Properties and Behavior of Prerigor Meat

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In several areas of meat science, the investigator may safely ignore the phenomenon of rigor mortis, secure in the knowledge that early-postmortem changes will have little or no influence on his results or conclusions. In most meat studies, however, no such comforting assumption can be made with any confidence at all. PSE pork and cold-shortened beef and lamb are perhaps the most conspicuous problems that demand a knowledge of early-postmortem events for their solution, but they are by no means the only such examples. A very wide range of meat properties, and of processes to be imposed on the tissue, are dependent on the nature, the extent, and the rate of changes in those first few critical hours following slaughter; we may note in particular water binding, tenderness, emulsification, color, bacterial control, juiciness, fermentation and aging. It is true, of course, that understanding will not necessarily provide instant answers. But it is also true that most current problems are still with us simply because they have withstood all our empirical, try-it-and-see onslaughts, and will remain with us at least until we comprehend their origins and mechanisms.

This is particularly so when we come to address the subject of hot boning, for in no area of meat research are we more dependent on a knowledge of the living tissue. The word "living" is the key if we are to enter this field; the animal may be dead, but its musculature remains alive (even though irreversibly dying) until rigor is finally established. Its reactions, its behavior, and in particular its responses to imposed conditions and provocations are those of living muscle, and many of the effects we induce at this time will carry over to influence the properties and qualities of the inert end-product. If we have no liking for the dynamic nature of early-postmortem muscle, or if we pay only polite lip service to the concept that meat and muscle are one and the same tissue, then we have no business in hot-boning research.

My fairly obvious intentions in this paper are, first, to examine the conditions and changes in muscle that lead to rigor mortis; second, to note the variability in rigor pattern within and among species; then finally, to relate our current knowledge of these early-postmortem events to the practical scene. My less obvious (but by no means concealed) objective is to encourage a wider interest in the early post-slaughter period, for it is then that muscle is uniquely vulnerable to a wide range of insults: heating, cooling, freezing, excision, salt addition, electrical stimulation, and perhaps other aggravations yet to be devised.

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Early-postmortem Events

Our knowledge of rigor mortis is based on the strong foundation laid in the pioneering studies of rabbit psoas muscle by Bate-Smith and Bendall (1947, 1949) and Bendall (1951). Their results were confirmed and extended to several other species during the next decade: whales (Marsh, 1952), horses (Lawrie, 1953), cattle (Marsh, 1954), sheep (Marsh and Thompson, 1958), and pigs (Lawrie, 1960)—a rather curious chronological order that is partly explained by the extremely severe shortage of meat in Britain during the years following World War II. Much additional information from peripherally related fields has been added since that time, the sliding-filament concept of contraction (Hanson and Huxley, 1955) in particular accounting very elegantly for the declining extensibility that characterizes rigor development. A very thorough account of rigor mortis and related phenomena was given by Bendall (1973).

Of the many processes that are either triggered or arrested by death, only the cessation of blood flow is needed to initiate the train of events culminating in rigor. Two consequences of this circulation arrest are responsible for all ensuing events: no more oxygen enters the tissue, and no more waste products leave it. The oxygen in the musculature at death is exhausted within a very few minutes, and all oxidative metabolism ceases; but those processes that are not oxygen-dependent are unaffected. Thus glycogen continues to break down, but normal metabolism beyond pyruvate is impossible because of the oxidative nature of the later changes (Fig. 1). Instead, pyruvate is converted to lactate, and this accumulates in the tissue because there is now no circulation to remove end products; it usually attains a wet-weight concentration of 1% or more, and is the component responsible for the normal decline of muscle pH from about 7 at death to roughly 5.5 after rigor onset.

