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ELECTRICAL STIMULATION FOR IMPROVING MEAT QUALITY

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INTRODUCTION

Before reviewing the present status of electrical stimulation as a means of improving meat quality, it is desirable to briefly consider the beckground.

In 1749 Benjamin Franklin had observed that electrical stimulation at time of slaughter tenderized the flesh of turkeys; and it might well have been applied to the musculature of meat animals by the French 200 years ago had not Franklin himself discouraged the idea [37]. A patent was filled in U.S.A. by Harsham and Deatherage in 1951 [28] for the tenderisation of meat by electrical stimulation of carcases *post-mortem*. They noted, incidentally, that electrical stimulation caused a considerable increase in the rate of *post-mortem* glycolysis. But the significance of these observations for commercial procedure has only become apparent during the past eight years, following the work of Carse [14] in New Zealand.

Complaints about the toughness of New Zealand lamb became quite prevalent about twenty years ago. This was puzzling because the commodity had a well-established reputation for uniformly good quality; and husbandry practices had in no way deteriorated. The reason proved to be the increasing efficiency of abattoir refrigeration systems in that country and the understandable desire to minimize throughput time by placing hot carcases into chillers and freezers without a prior period of holding at ambient temperature. It was shown [33, 34, 36] that these circumstances could accelerate *post-mortem* glycolysis, causing shortening of muscles and toughness in the meat subsequently. Although shortening of muscles had long been associated with the onset of *rigor mortis* at *in vivo* temperatures [2] the "cold-shortening" phenomenon was unexpected.

In essence, if the temperature of a muscle, which is free to shorten, is brought below about 10°C whilst it is still in the early prerigor condition — which would normally signify a pH above about 6.0 — "cold-shortening" and toughening will result [36]. The phenomenon is now known to be due to the stimulation of the contractile ATP-ase of actomyosin by calcium ions released from the sarcotubular system and/or mitochondria under conditions which preclude their effective recapture [3, 10, 38].

Since *pre-rigor* muscle no longer shortens on exposure to temperatures below ca. 10°C, once the *post-mortem* pH has fallen below ca. 6 — 6.4 [14] and since electrical stimulation of muscles was known to accelerate *post-mortem* glycolysis [28], the primary biochemical purpose of renewed interest in the procedure has been to swiftly lower the pH below 6 — so that speedy refrigeration can be applied soon after death without risk of toughening the meat from "cold-shortening".

BIOCHEMICAL FEATURES

During electrical stimulation, pH falls of the order of 0.7 unit can occur in two minutes, representing a 100-150-fold increase in the rate [17]. Whilst in lamb and rabbit [4] and pig [29] the post-stimulation rate of pH fall appears to be about twice as fast as normal (for a given temperature range), the position with bovine muscles is less clear. Thus, when the current is applied along the fibres, the rate of pH fall in the post-stimulation phase is enhanced [15]; whereas, when stimulation is across the fibres, the rate of pH fall does not increase until the current is about 60-fold greater [5], when partial breakdown of muscle cell membranes may well occur. The vast acceleration of *post-mortem* glycolysis produced by electrical stimulation whilst the current is flowing signifies a concomitantly high rate of ATP breakdown [8]; which, in turn, reflects marked activation of the contractile actomyosin ATP-ase by released Ca^{++}ions. Yet such are precisely the circumstances which cause the toughening in "cold-shortening", the avoidance of which is the principal reason for applying electrical stimulation in the first place. It is possible that the membrane of the sarcoplasmic reticulum becomes more able to retain Ca^{++}ions [49]; but this feature seems unable to explain the anomaly fully.

Moreover, the attainment of a relatively low pH, whilst temperatures are at *in vivo* levels, denatures muscle proteins, thus causing loss of water-binding capacity. This can be exemplified by *in vitro* experiments [46], by the musculature of pigs which produce pale, soft exudative pok [9, 39] and by the slow cooling deep musculature of beef hindquarters [25]. Yet electrically-stimulated muscles do not overtly lose drip

fluid (at least not initially). There is some eventual loss of fluid however. This has been demonstrated [48]. Thus, whereas the expected benefits of diminished drip were found in hot-deboned, vacuum-packed primal cuts (whether these had been removed from electrically-stimulated sides or not), in comparison with cold-deboned primal cuts after 5 days storage (Table 1), the electrically-stimulated, hot-boned joints had lost relatively more fluid after 21 days storage — although the quantity was still less than that of corresponding joints from cold-deboned sides.

