The Innervation of Muscle and the Electrophysiology of Meat

H.J. Swatland*

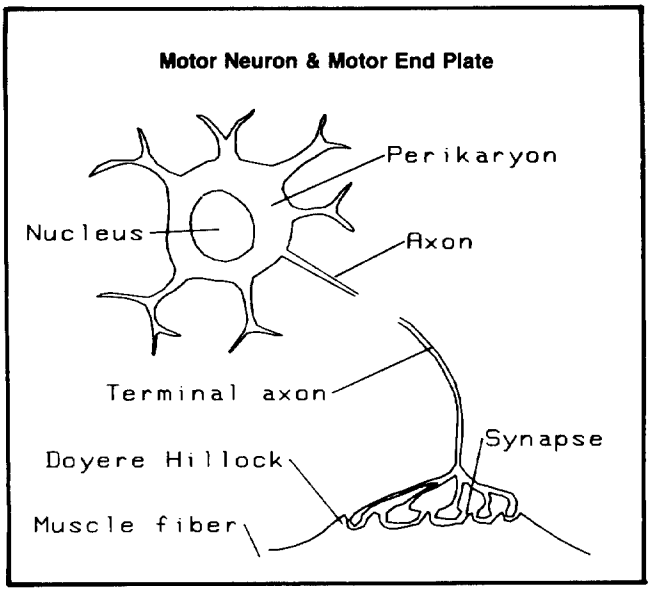
Introduction

There are two main types of interactions between nerves and muscles: (1) short-term interactions in the control of muscle contraction and (2) long-term interactions in the control of muscle metabolism. Although the most obvious actions are directed from nerve to muscle, the more subtle actions in the reverse direction are equally important. In short-term relationships, for example, there is a feed-back flow of information from neuromuscular spindles so that muscle contraction may be regulated to achieve purposeful muscle movements. Long-term interactions are more subtle, but some type of feed-back information is probably present so that the neuron can relate correctly to its field of innervation. Short-term interactions involving the initiation and regulation of muscle contraction are adequately described in physiology text-books. Far less is known, however, about long-term interactions and the fundamentals of the subject are still slowly evolving.

The importance of long-term interactions in meat science is that the nervous system regulates muscle growth and metabolism, and it is but a short step from growth rate to meat yield and from antemortem muscle metabolism to postmortem metabolism and meat quality. With regard to future attempts to manipulate patterns of muscle growth in meat animals, nerve-muscle interactions may represent the highest level of the control system that we might wish to manipulate. Hence, it is the nature of long-term interactions that will be addressed in the first part of this presentation.

The second part of the presentation relates to those aspects of short-term interactions that are seldom covered in physiology texts, yet which are of considerable interest to meat scientists involved with the pH-dependent aspects of meat quality. Although many classical physiological preparations are based on moribund animals that have been anesthetized, partly dissected or radically altered in some way, physiologists have made little attempt to understand the terminal physiology of dying and dead animals. These events are of extreme importance in understanding the mechanisms involved in the conversion of living muscles into meat. Three topics are of special importance: (1) the acceleration of postmortem glycolysis by reflex neural activity; (2) the role of the nervous system in postmortem electrical stimulation, and (3) the biophysical changes that occur in muscle fiber membranes. These topics are addressed in the second part of this presentation. A detailed review of these topics with an extensive bibliography has been undertaken in a recent textbook (Swatland, 1984) and the objective here is simply to discuss these topics in a general way. Figure 1 shows some of the key features of the motor innervation of muscle.

Figure 1



Long-Term Interactions

Historical Background

Early physiologists were aware that denervated organs often underwent a series of degenerative changes that usually culminated in a severe loss of function. This phenomenon was particularly obvious in skeletal muscles. Hence, in attempting to categorize the various parts of the nervous system on a functional basis, a special status was given to trophic nerves – those that were in some way involved with the nutrition of their target organs. It was realized that ordinary motor and sensory neurons to the musculature had a trophic function and that the loss of this activity might be responsible for the atrophy of denervated muscles. The term "atrophy" survives in current use and is a reminder of how widespread must have been the concept of neurotrophy. Like

*H.J. Swatland, Departments of Food Science and Animal & Poultry Science, University of Guelph, Guelph, Ontario N1G2W1, Canada

Reciprocal Meat Conference Proceedings, Volume 38, 1985.

many other of the clairvoyant ideas of the time, however, the idea of trophic neural functions gradually passed from sight as progressive generations of textbooks had to weed out older information to make room for new findings.

The concept of trophic neural functions underwent a dramatic renascence when the cross-reinnervation experiments of Buller, Eccles and Eccles were published in 1960. As its name suggests, and as most readers will already know, the basic feature of the crossed-reinnervation experiment is to transect two nearby nerves that innervate separate muscles, preferably muscles with different contraction speeds, and then to re-unite the proximal end of one nerve with the distal end of the other nerve and vice versa. When successful, this experimental manipulation results in both muscles becoming reinnervated – but by crossed nerves.