The degradation of glycogen is always accompanied by the regeneration of adenosine triphosphate (ATP). The anaerobic phase of glycolysis produces significant but quite small amounts of ATP; it is only in the oxidation phase (pyruvate to CO_2) that ATP resynthesis is really efficient. With the exhaustion of oxygen just after slaughter, therefore, the sudden transition from an aerobic to an anaerobic metabolism causes ATP

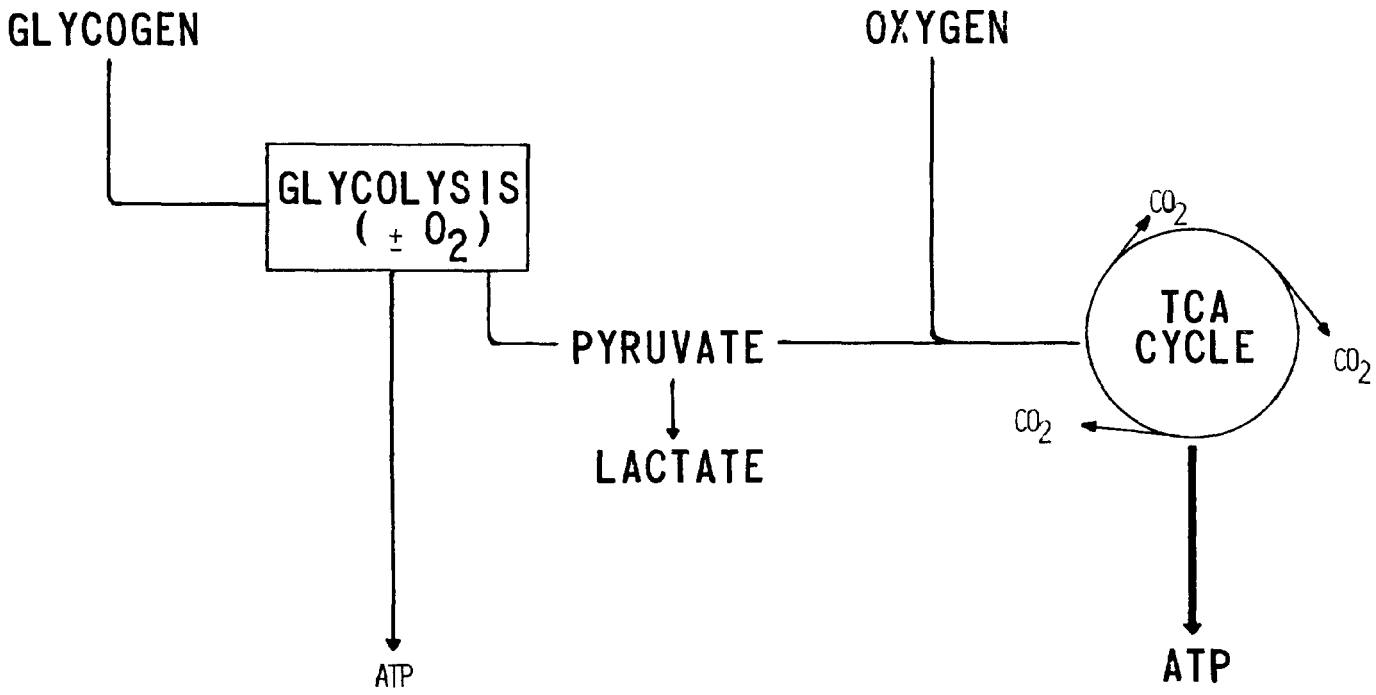

Figure 1. Glycogen breakdown and ATP regeneration in living ($+O_2$) and postmortem ($-O_2$) muscle.

regeneration to decline very considerably. In fact, ATP is glycolytically produced somewhat more slowly than it is being enzymically split, and we might reasonably expect, therefore, that it would start its irreversible decline within minutes of slaughter, as soon as the last traces of oxygen have been consumed.

