may slow the exchange. It is known, however, that muscles can be maintained for some hours with values of sodium and potassium which are very similar to those found *in vivo* if insulin or certain plasma proteins are present[3,4], and this finding led to attempts to detect the effect of insulin on sodium movements. A method has been used which can give consistent results when applied to diaphragm muscle and in which the effects due to the rib and attached tissue are avoided.

The muscles were from rats of 50–60 g and the thickness was 0·35 mm (mean of ten measurements). The diaphragms were rapidly dissected and suspended on holders in oxygenated saline[4]. After 1 h at 38° C the uptake of sodium-24 was complete, the specific activity of the sodium in the muscle as compared with that in the saline being close to unity (ratio 1·01, mean of eight determinations, range 0·96–1·05). The holders containing the muscles were afterwards passed through a series of tubes containing inactive saline, the tubes being changed every minute. It was found that the rapidly exchanging fraction which includes the extracellular sodium was lost after 5 min, as found previously[1]. The washout was stopped after 5, 10, 15 or 21 min, the diaphragm was freed from rib and tendon and the radioactivity which remained was measured.

Since the specific activity before the washout equalled that of the saline the radioactivity of the muscle could be expressed in a standard manner as μmole sodium per g wet tissue. The points when plotted on semilogarithmic paper were consistent with a linear fall, and from the slope of the regression the rate-constant was 0·122 min^{-1} ± 0·0057 (standard deviation estimated from twenty muscles), so that the mean half-time was 5·7 min. Fifteen muscles treated throughout with insulin (0·02 unit/ml.) gave a rate-constant of 0·167 min^{-1} ± 0·015 (*S.D.* of fifteen), with mean half-time of 4·2 min ($P < 0·01$).

In the presence of insulin the potassium content of diaphragm is maintained at a higher level than in controls[4], and in the present series this finding has been confirmed. The mean potassium content of nine muscles which had been treated with insulin was 102 μmoles/g ± 6·6 (*S.D.* of nine), while the content found in controls was 91 μmole/g ± 4·9 (*S.D.* of fifteen, $P < 0·01$).

Insulin has long been known to affect the potassium content of muscle, and this hormone has also been found to increase the resting potential of isolated rat muscle[5]. These effects appear also to be associated with an increase in the rate of turnover of sodium.

This work was supported by a grant from the Medical Research Council. The insulin was kindly supplied by Burroughs Wellcome and Co.

R. Creese

Physiology Department,
St. Mary's Hospital Medical School,
London, W.2.

[1] Creese, R., *Proc. Roy. Soc.*, B, **142**, 497 (1954).
[2] Jenkinson, D. H., and Nicholls, J. G., *J. Physiol.*, **159**, 111 (1961).
[3] Flückiger, E., and Verzár, F., *Helv. physiol. pharm. Acta*, **15**, 293 (1957).
[4] Creese, R., and Northover, J., *J. Physiol.*, **155**, 343 (1961).
[5] Zierler, K. L., *Science*, **126**, 1067 (1957).

Innervation of Laryngeal Joints, and Laryngeal Reflexes

Precisely regulated movements of the vocal folds (cords) are known to occur during respiration, swallowing and phonation in most mammals, including man[1]. That such movements must involve integrated reflex adjustments of the tone of the laryngeal muscles is apparent; and, by analogy with the situation in the limbs, the source of such reflex regulation has usually been sought in the laryngeal muscles themselves (or in the laryngeal mucosa). There is, however, no agreement on whether appropriate receptor nerve endings are present in the laryngeal muscles[2,3]; and attempts to record afferent proprioceptive discharges from the nerves supplying the laryngeal muscles have failed to demonstrate them[4]. Nevertheless, direct electrical stimulation of afferent fibres in the laryngeal nerves does produce reflex alterations in the tone of laryngeal muscles[4,5].

It occurred to us that the source of some of the afferent activity involved in the production of intrinsic laryngeal reflexes might be in receptor nerve endings located in the laryngeal joint tissues. Accordingly, we have carried out microdissections of the nerves in the articular regions of the cat's larynx, and neurohistological studies of the laryngeal joint tissues in the crico-arytenoid, crico-thyroid and thyrohydroid joints and of the tissue of the thyro-epiglottic junction. The capsules of the crico-arytenoid and crico-thyroid joints have been found to have a dense innervation, and to contain nerve fibres supplying receptor nerve endings embedded in the fibrous capsules of the joints. The innervation is more dense in the crico-arytenoid joint than in the other laryngeal joints. The nerve endings in the crico-arytenoid joint capsule are supplied from at least one specific articular nerve, some of the fibres of which join the superior laryngeal nerve while others descend in the recurrent laryngeal nerve. The nerve endings in the crico-thyroid joint capsule are supplied from another articular nerve that joins the recurrent laryngeal nerve.

Further neuroanatomical and neurophysiological studies are being undertaken in the cat and in man to define the contribution made by these laryngeal articular receptors to the reflex regulation of the laryngeal musculature. In this respect, it may be noted that nerve endings have previously been identified in the thyro-epiglottic joint of the rat[6], and in the perichondrium and related fibrous tissue of the laryngeal cartilages of the cat and man[7]; and that afferent discharges from the receptor endings in the thyro-epiglottic joint have been recorded in the rat[6] and the rabbit[8].

John A. Kirchner*
Barry Wyke

Department of Applied Physiology,
Royal College of Surgeons of England.
Lincoln's Inn Fields,
London, W.C.2.

* On sabbatical leave from the Department of Otolaryngology, Yale University School of Medicine.

[1] Negus, V. E., *The Comparative Anatomy and Physiology of the Larynx* (Heinemann, London, 1949).
[2] Mündnich, K., *Archiv. Ohren-, Nasen- Kehlk. Heilk.*, **169**, 190 (1956).
[3] Lucas Keene, M. F., *J. Anat.*, **95**, 25 (1961).
[4] Mórtensson, A., *Acta physiol. Scand.*, **57**, 248 (1963).
[5] Esslen, E., and Schlosshauer, B., *Folia phoniat.*, **12**, 161 (1960).
[6] Andrew, B. L., *J. Physiol.*, **126**, 507 (1954).
[7] Jankovskaya, N. F., *Arkhiv. Anat.*, **8**, 70 (1959).
[8] Petitpierre, C., *Helv. physiol. Acta*, **1**, 325 (1943).

Uptake of Iodine-131 by the Thyroids of Female Mice during the Œstrous Cycle

Previous investigations have shown that thyroid activity undergoes cyclic variation correlated with the phases of the œstrous cycle. The thyroid gland reaches its maximal activity during the phase of œstrus in rats[1,2], rabbits[3], ewes[4,5], buffaloes[6], and cows[7]. In the case of mice and guinea pigs the picture differs as the activity of their thyroids is low at œstrus[8,9]. In the present investigation it was decided to study the uptake of iodine-131 by the thyroid glands of female mice at short intervals during the œstrous cycle and correlate it with ovary histological appearance.

One hundred mature female albino mice were used. Vaginal smears were obtained at 12-h intervals for 5 days. Smears were then taken every 6 h for another 5 days. The animals were then divided into 6 groups representing: early pro-œstrus, late pro-œstrus, œstrus, metœstrus, early and late di-œstrus. The mice were injected with