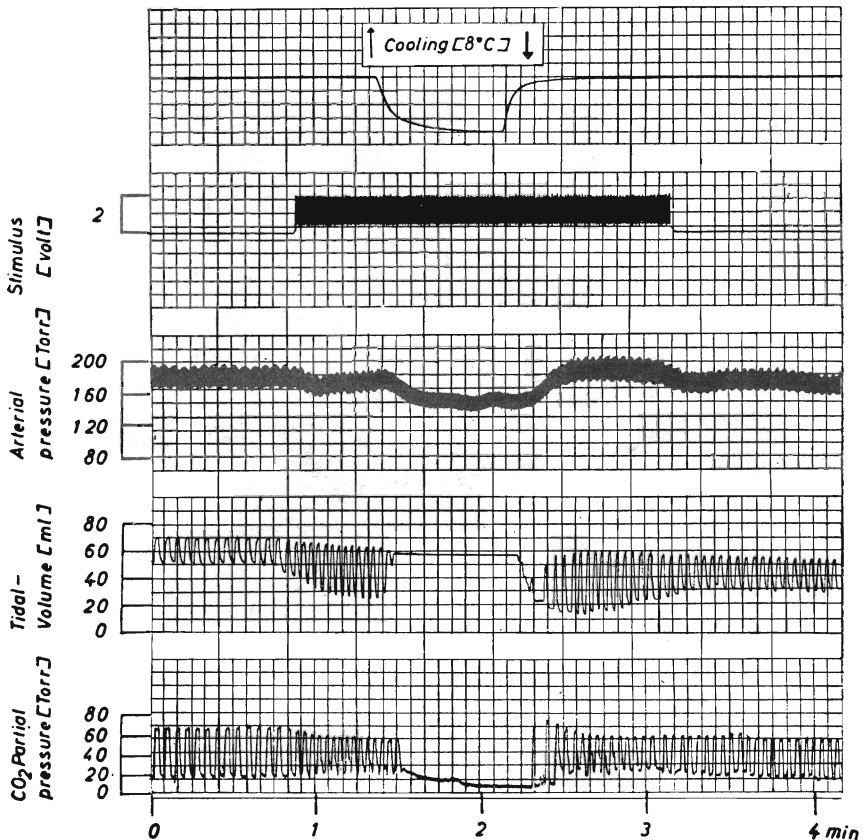
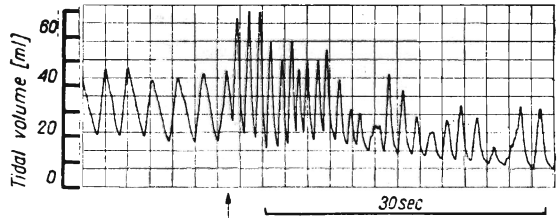


Fig. 4. Unanaesthetized decerebrate cat, 4-28-1969. Vagi and sinus nerves cut, area S coagulated unilaterally. Electrical stimulation of the left femoral nerve (2 v, 40 shocks/sec, 1 msec). Cold block of the intact area S. Inspiration downwards. (From Schlaefke et al. 1969.)

CO₂-sensitivity was abolished chronically (Schlaefke et al. 1971) by coagulation of both areas S. Vagi and sinus nerves were intact in these cats.

Similar observations were reported previously (Schlaefke et al. 1969). In decerebrate cats vagi and sinus nerves were cut and area S was coagulated on one side. Stimulation of a femoral nerve increased ventilation. Cooling the intact area S during the phase of stimulation, however, was followed by complete apnoea (Fig. 4). The 'non-specific' influence had lost its effect under these conditions. Similar observations were made when stimulating the cornea, dorsal roots of the spinal cord, or pontine reticular formation.

A different result was obtained when stimulating the sinus nerve (Fig. 5). Vagi and sinus nerves were cut, and area S was coagulated on one side. Cooling the intact area S caused apnoea as usual. During this cold block and phase of apnoea stimulation of the rostral end of a sinus