Except in rather special circumstances, however, this does not occur. ATP does not fall from its at-death level for some time, and several hours may elapse before any decrease is detected. This delay in ATP disappearance is due to the presence of the high-energy compound creatine phosphate (CP), and of the enzyme creatine phosphokinase (CPK) which promotes the rephosphorylation of ADP as long as supplies of CP last (Fig. 2): ADP + CP → ATP + C. (A second resynthesizing mechanism also assists, but to a lesser extent; myokinase rephosphorylates one molecule of ADP at the irreversible expense of another: 2ADP → ATP + AMP.) Eventually, however, all of the CP reserve is exhausted, and the initial level of ATP can be maintained no longer; the rapid phase of rigor onset has replaced the delay phase.

The inter-relationships of these separate processes can now be brought together in a single diagram (Fig. 3), taken from Marsh (1977). Muscle pH falls steadily from slaughter until rigor is complete; creatine phosphate declines to zero during the first few postmortem hours as it strives to maintain the ATP level; and the ATP content commences its rapid fall when creatine phosphate is exhausted. Changes in muscle extensibility precisely parallel those in ATP content; in prerigor muscle, it is the presence of ATP which prevents cross-bridging between the thick and thin filaments (upper diagram of Fig. 3), thereby allowing these filaments to slide over each other when the muscle is loaded or unloaded. With a falling ATP level, however, progressive cross-bridging occurs between the two sets of filaments, and sliding becomes increasingly difficult. When ATP reaches a very low level, the structure's extensibility is correspondingly very low—perhaps only 5-10% of that in the tissue's prerigor state—and rigor is established.

Variability in Early-postmortem Behavior

This basic pattern of early-postmortem events applies in general terms to all species so far examined. There are significant variations among species, however, and many examples could be quoted to illustrate the dangers of extrapolation from one to another. Rigor completion takes appreciably longer in beef than in rabbit psoas muscle, for instance; yet the prerigor delay phase persists for a greater period in the rabbit (because of the latter's much higher content of creatine phosphate). Cold shortening, so prominent and troublesome in bovine and ovine muscles, is undetectable in rabbit psoas (because, we believe, the latter contains too few mitochondria to release enough calcium ions for contraction: Buege and Marsh, 1975). Rigor onset and its accompanying pH decline may be precipitously rapid in porcine muscle and in an occasional unfortunate human being under anesthesia. Glycolytic rate may be far from uniform among corresponding muscles in different beef carcasses (Tarrant and Mothersill, 1977; Bendall, 1978), and we have recently detected remarkably wide ranges of early-postmortem pH within single bovine longissimus muscles (Marsh, Lochner, Takahashi and Kragness, 1981). Whale muscle sometimes displays a still-unexplained "stationary phase" in which pH, ATP and extensibility maintain their strictly prerigor values for 18 hours or more before normal rigor onset commences (Marsh, 1952). This pattern perhaps parallels the bizarre observations made over a cen-