The first successful crossed-reinnervation experiment may have been undertaken by Kennedy some time prior to 1909. The experiment was undertaken on flexor and extensor muscles in the thighs of dogs. Although there was quite a widespread awareness of trophic neural functions at the time, the possibility of using the crossed-reinnervation strategy to investigate trophic effects was missed completely. The most likely reason for this lost opportunity is that other problems were more pressing at the time. Kennedy exposed the motor cortex of the brain and reported that stimulation of the original flexor site now caused extensor activity and vice versa. In other words, the more important issue of the day was the investigation of cortico-spinal pathways rather than the investigation of trophic effects.

Since the 1960's, there has been an ongoing debate as to whether the trophic effect of nerve on muscle is due to the activity patterns imposed by the nerve on the muscle or to the passage of some type of trophic substance to the muscle. The activity pattern hypothesis is that muscle fibers that are frequently stimulated to contract tend to become specialized for tonic activity. Tonic activity is indicated by slow ATPase (myofibrillar adenosine triphosphatase) and a slow contraction speed coupled with a strong capacity for aerobic metabolism. Conversely, muscle fibers that are used infrequently but with an intensity that can only be supported by anaerobic glycolysis tend to develop a fast ATPase and contraction speed coupled with a reliance on anaerobic metabolism.

For the sake of argument, the activity pattern hypothesis might be countered by the suggestion that it might be the muscle fibers of the motor unit that influence the motor neuron instead of vice versa. Neurophysiologists constantly remind us of the plasticity of neural pathways that enables learning to occur. Perhaps, when an animal learns how to regulate the activity of its skeletal muscles, what might happen is that it may learn to activate the motor neurons that innervate slow-contracting muscle fibers since these more readily give the result that is desired. Alternatively, perhaps the muscle fibers in some way influence the resting potentials of their neurons so that a lowered resting potential enables the more frequent development of action potentials in response to a standard activation of the dendrites.

Experimentally, the major problem in attempts to demonstrate the existence of a possible activity pattern control system is that it is very difficult to set up a clear-cut model experiment. There are two main isolation strategies that may be applied to the nervous system, one is to isolate the muscle fibers by transection of the final common pathway from the motor neuron to the muscle fibers and the other is to isolate the motor neurons from descending and ascending excitatory pathways in the spinal cord. Neither of these strategies is as simple as it first appears. Denervated muscle fibers often become hypersensitive and may spend much of their time twitching wildly (fasciculation) in responses to local irritation, either mechanical or hormonal. Motor neurons in an isolated segment of spinal cord are still subjected to segmental inputs from contralateral and ipsilateral sources. Thus, when the experimenter finally implants a stimulator to put the isolated muscle fibers through a programmed pattern of stimulation designed to simulate either tonic or phasic muscle fiber activity, this programmed activity may be superimposed on an already substantial level of intrinsic activity. There is evidence that extrinsically programmed activity may regulate the metabolic specializations of muscle fibers, but the evidence is not beyond criticism.

In the other camp, the proponents of a control system mediated by the passage of trophic substances have had a lucky break – the elucidation of the mechanism of axoplasmic transport. Augustus Waller in 1852 formulated a law that describes what we now call Wallerian degeneration. In simple language, "Degeneration occurs along the whole length of any nerve-fibre which is cut off from the cell which governs its nutrition." Again, we might note that this concept too was originally described in trophic terms. The events that occur at the proximal stump of a transected axon are noticeably different from the events at the distal stump. The proximal stump bursts into life and numerous small axonal sprouts appear to explore the vicinity of the wound searching for the vacant tubes of distal Schwann cells that can guide the regenerating axon back to its muscle. Schwann cells slide off the proximal stump and appear to help the axonal sprouts to relocate their distal Schwann cell tube. The sprout that gets there first usually becomes dominant while the others regress. The distal stump simply degenerates.

In the early 1900's, axonal degeneration and regeneration attracted many of the great names in neurohistology but, as we saw before, there were more important issues at stake than simply studying trophic effects. Our present understanding of the nervous system is firmly founded on the neuron theory – the theory that discrete nerve cells, often with enormously long axons, are the building blocks of the nervous system. Although the neuron theory goes back a long way in one form or another, in the latter half of the nineteenth century it was under attack from all sides. Some histologists thought that nerve cells were fused together instead of being anatomically separated by synapses, while others thought that the basic conducting elements of nerve fibers were multicellular structures composed of chains of Schwann cells. Against this background, the evidence of embryonic motor axons growing out from the spinal cord to innervate peripheral muscles and the evidence of the proximal stumps of transected axons growing back to their peripheral muscles comprised a major argument for the supporters of the neuron theory led by the great Spanish neurohistologist Ramon y Cajal.