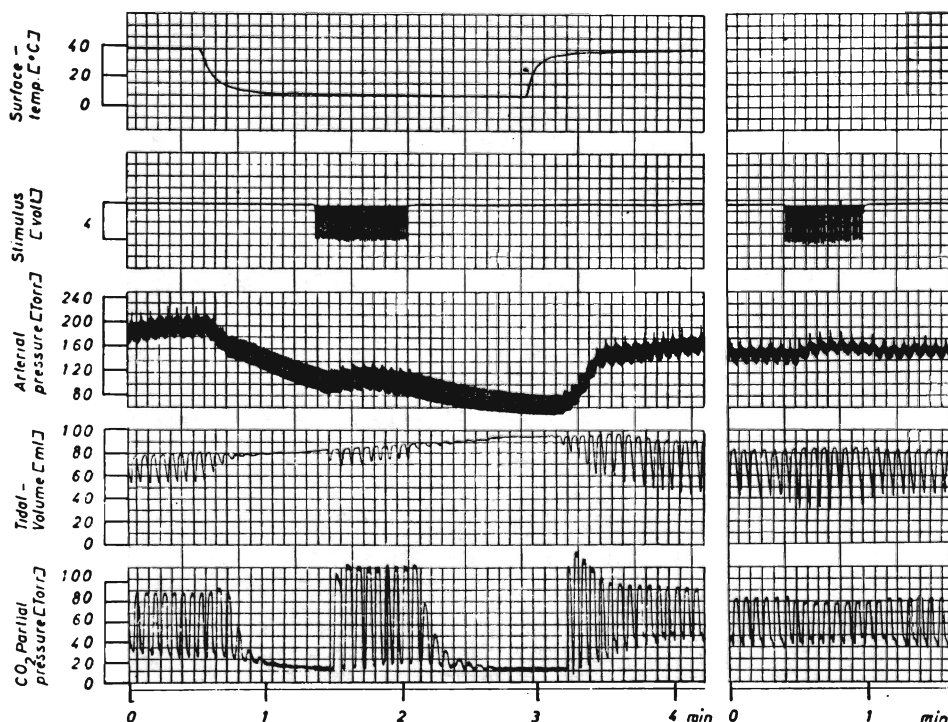


Fig. 5. Unanaesthetized decerebrate cat, 5-19-1969. Vagi and sinus nerves cut, area S coagulated unilaterally. Electrical stimulation of the left sinus nerve (4 v, 20 shocks/sec, 1 msec) during cold blockade of the intact area S (left side of the recording), with one area S intact (right side of the recording). (From Schlaefke et al. 1969.)

nerve elicited respiratory activity for the time of stimulation. Such an effect could never be seen when 'non-specific' pathways were stimulated.

However, there is one observation, of which Fig. 6 is an example, that under the condition of a raised body temperature an additional drive becomes able to maintain respiratory rhythm even when the stimuli from vagi, sinus nerves, and the central chemosensitive structures are cut off. The body temperature was 41°C. The cat was panting and gasping. Vagi

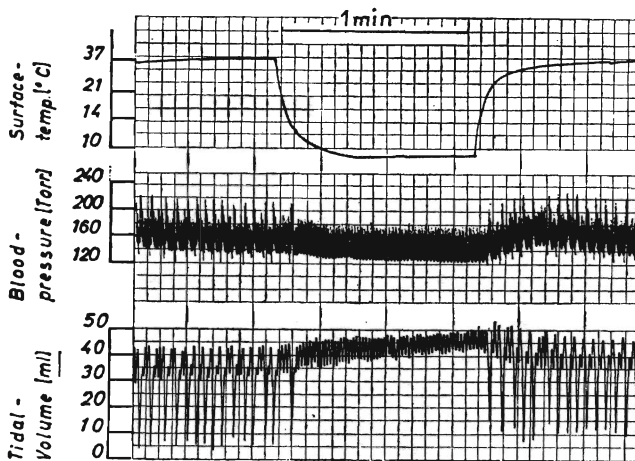


Fig. 6. Anaesthetized cat (chloralose-urethane), 1-24-1967. Body temperature 41°C. Vagi and sinus nerves cut, one area S coagulated. Cold block of the intact area S. Inspiration downwards. The gasps disappear during cooling of area S.

and sinus nerves were cut, and one area S was coagulated. The gasps disappeared during cold block of the intact area S, but the panting continued.

It is concluded that in the normothermic animal vagi, sinus nerves, and the central chemosensitivity play an exceptional and essential role in the drive of respiration, and cannot be replaced by any other drive under the conditions of these experiments. They are therefore in this paper called 'specific' afferent influences. In contrast to this the 'non-specific' afferent influences depend on at least one intact 'specific' influence which seems to guarantee their 'non-specific' action on respiration. It is possible that in these experiments stimulation of 'non-specific' drives remained below the threshold of the integrating respiratory neurons as long as 'specific' pathways did not co-operate. This would mean that the 'specificity' discussed here is based on a critical level of activity. Hyperthermia could be an example where additional drives may

become sufficient to elicit respiratory activity not necessarily dependant upon the operation of 'specific' influences like vagi, sinus nerves, and central chemosensitivity.

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