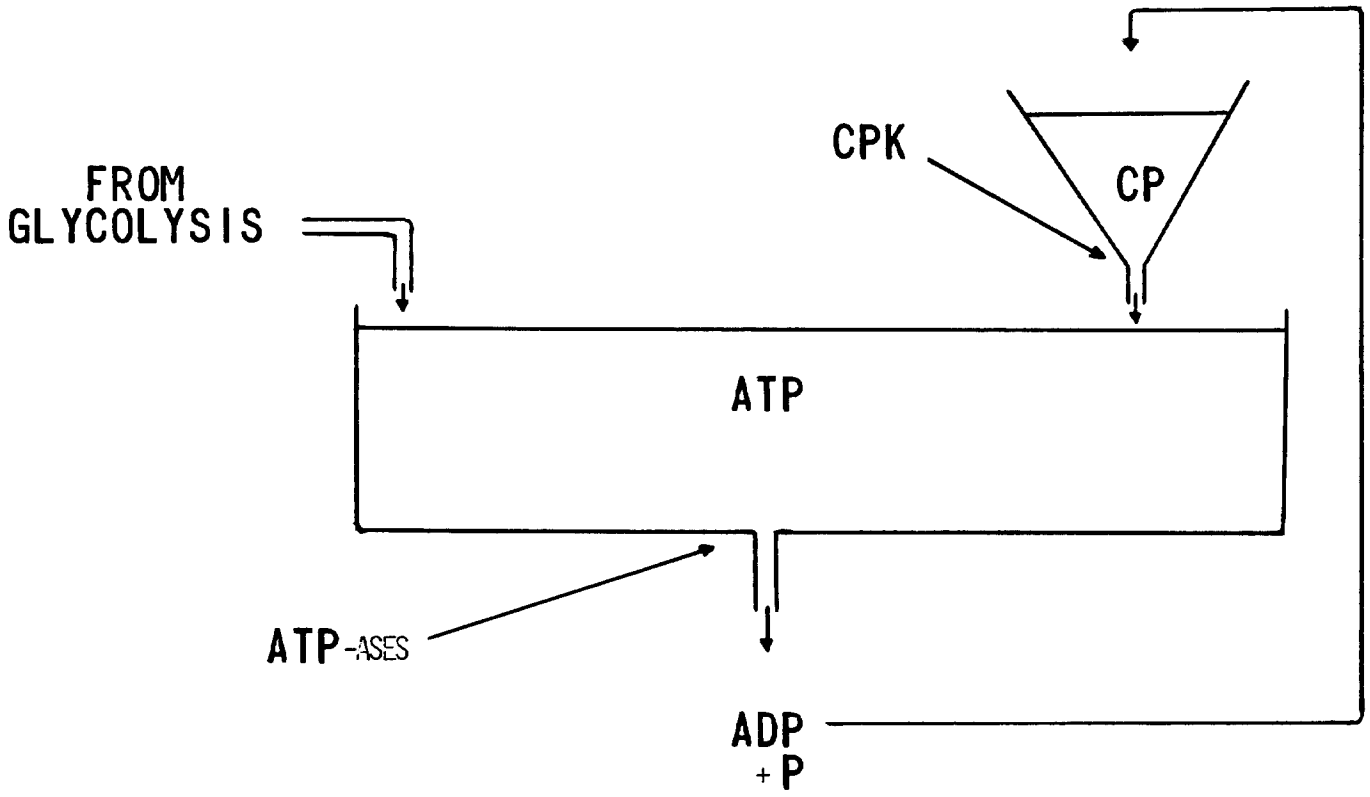

Figure 2. Maintenance of ATP reservoir in prerigor muscle by combined action of anaerobic glycolysis and rephosphorylation of ADP by creatine phosphate.

tury ago on French criminals, whose muscles remained contractile for 13-26 hours after guillotining (quoted by Taylor, 1905).

Externally imposed conditions, too, can greatly influence the rigor pattern. Glycogen depletion in the live animal, if taken far enough, will result in an elevated ultimate pH in the meat, simply because less lactic acid can be formed in the early-postmortem hours; dark-cutting beef and DFD pork are likely consequences. Exhausting exercise just prior to slaughter may not cause a significant elevation of ultimate pH, but it certainly can produce a significant elevation of lactic acid in the musculature at the moment of slaughter, so the overall time to rigor onset is shortened appreciably. Finally, postmortem muscle temperature strongly influences the rigor pattern, higher values accelerating the process appreciably. It is to be noted in this connection that muscle temperature is the resultant of many variables, including carcass size, shape and fatness; a mere recording of air temperature and velocity is thus of very little value in rigor-related studies.

Rigor-related Effects on Beef Quality and Properties

Of the early-postmortem events and conditions that influence meat properties, many are independent of the time at which the muscle is excised. Thus a muscle will reach the same ultimate pH, through glycolytic acid production, regardless of the time of boning: 0, 10 or 100 hours postmortem. These aspects we may safely ignore in specific relation to hot boning, in order to concentrate on those pre-rigor features whose effects are strongly influenced by the length of time between slaughter and excision.