Table 1. Effect of treatment on accumulation of drip (percent of joint weight) in vacuum packed primal joints stored for 5 or 21 days at 1°C (after Taylor, Shaw & MacDougall, [48])

	Treatments					
	stored 5 days			stored 21 days		
	C*(16)	H**(8)	E***(8)	C*(16)	H**(8)	E***(18)
Forequarter joints	0.06	0.16	0.03	0.32	0.20	0.20
Hindquarter joints	0.16	0.05	0.05	0.42	0.12	0.32
All joints	0.11	0.11	0.07	0.37	0.16	0.26

Figures in brackets represent no. of replicates

*C Beef sides held 15°C for 7 h, 0°C for 41 h. 15 primal joints deboned: vacuum packed: then stored at +1°C

H Beef sides hot-deboned at 1-2 h pm, vacuum packed at 3 h pm, placed at 10°C for 9 h and at 1°C for 18 h: then stored at +1°C

E Beef sides electrically stimulated at 50 min. pm, (4 x 30 sec periods, 700 v. at 25 pulse/sec): hot deboned at 12 h pm, vacuum packed, placed – 1°C for 24 h: then stored at +1°C

These seeming biochemical paradoxes have not been explained fully; but recent findings resolve them partially.

That electrical stimulation is not associated with permanently shortened sarcomeres (and toughening) may reflect the fact that the current is short-lived and that, when it is discontinued, the ATP level is still relatively high and the temperature has fallen little from its *in vivo* value. In these circumstances the sarcotubular system can recapture Ca^{++} ions readily, thus supressing ATP-ase activity whilst the ATP level is sufficient to effect muscular relaxation and the restoration of resting sarcomere length. (The data in Table 2 support this view). In "cold-shortening", on the other hand, the low temperature prevents effective operation of the ATP-fuelled Ca^{++} pump of the sarcotubular system; and stimulation of ATP breakdown is thus not arrested.

There has been apparent disagreement between research workers on the reason for the association of *post-mortem* electrical stimulation with tenderness. Some [12] attribute this benefit to the avoidance of "cold-shortening"; and they have published data showing the retention of rest length in the sarcomeres of muscles which were electrically stimulated, whereas control sarcomeres shortened, when both were exposed

Table 2. Mean sarcomere lengths (μm) in control and electrically-stimulated bovine *l. dorsi* muscles

Control	Electrically-stimulated *post-mortem*	Subsequent treatment		References
		induced "cold-shortening"	high temperature conditioning	
1.07	1.59	+	—	[12]
1.58	1.60	—	+	[26]
1.86	1.86	—	+	[45]

to environments causing "cold-shortening" (Table 2). Others have attributed the benefit to early and extensive conditioning changes arising from the combination of low pH with *in vivo* temperatures [45, 26]; and their histological data show no significant differences between the sarcomere lengths of electrically-stimulated and control muscles (Table 2). Those who support the former view, however, have deliberately exposed their samples to "cold-shortening" circumstances and thus have precluded high temperature conditioning [12]; whereas those who implicate high temperature conditioning have carefully avoided "cold-shortening" [26]. It is evident that both phenomena can be involved in accounting for the tenderizing associated with *post-mortem* electrical stimulation : the temperature which the meat subsequently attains will determine which predominates.

Although the breakdown of various components of muscle has been detected during conditioning, the changes in no one of these has been unequivocally shown as crucial for tenderness increments. Penny and Ferguson-Pryce [41] demonstrated that troponin T was proteolysed during conditioning by calcium-activated sarcoplasmic factor (CASF : calcium-activated neutral protease) above pH6, and by cathepsin B below it. CASF also attacks proteins in the vicinity of the Z-lines (probably desmin), M-line proteins, and tropomyosin [40] and the so-called "gap-filaments" [35]. At low pH, and related high temperature, during conditioning, lysosomal enzymes degrade the micopolysaccharides of the ground substance [23] telopeptide cross-links in collagen [24] and, below pH5 or above 35°C, over some days, myosin and actin [41].

Conditioning changes — as assessed by the decrease in shear force — have a high temperature coefficient [20]. By comparing the temperature/ /pH history of control and electrically-stimulated muscle, George et al. [26] calculated that such conditioning changes proceed at about twice the rate in the latter during the first 24-30 hours *post-mortem* — in circumstances when "cold-shortening" was avoided by not exposing the muscles to environmental temperatures below 16°C until 8 hours *post-mortem*. After 21 days storage at 0°C, the tenderness of control muscles lemgths of electrically-stimulated and control muscles (Table 2). Those

had increased substantially through the operation of conditioning processes at the normal rate; but it was still slightly less than that of the electrically stimulated muscles at this time [26].