Interest in axonal regeneration as an example of a devel-

opmental process was re-awakened in the 1940's, particularly by the experiments of Paul Weiss and coworkers. The battlefield casualties of World War II, many of whom suffered serious denervation problems, made the medical understanding of reinnervation a top priority. In experimental animals, when constricting rings were clamped around distal nerves that subsequently became reinnervated, it was discovered that the diameter of the restriction limited the diameter of the regenerating axon once it had grown through the ring on its way to the periphery. Upstream of the restriction, towards the motor neuron cell body, the regenerating axon was swollen. When restrictions were removed, the damming of the axoplasm was relieved and the diameter of the distal part of the axon was enlarged. Three essential features of the system thus became apparent: (1) that axoplasm was synthesized in the motor neuron cell body around the nucleus; (2) that the peripheral flow of axoplasm was matched to the axonal diameter and; (3) that there was a continuous catabolism of axoplasm all the way down the axon so that the peripheral endings of normal axons did not explode with the continuous influx of axoplasm.

Axonal Transport

The list of cellular structures that may sometimes be transported down an axon is quite extensive. It includes low molecular weight compounds such as amino acids, sugars and neurotransmitters; large proteins such as enzymes; glycoproteins and components of the cytoskeleton; and organelles such as mitochondria, synaptic vesicles and neurosecretory granules. However, the flow of cellular components down the axon appears to be tightly controlled at the source. Some components of the nerve cell body, such as ribosomes, never get farther than the axon hillock where the axon joins the perikaryon, and there is a considerable degree of selectivity whereby packages of associated materials, such as membrane proteins and lipids, are selectively directed towards certain destinations to travel at certain speeds. There is also a clear separation of components travelling away from the perikaryon (orthograde transport) and components travelling toward the perikaryon (retrograde transport). Thus, when the axon diameter is externally restricted, some degree of damming occurs on both sides of the restriction. Fast-moving components accumulate closer to the restriction than slow-moving components. Components that are large enough to be optically visible have often been seen to move in a series of small jerks (saltatory movement).

The conduction velocities of axoplasmic transport are fairly constant for each group of substances that may be transported, but each group of substances has a characteristic velocity. The separation of different velocity groups is rather arbitrary and varies from author to author, often depending on the type of experimental preparation. Some components move at remarkably fast rates up to about 400 mm/day. These are usually membranous components that are en route to the axon terminal or neuromuscular junction. The slowest conduction velocities (about 0.5 mm/day) are found among the cytoskeletal components that appear to be replacement parts for along the whole length of the axon. Retrograde transport appears to be dominated by worn-out cellular components that are returning to the lysosomal system of the perikaryon for recycling. However, substances that have been taken up peripherally by endocytosis may also appear in the retrograde stream. Thus, although axoplasmic transport might well be the vehicle by which trophic substances are moved from the neuron to the muscle fiber, movement in the return direction is also feasible. In the absence of any clear proof to the contrary, it seems reasonable to keep this possibility in mind. Conventional physiology, what Thomas Kuhn has called our current paradigm, teaches us that many physiological control mechanisms have a two-way flow of information. Axoplasmic flow is a mechanism that allows for this possibility in the case of the neurotrophic regulation of muscle metabolism.

Neurotrophic Factors

The tacit assumption in much of the research on possible neurotrophic factors is that some as yet unidentified substance passes from the motor neuron to the muscle fiber and then modifies protein synthesis in the muscle fiber. The implication is usually that the control system is qualitative in nature. In other words, that there might be different substances to produce different end results. This does not, however, preclude the possibility that neurotrophic factors might also function in a quantitative manner. If a particular substance produces a certain end result, then a high concentration might produce a strong response and vice versa. However, suppose that the muscle fiber has a basic pattern of gene expression that might, for the sake of argument, cause the muscle fiber to exhibit strong ATPase activity, a fast contraction speed and a reliance on anaerobic metabolic pathways. In this case, a single neurotrophic factor that induced the opposite response might change muscle fiber metabolism in a way that was proportional to the amount of the substance. When we start to consider possibilities such as this, it becomes apparent that, whatever the nature of the neurotrophic mechanism, it must be able to cope with the enormous increase in muscle fiber volume that occurs during postnatal development. In other words, a motor neuron in an adult animal with greatly hypertrophied muscle fibers might have to synthesize a lot more trophic substance to maintain control over its motor unit. On the other hand, it might not. And in this case, muscle fiber metabolism might undergo some type of transformation. The reason for pushing this point may be obvious to all those who have investigated histochemical fiber-type ratios in growing meat animals, since fiber-type ratios change with age. If the histochemical characteristics of the muscle fiber are neurally regulated, then something must be changing in the neurotrophic control system as animals grow to market weight.