1. *Shortening:* Appreciable shortening may accompany rigor onset at higher temperatures, but it is a length change of little or no practical significance since it will be encountered only in very unusual circumstances or under contrived experimental conditions. Of much greater importance is the phenomenon of cold shortening (Locker and Hagyard, 1963), which may be accompanied by massive toughening. Skeletal restraint does not necessarily prevent its occurrence, but certainly does reduce its intensity, so removal of this deterring influence by hot boning clearly increases the likelihood of shortening and toughening problems. Strongly overshadowing this restraint factor, however, is the greatly accelerated cooling that may take place in hot-boned meat if specific precautions are not taken to oppose it. The great insulating effects of both fat cover and sheer bulk are sometimes overlooked, and when both of these chilling retardants are removed by hot boning, special means must be introduced—either a drastic slowing of the chilling operation, or a several-fold hastening of rigor onset—to prevent or limit the length change. More will be said later of rigor acceleration as a preventive measure.

   Increasing interest in hot boning raises the possibility of a third, and this time extreme, form of length change: thaw shortening or thaw rigor. This is the great contraction-like effect, associated with toughening and drip exudation, that

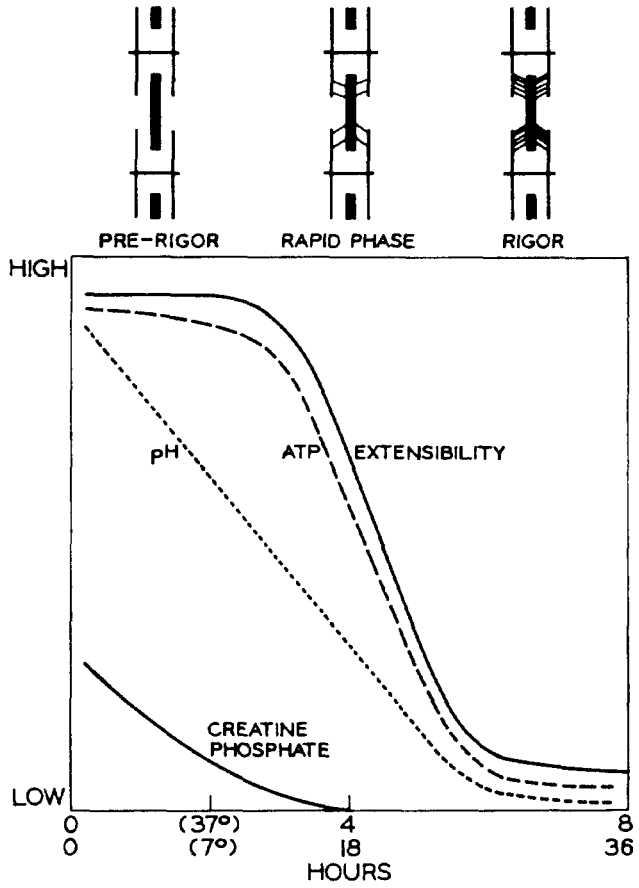

Figure 3. Rigor onset in beef at 37°C and 7°C, and cross-bridge formation between thick and thin filaments as rigor develops.

accompanies the rapid thawing of meat previously frozen *before* rigor completion. No great imagination is needed to envision a future situation in which hot-boned meat, frozen rapidly, is later thawed by micro-wave to accelerate further processing. Thaw rigor first appeared as a practical problem in Antarctic whalemeat in 1947 (Sharp and Marsh, 1953), and arose because of the need to dispose of the carcass quickly and of the impossibility of freezing whales as carcasses or sides. It became a much more serious problem in the New Zealand packing industry in the mid-sixties, when lamb carcasses were exposed to early-postmortem blast freezing (Marsh, Woodhams and Leet, 1968). It can be overcome by measures similar to those used to prevent cold shortening, but this time the tissue should be *completely* in rigor (and not merely approaching the rigor state) before low temperatures are attained.