In their paper, George et al. [26] presented histological evidence which showed clearly that, in electrically stimulated bovine muscles, in which near *in vivo* temperature and low pH had prevailed, bands of denatured protein formed gradually within the fibres. These were similar to those observed in the musculature of pale, soft, exudative pork [9]. In so far as the proteins of electrically stimulated muscles conform to expectation and denature, therefore, one apparent anomaly of the procedure has been resolved. There remains to be explained, however, the absence of marked exudation in electrically-stimulated bovine muscle. As remarked above, such loss of water-holding capacity is striking in PSE pork, in the deep musculature of beef hindquarters and when *in vivo* temperature and low pH are combined *in vitro*. It is feasible that the acceleration of conditioning changes, which is one effect of the fast pH fall achieved by such stimulation enhances intracellular osmotic pressure sufficiently to accomodate the loss of water-holding capacity by the muscle proteins. Certainly normal slow conditioning of beef at 0°C has this effect [13, 18]. On the other hand pork undergoes conditioning changes to a greater extent than beef [30] and this might have been expected to raise intracellular osmotic pressure even more than with the former type of meat. Possibly the proteins of pork ($\approx$ "white" muscle) are intrinsically more labile than those of beef. There is some evidence that the proteins of so-called "white" muscle generally are more readily denatured *post-mortem* than those of the so-called "red" type [32]. Again, the sarcolemma of porcine muscle is more permeable to water than that of beef [26].

STIMULATION PROCEDURES

The scientific literature on electrical stimulation indicates that the eulectrode system, the type of current (voltage, frequency of pulses and duration), the pathway (via nerve or direct) and the time *post-mortem* have varied considerably between investigators. Bendall [7] has comprehensively reviewed the position.

A selection from the many variants has been given in Table 3. Most investigators appear to have applied the current via the thoracic region and to have used the Achilles tendon region for the return to earth. Substantially different voltages have been used. Although low voltages ($\leqslant 100$) are intrinsically safer in operation [7, 12], than voltages of 500-1000 or more, most investigators have found them less consistent in effect. Table 4 illustrates this for bovine *l. dorsi* and *semimembranosus* muscles. Moreover, high voltages are effective when applied for 1.5-2 mi-

Table 3. Modes of application of electrical stimulation to lamb and beef carcases

Species	Unit of operation	Arrangement of electrodes		Current voltage PPS	References
		live	earth		
Lamb	Carcase: fleece intact	Multiple: horizontal: contact w. dorsal region	Hook, Archilles tendon to hanging rail	3000 12.5	[17]
	Carcase: fleece removed	,, ,,	,, ,,	1600 12.5	
Beef	Dressed side	Steel pin at 3rd/4th cervical vertebrae	,, ,,	2000 15	[21]
Beef	Intact or dressed: carcases or sides	Metal clip attached severed neck	Clip, Archilles tendon to hanging rail	700 25 (Peak)	[8]
Beef	Dressed side	Steel pin: inserted in muscles between scapula/thoracic vert.	Pin: in muscles of round nr. Archilles tendon	440 50 800 (Peak)	[43]
Beef	Intact and dressed side	Rectal probe. Multipoint-pins in hindlimb (biceps/ /ST) and neck (brachiocephal.)	Hook, Archilles tendon to rail	110 40 (max)	[11]
Beef	Dressed sides	Steel pin at 3rd/4th cervical vert.	,, ,,	1000 14 (Peak)	[11]
	,, ,,	4 (multipoint) in hindlimb muscles	,, ,,	45 14 (Peak)	

Table 4. pH changes in bovine *l. dorsi* and *semimembranosus* muscles, stimulated immediately after bleeding, at 25 pps and various voltages over 2 minutes (after [8])

Peak voltage	pH	
	l. dorsi	*semimembranosus*
0	7.04	7.04
20	6.66	6.90
100	6.38	6.64
250	6.34	6.38
600-700	6.28	6.28

nutes; whereas longer times (~ 4 minutes) are normally required with voltages of the order of 100.