While we are expanding our horizons in this way, we may as well introduce yet another possibility. There is certainly some evidence that a neurotrophic factor (or factors) passes from the neuron to the muscle fiber, but is that the only source throughout the animal's lifespan? On scanning some of the thousands of published scientific papers in this area, a striking feature is the similarity of growth-promoting substances in the embryo and those thought to be produced by motor neurons in adult animals. Embryonic tissue is a rich source of growth-promoting substances, many of which have now been characterized as somatomedin or somatomedin-

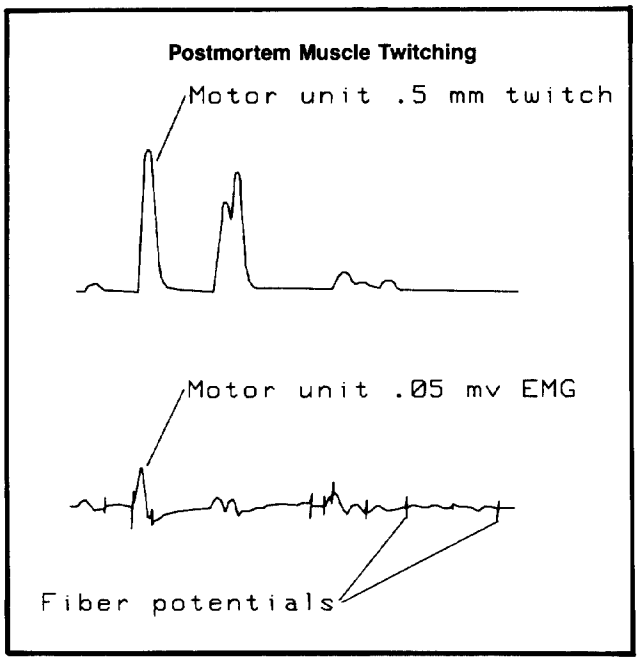
like substances (such as MSA – multiplication-stimulating activity). In the embryonic environment, it appears that muscle fibers are able to develop fairly well when experimentally deprived of their innervation. Around the time of birth, however, the level of circulating growth-promoting factors begins to decline toward adult levels. This is just about the time that the survival of the muscle fiber often becomes dependent on its motor innervation. It is tempting to attribute something more than mere coincidence to this timing. This is an important point relative to practical agriculture. Off-hand, it is rather difficult to see how we could manipulate neurotrophic factors if they can only be delivered down motor axons. However, if they can also be delivered as circulating factors in the blood stream, then commercial implementation is one step nearer.

Oh and Markelonis (1982) have been working for some time on a glycoprotein called sciatin that can be isolated from chicken sciatic nerves. It has a number of trophic effects on cultured muscle cells. It enhances their rate of maturation, it sustains them in the absence of any innervation, it increases their rate of protein synthesis and it increases their rate of acetylcholine receptor production. Sciatin is an effective substitute for the embryo extract that is normally added to routine culture media to make cells flourish. Oh and Markelonis found that sciatin is structurally similar to transferrin, the iron-carrying serum protein of vertebrates. Transferrin and sciatin appear to be identical in their biological effect on developing muscle. Results such as this cannot fail to interest anyone involved in the manipulation of muscle growth in meat animals.

Electrophysiology of Meat

The starting point for my research on the electrophysiology of meat was my curiosity concerning postmortem fasciculation – the frantic twitching of muscle fibers that may be observed in beef neck muscles when the carcass is dressed. The prevailing view at the time was that the cellular integrity of muscle fibers was lost once all their ATP had been depleted. Everyone knew that red and white muscles differed in their rates of postmortem glycolysis, but there was no published evidence that individual red and white fibers did the same thing. The periodic acid – Schiff (PAS) reaction for glycogen, however, revealed otherwise and showed that different histochemical fiber types had different rates of postmortem glycogenolysis, depending on their situation with regard to temperature and degree of postmortem stimulation (neural as well as electrical stimulation). The histochemical evidence of cellular integrity being maintained for some time postmortem naturally prompted a search for physiological evidence of the same phenomenon. If individual muscle fibers could twitch postmortem, then they might also maintain independent action potentials and resting potentials postmortem. This had been shown previously in intracellular recordings made on the fibers of intact longissimus dorsi muscles in stress-susceptible and control pigs (Schmidt et al., 1972). In due course, resting potentials and action potentials were detected in excised samples of bovine muscle that exhibited fasciculation – there was even evidence of the survival of motor unit integrity in excised muscle (Figure 2). From this point, two separate lines of investigation were developed – (1) neuromuscular systems in meat and, (2), membrane capacitance.