2. *Aging:* Despite the great influence that cold shortening can exert on meat quality, it is now clear that most of the tenderness of variability in beef *chilled as sides* cannot be accounted for by this effect. If very lean or small carcasses are exposed early-postmortem to very severe chilling conditions, of course, cold shortening and its associated toughening will take place, and the same thing will happen, though to a greater degree, if the hot-boned product receives a similar treatment. Yet beef in which cold shortening has been prevented completely is not necessarily tender. We must face the fact, long overlooked in our enthusiastic pursuit of the shortening/toughness connection, that mere suppression of the length change does not convert beef to a desirable product. The absence of toughness does not signify the presence of tenderness. There is a "neutral" zone between the extremes; cold shortening forces the meat from neutrality to toughness, whereas aging moves it in the other direction, from neutrality to tenderness. Certainly we must prevent shortening if the product is to be even marginally acceptable, but much more is needed if we are to claim it as desirable.

   Aging appears at first sight to be incompatible with hot boning, a principal aim of which is to accelerate operations and move the product quickly into distribution channels or further processing stages. Aging (it is argued) is a prolonged process occupying at least several days; besides (it is claimed), aging commences only with the attainment of a low pH and a rigor or near-rigor state (Davey and Gilbert, 1976; Chrystall and Devine, 1980; Dutson, Smith, Savell and Carpenter, 1980). Yet a recent Wisconsin study (Lochner, Kauffman and Marsh, 1980) indicated the importance of very early-postmortem temperature as a determinant of beef tenderness, and suggested that body-temperature maintenance in this period is the principal reason for the superior quality of well-finished beef. In a follow-up investigation (Marsh and Lochner, 1981), very significant tenderizing was shown to result from a 2-3 hour exposure of lean beef sides to 37°C, while the musculature was still prerigor and of high pH. This observation we interpret as clear evidence that prerigor aging can and does take place if the right temperature conditions prevail, and occurs at a very high rate. I propose, therefore, that a quite brief, very early-postmortem, 37°C aging, prior to hot boning, might be worthy of your serious consideration. While not greatly impeding rapid carcass disposal, the treatment would do much more than eliminate shortening-induced toughening; it would quickly raise the quality from the neutral to the desirable zone.

3. *Prerigor Cooking:* For some products reaching the consumer in ready-to-eat form, the potential advantages of hot boning would be further augmented if cooking could follow immediatley on the heels of prerigor excision. Results of studies in this area are not always consistent, and I want to suggest that an improved knowledge of prerigor events could both account for the observations and point the way to end-product improvement.

   The application of heat to early-postmortem muscle accelerates or activates several processes. Glycolysis is speeded greatly because of its high and increasing Q_{10} in the range of about 30-45°C, but is terminated (probably at 48-50°C) by heat inactivation of one or more of its enzymes. Contraction is triggered, presumably by calcium-ion release, a great sliding-filament type of shortening taking place at about 40-45° and attaining 50% or more if the tissue is still strictly prerigor. Finally, heat denaturation of the contractile machinery in the 50-55° range prevents further length changes of the sliding-filament variety.

   With this information, we can now explain some of the discrepancies and conflicting results. The key is heating rate.

With extremely rapid heating—say microwaving of small pieces (Cia and Marsh, 1976)—glycolysis is arrested almost as soon as its acceleration phase has commenced. The pH thus remains high, and the tissue is still strictly prerigor when the heat stimulus for contraction occurs; the shortening is thus very large, and is accompanied (just as in high cold shortening) by supercontraction and fiber fracture. The final product is thus highly distorted through its great length change, highly retentive of water through its high pH, and very tender because of the marked fiber rupture. With somewhat slower (but still quite rapid) heating, glycolysis is accelerated, and this time it proceeds further because a longer time is needed at this reduced heating rate to reach the enzyme's lethal temperature. Progress toward rigor is thus further than before, and some cross-bridging occurs before the heat stimulus triggers contraction. Contraction is consequently smaller, though still appreciable; it is large enough to produce toughening, but insufficient to cause supercontraction and fracture. Compared with the earlier product, therefore, this material will be somewhat less distorted because of its smaller shortening, less water-retentive because of its lower pH, and appreciably less tender because of the absence of fiber rupture.