Because of differences in their intrinsic electrical resistance, intact carcases will allow more current flow for the application of a given voltage. Thus, for example, a peak voltage of 680, between electrodes 200 cm, apart gave a peak current of 5.2 amps with intact beef carcases (wherein conductivity is high because of the relatively large cross-sectional area and the presence of the wet gut contents), 3.3 amps with dressed carcases and only 2.4 amps with dressed sides [7]. The latter tend to jerk outwards since there are no *contralateral intracostal* or *l. dorsi* muscles to oppose movement [6].

In respect of other aspects of the current, optimum pulse rate appears to be between 15-25 pps (higher frequencies tend to be relatively ineffective since they fall within the latency period of the muscles concerned); and the optimum pulse width is about 20-40 milliseconds (shorter widths may fail to activate all the muscle fibres).

The response of beef carcases to electrical stimulation falls off quickly after about 50 minutes *post-mortem* [8] and that of lamb carcases even sooner. It is thus desirable to apply the current within about 30 minutes of slaughter. Since isolated muscles, on the other hand, will respond as well at 3 hours *post-mortem* as at 20 minutes [5] it appears respond as well at 3 hours *post-mortem* as at 20 minutes [5] it appears that in electrical stimulation of the carcase or side the musculature is usually high, direct stimulation of the muscles can occur at a later time [16] when decay of the nervous pathways has made low voltages ineffective.

The importance of a still-functioning nervous system in making low voltages effective has been re-emphasized by recent Swedish work [42]. By placing one electrode in the nerve centre of the muzzle (and earthing via the Achilles tendon) during the first 10 minutes *post-mortem*, a typical acceleration of *post-mortem* glycolysis was achieved by applying various stimulation procedures lasting for 1 minute and having a peak voltage of 80. A current of 14 pps, applied either in 1 sec pulses or continuously, was effective. Although the peak voltage was 80, the duration of each

peak was only 5 msec and the actual voltage thus very low; being thus relatively safe in operation. Another low voltage procedure is to push a plastic pithing rod (with an electrode at its tip) down the spinal cord immediately after bleeding. The current used with this device is pulsed at 25 pps for 2 minutes, the peak voltage being 140 [7]. Such procedures also have obvious economic advantages; but not all abattoirs would be in the position to stimulate carcases so early *post-mortem*.

PRACTICAL IMPLICATION

It is evident that, whether applied to intact or dressed carcases, or sides, electrical stimulation immediately *post-mortem* permits fast cooling of meat without danger of "cold-shortening" and toughening. Such early stimulation might not be feasible in all abattoirs; and the procedure can be delayed until about 30 minutes *post-mortem* [27] without much loss of efficacy.

Moreover, in most cases carcases can be frozen swiftly, after electrical stimulation, without *pre-rigor* freezing and resultant "thaw-*rigor*". There is a phase in *post-mortem* glycolysis, however, when the muscles, although no longer susceptible to "cold-shortening", have still sufficient ATP to permit *pre-rigor* freezing and thus the marked toughening of "thaw-*rigor*" subsequently. Pelvic hanging appears necessary for maximum tenderness in electrically stimulated beef carcases if these are to be placed into blast freezers within 30 minutes of stimulation [47]. When such rapid freezing is intended this should be delayed until 6 hours after stimulation [7].

Apart from the avoidance of toughening, electrical stimulation has been associated with an enhanced brightness of the red colour on meat surfaces [44], possibly because the process depletes the metabolites of surviving oxidative pathways in the muscle and damages their enzymic organization, or because the fast fall in pH causes increased light scattering. Particularly in USA, the early carcase grading which electrical stimulation permits has been an additional advantage of the procedure.

In endeavouring to assess the practical implications of the process it is important to appreciate that, concomitantly with the desire to enhance the efficiency of abattoir operations by speeding throughout, there has been an increased tendency for abattoirs to undertake the centralized preparation of prepackaged cuts, both of commercial joints and of portions for the individual consumer. This has reflected changing patterns of consumption-smaller families, canteen meals, convenience foods and domestic freezer-cabinet storage. Moreover, deboning of the still hot carcase and vacuum-packaging of the warm cuts is a further extension

of these tendencies [19], which have been shown to diminish evaporative and exudative losses.