Figure 2



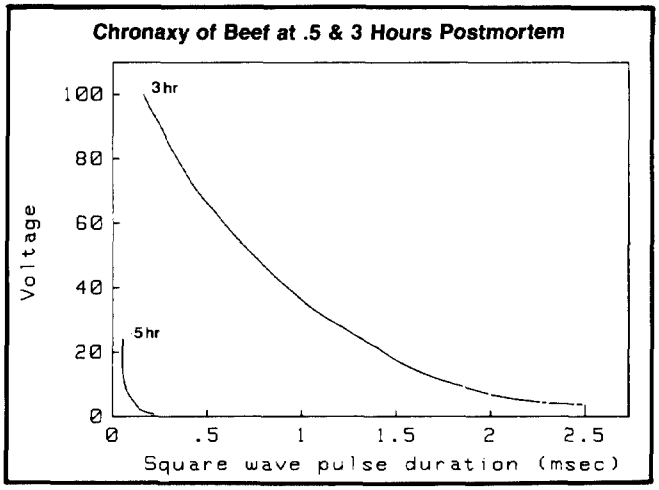
Neuromuscular Systems in Meat

The patterns of postmortem glycogenolysis that were detected in quiescent muscles were relatively straightforward. In muscles that were electrically quiet when monitored with surface EMG (electromyography) electrodes, the red fibers (with weak ATPase and strong aerobic enzyme activity) had the most rapid rate of glycogenolysis (when corrected for their initial postmortem glycogen concentration) and the white fibers (with strong ATPase and weak aerobic enzyme activity) had the slowest rate of glycogenolysis. This pattern was dramatically reversed in electrically stimulated muscles. During early investigations, the experimental material was composed of long lengths of otherwise intact beef sternomandibularis muscle. The first electrodes that were used were metal plates on the muscle surface, but the system was later changed to a pair of hypodermic needle electrodes inserted perpendicularly through the muscle. The needles were inserted through the muscle several centimeters apart near the midlength.

Acting initially in the belief that the electrical stimulation acted directly on the muscle fibers, it was anticipated that muscle fibers that had been directly impaled by the stimulatory electrodes would exhibit more rapid or complete glycogenolysis than those that were not directly impaled and which were located at a distance of several centimeters away from the electrodes, particularly those fibers at the ends of the long strip of muscle. The results of the experiment, however, suggested otherwise. It was never possible to demonstrate such an effect, even with very weak stimulation, and the usual result of stimulation by this method was that all the white fibers throughout the muscle exhibited a similar acceleration of glycogenolysis. This suggested that the stimulatory current was acting through the nervous system, routed either through terminal axons or through neuromuscular junctions.

To test this idea, curare in physiological saline was injected interfascicularly throughout the muscle and the muscle was subjected to several minutes of massage. The control samples were injected with saline but without curare. The saline alone had quite an effect but, in comparing the muscles with and without curare, there was a noticeable difference. Physiologically (stimulus duration or strength versus contractile response) and histochemically (glycogen depletion), the curare-treated muscles behaved like muscles that had undergone a delay of several hours between slaughter and stimulation (Figure 3). In other words, curare greatly reduced the effect of electrical stimulation on muscles from recently slaughtered animals but had no effect when there was a delay of several hours between slaughter and stimulation. From these findings, it was concluded that the strong response of muscles stimulated soon after slaughter was mediated through the nervous system but that this pathway was progressively lost postmortem so that late stimulation could only act directly on the muscle fibers. Research along this line of investigation then moved on to the next most obvious topic – the survival times of neuromuscular pathways postmortem.

Figure 3



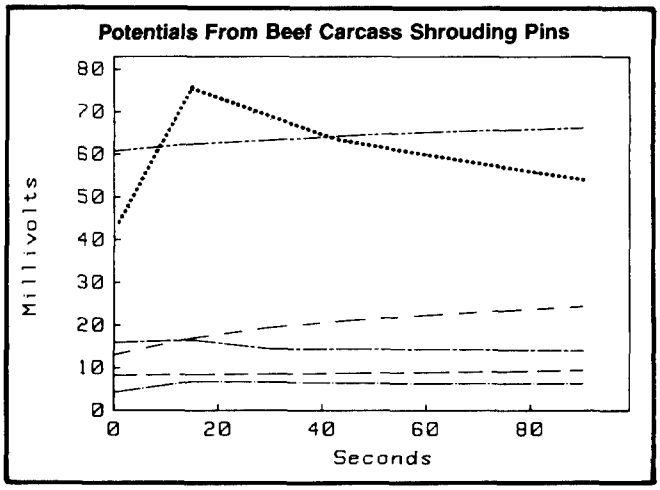
Electrical Capacitance of Meat

Before moving on to consider the survival times of neuromuscular pathways, we will return to complete the other aspect of the investigation – membrane capacitance. There are two main technical problems in recording intracellular potentials in meat. The first is quite simple: If a muscle fiber gives a good strong twitch, it often destroys the microelectrode with which it is impaled. Since muscle fibers may twitch when they are first impaled, it is sometimes quite difficult to obtain good recordings. The second problem is electrical capacitance. Many of the conducting elements of the microelectrode system are able to store surface charges like the plates of an electrical capacitor. Then, when rapid physiological events such as action potentials occur, the true waveforms of the events are rounded-off by the ebb and flow of current in and out of the capacitive components. To minimize the effects of this phenomenon, it is customary to balance the system capacitance against a variable capacitor that acts in the opposite direction. The muscle fiber membranes themselves also contribute toward the system capacitance that must be balanced.