This explanation, though still partly conjectural, appears to account for the observed effects. More important for our present purpose, however, is the illustration it provides of the complex interplay among several quite transient components and changes during the early-postmortem period, and of the great influence upon them of relatively small temperature differences. In more general terms, the example stresses the necessity of adopting a highly dynamic approach to the whole area of prerigor manipulation, handling and processing, and the need to integrate several concurrent mechanisms if we are to understand, control, and perhaps improve the quality of the product.

4. *Electrical Stimulation:* Most of what can be said about electrical stimulation has been said repeatedly in recent times. Since it is a process applied exclusively to meat in a prerigor state, however, it is appropriate to relate its more obvious effects to its mode(s) of action. Three mechanisms have been proposed to account for the beneficial influence it exerts on beef quality: cold-shortening prevention, enzyme release/activation, and fiber rupture.

Specifically for hot-boned beef, there can be no doubt that, by greatly accelerating rigor onset, electrical stimulation ensures that the cold-shortening ability of the tissue is exhausted before the internal temperature enters the contraction-provoking range. Of the two conditions, a prerigor state and a low temperature, that must exist simultaneously for shortening to occur, the treatment thus very effectively removes one, leaving the other totally ineffective.

The second mechanism proposed to explain the action of stimulation is enzyme release and/or activation by the combined action of high early-postmortem temperature and low pH. This concept remains unproven, and must be viewed, I believe, with some degree of skepticism until it has been verified *in situ* but without the accompanying (and possibly over-riding) effects of the other two proposed mechanisms.

The third means by which stimulation may do its job is fiber rupture, a fracturing of the muscle structure produced by super contraction or contraction-node formation in adjacent areas. This proposal has met with some opposition on the ground that very little damage can be found microscopically in stimulated meat. Nevertheless, it remains a highly viable concept for several reasons: it parallels the changes observed in highly cold-shortened muscle; alternating zones of contracture and stretch or fracture do indeed occur in previously stimulated muscle; and very little observable damage would be needed to produce a significant tenderizing effect. It is highly relevant that, if only one sarcomere in every 250 was broken by the treatment, each fiber would be severed at mean intervals of only 0.5 mm.; thus almost undetectable damage could cause very considerable tenderizing.

Yet despite its effectiveness and its suitability for incorporation into a hot-boning operation, electrical stimulation remains an imperfect solution. Its tenderizing action appears to be far less in some major muscles than in the almost invariably studied longissimus. In beef chilled as sides, its beneficial effect on color is by no means consistently accompanied by a corresponding enhancement of eating quality; furthermore, the emphasis placed on visual improvement as a selling point for stimulators is perpetuating an undesirable and ineffective quality grading system. These somewhat negative points are raised, not to diminish the significance of electrical stimulation, but rather to suggest that the greatest need now is research rather than development. Without a better understanding of the stimulation process and its basic mechanism, we run the risk of locking ourselves and the meat industry into a system that, a few years from now, may prove to be far from the best.

Conclusion

Let me stress again, in closing, the very mobile nature of early-postmortem muscle. Prerigor events in the tissue are fast in rate, large in magnitude, tightly coupled to each other, and quite capable of exerting major effects on the ultimate product, as the PSE problem has so clearly demonstrated. If we recognize the dynamic state of muscle in the first few post-slaughter hours, and keep the basics clearly in mind as we approach applied situations, I believe that the *one* science of meat and muscle will advance at a rate that, to the present, has been utterly unattainable.