With such relatively small portions of meat, electrical stimulation of the carcase or side could prove especially useful in avoiding "cold-shortening", since the latter would otherwise be readily induced under the very rapid rates of cooling which would occur with them. Moreover the rapid lowering of temperature would markedly lessen microbial growth — a factor of importance with portions of meat having a large surface to volume ratio. Unfortunately, however, although the toughening of "cold-shortening" would be easily avoided, these very circumstances would prevent the positive contribution of tenderness of electrical stimulation through its creating favourable circumstances for early and rapid conditioning : i.e. a combination of low pH with *in vivo* temperatures, as alluded to above. Thus, storage of hot-deboned muscles from electrically-stimulated beef sides demonstrated that whilst the benefits of accelerated conditioning were noteworthy at holding temperatures of 25°-35°C, when toughness was reduced to half its initial value in 0.8 day, this degree of tenderness took 22 days at 0°C [1]. Moreover, it was found that microbial numbers doubled for each 5°C rise in storage temperature.

In this general context, electrical stimulation has also been applied to muscles after their removal from the hot side, using various combinations of voltage (25-300) and frequencies (8-25 pps). Since hot deboning in certain tropical countries may involve exposure to ambient temperatures of 30°C-40°C, it has been of interest to assess the effects of electrical stimulation in such circumstances, where early excision of muscles can cause contraction and toughening during the onset of *rigor mortis*. Electrical stimulation (100v, 25 pps, 4 mins.) of hot-deboned bovine *l. dorsi* muscles was found to achieve a marked tenderizing effect over control muscles either held on the carcase or excised when the meat was subsequently exposed to temperatures of 30° or 40°C for 5 hours. (S. A. Babiker and R. A. Lawrie, unpublished data). At the latter temperature, however, and in contrast with the findings at 30°C, there was appreciably greater loss of water-holding capacity and, subsequently, greater development of microbial numbers. Moreover, the tenderness of the electrically stimulated muscles was less marked.

CONCLUSION

It may be that the solution to this dilemma lies in subjecting the various joints and portions to different cooling regimes. Muscles differ biochemically. Neither in their response to "cold-shortening" nor in their conditioning behaviour are they identical [3, 7, 26]. This approach may well

be expedited by the electrical stimulation of individual muscles or groups in a manner appropriate for each following their partial or complete removal from the hot carcase.

In conclusion, it should be recollected that the musculature from electrically stimulated carcases, notwithstanding its advantages in terms of tenderness and colour might not be suitable for all purposes. Thus, it would be less useful than nonstimulated muscle for the production of those cured and freeze-dried products which depend upon the near *in vivo* levels of ATP at time of processing for their high water-holding capacity [31].

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Streszczenie

Elektryczna stymulacja mięśni w tuszach bezpośrednio *post-mortem* wydatnie przyspiesza przemianę glikogenu w kwas mlekowy. Wskutek obniżenia pH mięśni do 6,0-6,4 w ciągu kilku minut proces stymulacji usuwa wrażliwość mięśni na skurcz chłodniczy i stwardnienie, przez co pozwala na szybkie zamrożenie mięsa (z zastosowaniem lub bez odkostnienia bez wychłodzenia), polepszając tym samym ekonomiczną efektywność procesu.

Wpływając na obniżenie pH mięsa w temperaturze przyżyciowej zwierzęcia elektryczna stymulacja przyspiesza również znacznie przemiany dojrzewania (w tym również zwiększania kruchości), które normalnie zachodzą powoli w mięsie po uboju pod wpływem enzymów uwalnianych w tych warunkach z lizosomów. Ten drugi efekt zwiększania kruchości mięsa zatraca się jednakże, jeśli zamrożenie jest zbyt gwałtowne.

W większości stosowanych systemów elektrycznej stymulacji prąd jest podłączany do tuszy lub jej części w okolicy piersiowej zaś uziemiany przez ścięgno Achillesa. Jakkolwiek napięcia mniejsze lub równe 100 V są bezpieczne, to ich wpływ wydaje się mniej zdecydowany niż wpływ prądu o napięciu 500-1000 V. Optymalna częstotliwość wynosi 15-25 cykli/sekundę, zaś optymalna amplituda pulsacji 20-40 milisekund.

Ponieważ efekt tego działania na tuszę ustaje po 50 min po uboju, prąd najlepiej jest stosować ok. 30 min po uboju z powodu pogorszenia się z czasem drożności połączeń nerwowych. Izolowane mięśnie mogą być stymulowane bezpośrednio, nawet po wielu godzinach *post-mortem*, wymaga to jednakże zastosowania znacznie wyższych napięć.

Przebieg elektrycznej stymulacji w celu uzyskania optymalnej kruchości może być dokładnie określony na podstawie biochemicznej charakterystyki indywidualnych mięśni.