With square wave stimulation of meat, the main problem is polarization. The passage of square wave pulses (an interrupted direct current) causes electrochemical changes on the electrode surfaces. These changes effectively transform the meat-electrode system into a miniature battery that resists subsequent pulses. The voltage produced by this system is soon quite substantial and is sufficient to power a flashlight bulb connected to the electrodes after the stimulator has been disconnected. The battery effect in the meat prevents the use of a direct current measurement of meat resistance using an ordinary ohmeter, and what must be used is an alternating current bridge circuit in which the test current oscillates so fast that it neither causes muscle contraction nor allows galvanic effects to build up as a result of electrode polarization. The battery effect also exists with any metal electrodes that have not been carefully cleaned with abrasives. Figure 4, for example, shows the voltages that may be recorded from ordinary stainless steel shrouding pins on a beef carcass.

When an AC bridge was constructed with a dual trace

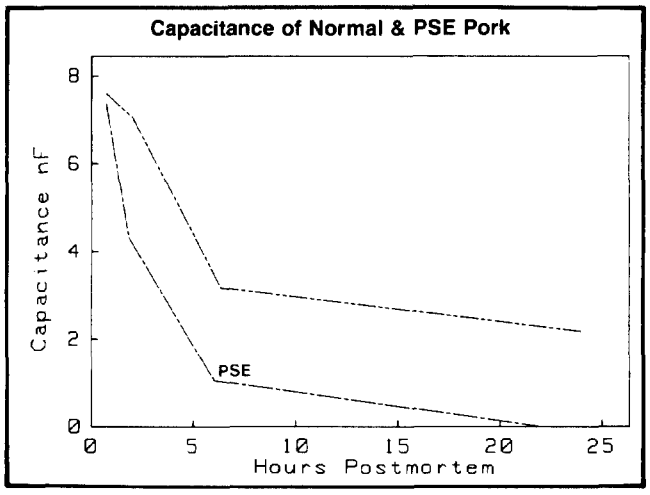
Figure 4



oscilloscope to balance the arms of the bridge, it was found that the waveform passing through the meat was substantially delayed by its capacitance. Thus, as the current flowed into the meat on the rising slope of the sinusoidal wave, it charged up the meat and lagged behind. Conversely, on the descending slope, the meat discharged its capacitance. In other words, the problems involved in measuring the electrical resistance of meat were rather similar to those that had been encountered earlier in the measurement of muscle fiber resting potentials. In examining the effects of time lapse postmortem on meat resistance, the balance capacitance values were included as one column in the computer matrix of resistance values. On calculating the appropriate linear regressions of resistance changes with time, capacitance turned out to be more interesting than resistance. To cut a long story short, capacitance declined more rapidly postmortem in PSE (pale, soft, exudative) pork than in normal pork (Figure 5).

The first explanation of this phenomenon which was proposed was that the muscle fiber membranes of the PSE pork

Figure 5



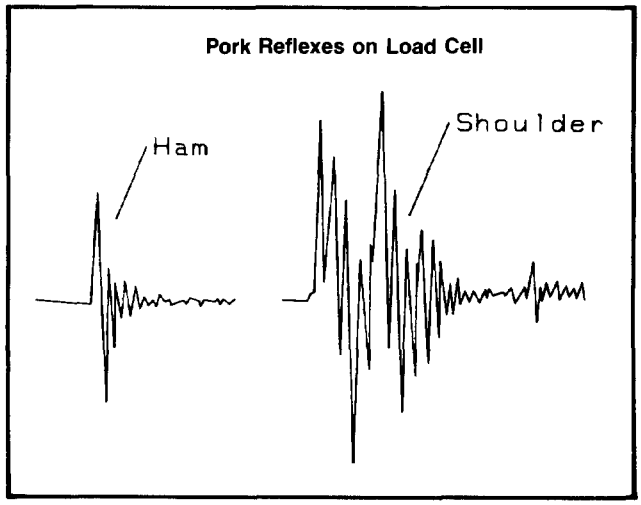
had been damaged by lactic acid. The values for capacitance that may be measured in meat are surprisingly high and are measured in nanofarads rather than in the more familiar picofarads of small electronic components. An analogy with plate capacitors suggested that high capacitance might originate from large areas of membranes with a high dielectric constant. Although muscle fiber resting potentials are measured in millivolts, the membranes that maintain this differential are extremely thin. If scaled up to a macroscopic level, they would insulate against several thousand volts. The conducting elements that were thought to connect these large sheets of membranes were the intracellular and extracellular electrolytes of muscles. Thus, it was thought that lactate-induced membrane damage might resemble small holes that allowed the mingling of the various electrolyte compartments so that the membrane capacitance was short-circuited. To test this idea, a few dark-cutting beef carcasses were measured, reasoning that, since they had a high pH, they should also maintain their high values of initial postmortem capacitance. The results, however, were the other way around. Dark-cutting beef had capacitance values slightly less than normal when appropriate corrections were made for the time lapse postmortem. To solve this dilemma, what had to be discovered was the factor that was common to both PSE pork and dark-cutting beef. The most obvious feature that they have in common is that both PSE pork and dark-cutting beef deplete their ATP at a relatively early time postmortem. PSE pork does so by having a very rapid rate of glycolysis, and dark-cutting beef does so by having virtually no postmortem glycolysis. In cooperation with Thayne Dutson, sides of beef were unilaterally subjected to electrical stimulation so as to generate meat with different rates of postmortem glycolysis. It was found that the relationship between pH and capacitance was, at most, only sporadic whereas ATP levels were correlated with capacitance over quite an extensive range (Swatland and Dutson, 1984).