References

Bate-Smith, E. C., and J. R. Bendall. 1974. Rigor mortis and adenosine triphosphate. J. Physiol. 106:177.

Bate-Smith, E. C., and J. R. Bendall. 1949. Factors determining the time course of rigor mortis. J. Physiol. 110:47.

Bendall, J. R. 1951. The shortening of rabbit muscles during rigor mortis. J. Physiol. 114:71.

Bendall, J. R. 1973. Postmortem changes in muscle. *In:* The Structure and Function of Muscle, 2nd edition (Academic Press; ed. G. H. Bourne) 2:244.

Bendall, J. R. 1978. Variability in rates of pH fall and of lactate production in the muscles on cooling beef carcasses. Meat Sci. 2:91.

Buege, D. R., and B. B. Marsh. 1975. Mitochondrial calcium and

postmortem muscle shortening. Biochem. Biophys. Res. Comm. 65:478.
Chrystall, B. B. and C. E. Devine. 1980. Electrical stimulation developments in New Zealand. Proc. 26th Europ. Mtg. Meat Res. Workers 2:104.
Cia, G., and B. B. Marsh. 1976. Properties of beef cooked before rigor onset. J. Fd. Sci. 41:1259.
Davey, C. L. and K. V. Gilbert. 1976. The temperature coefficient of beef aging. J. Sci. Fd. Agric. 27:244.
Dutson, T. R., G. C. Smith, J. W. Savell and Z. L. Carpenter. 1980. Possible mechanisms by which electrical stimulation improves meat tenderness. Proc. 26th Europ. Mtg. Meat Res. Workers 2:84.
Hanson, J., and H. E. Huxley. 1955. The structural basis of contraction in striated muscle. Symp. Soc. Exp. Biol. 9:228.
Lawrie, R. A. 1953. The onset of rigor mortis in various muscles of the draught horse. J. Physiol. 121:275.
Lawrie, R. A. 1960. Postmortem glycolysis in normal and exudative longissimus dorsi muscles of the pig. J. Comp. Path. Therap. 70:273.
Lochner, J. V., R. G. Kauffman and B. B. Marsh. 1980. Early-postmortem cooling rate and beef tenderness. Meat Sci. 4:227.
Locker, R. H., and C. J. Hagyard. 1963. A cold shortening effect in beef muscles. J. Sci. Fd. Agric. 14:787.
Marsh, B. B. 1952. Observations on rigor mortis in whale muscle. Biochem. Biophys. Acta 9:127.
Marsh, B. B. 1954. Rigor mortis in beef. J. Sci. Fd. Agric. 5:70.
Marsh, B. B. 1977. Temperature and postmortem change: energy use and meat quality. Proc. Meat Ind. Res. Conf. p. 13.
Marsh, B. B., and J. V. Lochner. 1981. New approaches to beef quality. Proc. Nat. Beef Grading Conf. (Iowa State Univ. Pub. No. CE-1633), p. 98.
Marsh, B. B., J. V. Lochner, G. Takahashi and D. D. Kragness. 1981. Effects of early-postmortem pH and temperature on beef tenderness. Accepted for publication, Meat Sci.
Marsh, B. B., and J. F. Thompson. 1958. Rigor mortis and thaw rigor in lamb. J. Sci. Fd. Agric. 9:417.
Marsh, B. B., P. R. Woodhams and N. G. Leet. 1968. Studies in meat tenderness 5. The effects on tenderness of carcass cooling and freezing before the completion of rigor mortis. J. Fd. Sci. 33:12.
Sharp, J. G., and B. B. Marsh. 1953. Whale meat: production and preservation. Special Report No. 58, Food Investigation Board (H.M.S.O., London).
Tarrant, P. V., and C. Mothersill. 1977. Glycolysis and associated changes in beef carcasses. J. Sci. Fd. Agric. 28:739.
Taylor, A. S. 1905. Principles and practice of medical jurisprudence, 5th edition, p. 267 (J. and A. Churchill, London).