This is as far as we have progressed at present, and some fundamental questions remain unanswered. Does capacitance originate from both the sarcoplasmic reticulum as well as the plasma membrane and T tubules? What is the structural nature of the sites that allow membranes to be short circuited when the ATP has been depleted?

Postmortem Reflex Activity

Most of our present abattoirs were planned and built with little or no consideration for the postmortem reflex activity of slaughtered meat animals. Consequently, abattoir workers are sometimes exposed to dangerous situations and the neural activation of muscle contraction and postmortem glycolysis is more or less uncontrolled. The problem is particularly acute with the slaughtering of pigs since the methods of inducing unconsciousness are less reliable and accelerated rates of pH decline have a serious effect on meat quality. The fact that postmortem reflex responses involve the integrated activity of large parts of the body indicates that these reflexes must be neurally regulated at the level of the spinal cord and possibly higher in the central nervous system (CNS). Large-scale reflex activity can be measured quite easily by incorporating a load cell into the shackling chain and then collecting the output on a chart recorder or microcomputer. Quite often it is possible to identify the type of reflex activity from the load cell recording. In pork carcasses, for example, a kick from one ham gives a simple recoil pattern while a kick from a foreleg gives a more complex pattern (if the other leg moves passively) of larger amplitude, as shown in Figure 6.

Figure 6



Although there are a number of minor types of reflex activity that may persist for a long time postmortem, the most damaging types of reflex activity are those that occur immediately after stunning and during exsanguination. Electrocorticography (ECoG) with electrodes located directly on the motor cortex shows that early postmortem episodes of reflex activity are correlated with brain activity (Figure 7). It is difficult, however, to attribute a cause and effect relationship to these observations since the activity observed in the cerebrum might be a sensory response to muscle activity initiated at a lower level in the CNS. Figure 8, for example, shows the types of ECoG activity that may be detected over the motor cortex when various mild stimuli are applied to parts of the body. The large peaks on these traces corresponded to the point of stimulation in a regular manner. The examples shown, however, were taken from a mildly sedated pig. In normal, actively struggling pigs, such simple patterns

Figure 7

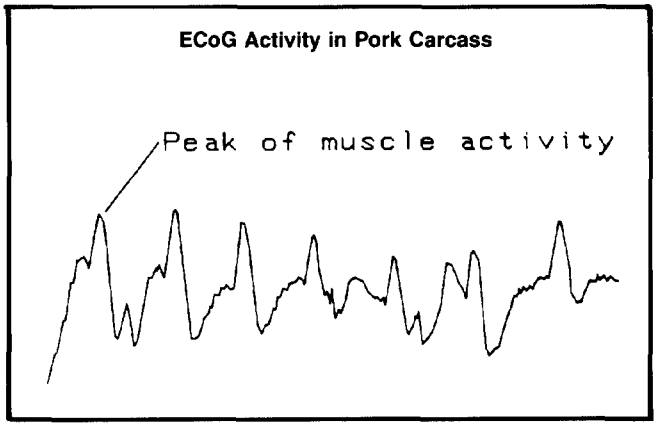
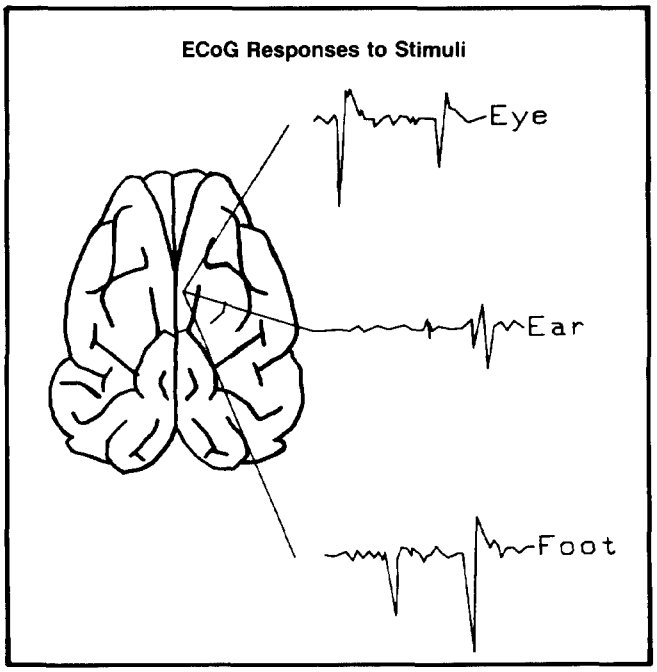


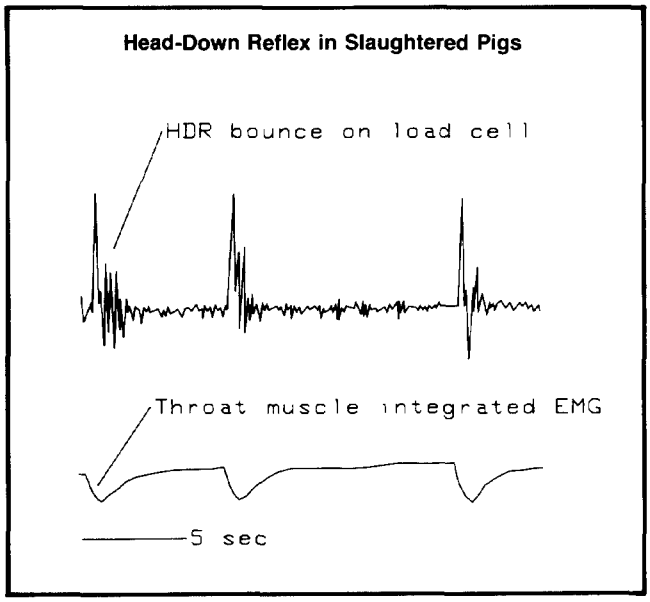
Figure 8



of ECoG activity were totally obliterated. Thus, my experience with ECoG is that it cannot easily be used to study loss of consciousness during the slaughtering of pigs, although researchers in Europe appear to have had more success. Reflex activity in the hindlimbs is affected by shackling and, when shackling follows the normal unilateral pattern, a trend toward more extreme PSE may be seen in the unshackled hams.

The causes of postmortem reflex activity in the ham, shoulder and loin are extremely difficult to investigate by EMG and ECoG in the abattoir because of the violence of the muscle contractions. A more easily investigated reflex is the "head down reflex" that is often seen toward the end of exsanguination when the ham reflexes have subsided (Figure 9). This reflex has been tracked down by EMG to a localized response of the ventral throat muscles associated with a coughing reflex, as if the pig were attempting to clear

Figure 9



an obstruction in the trachea. A "head back reflex" has also been described but this is less commonly seen. This reflex involves quite extensive bilateral contractions of the longissimus dorsi so that the head is thrown dorsally. Reflexes such as this might possibly explain some of the variability in the rate of postmortem pH decline found in the longissimus dorsi.

A further point that complicates attempts at the scientific investigation of postmortem reflexes is that there is a large variation in the results obtained by different slaughterers. Even when stunning amperages are monitored electronically, when the angle of the sticking knife ventral to the sternum is standardized and when the rates of exsanguination monitored electronically are almost identical, some operators are still able to slaughter a pig without producing any large-scale postmortem reflex activity. Other operators, however, may cause so much reflex activity that the pig drops off the shackling chain. Somewhere in this system is a trade secret that we must identify and explain in terms of the animal's postmortem physiology.

Conclusion

The nervous system offers an interesting field of research for the meat scientist. At one extreme it may include the neurotrophic control systems that regulate muscle fiber differentiation and growth – perhaps even the numbers of muscle fibers. At the other extreme, the nervous system plays a major role in determining the rates of postmortem glycolysis – at least in normal pork carcasses. The nervous system may facilitate an electrical stimulation of beef carcasses and is involved in animal responses to different slaughter methods. Research on the nervous system of meat animals offers the advantage of combining work on short-term technological objectives, such as improving slaughter methods, with long-term research on the nature of muscle growth in meat animals.

References

Oh, T.H.; Markelonis, G.J. 1982. Chicken serum transferrin duplicates the myotrophic effects of sciatin on cultured muscle cells. J. Neurosci. Res. 8: 535-545.

Schmidt, G.R.; Goldspink, G.; Roberts, T.; Kastenschmidt, L.L.; Cassens, R.G.; Briskey, E.J. 1972. Electromyography and resting membrane potential in longissimus muscle of stress-susceptible and stress-resistant pigs. J. Anim. Sci. 34: 379-383.

Swatland, H.J. 1984. Structure and Development of Meat Animals. Prentice-Hall, New Jersey.

Swatland, H.J.; Dutson, T.R. 1984. Postmortem changes in some optical, electrical and biochemical properties of electrically stimulated beef carcasses. Can. J. Anim. Sci. 64: 45-51.