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## Alcohol and public health

Winslow's long definition of public health not only includes comment about sanitation, personal hygiene and the organisation of medical and nursing services for the early diagnosis and preventative treatment of disease, but also the development of social machinery to ensure to every individual a standard of living adequate for maintenance of health, so organising these benefits as to enable every citizen to realise his birthright of health and longevity.<sup>1</sup>

In today's common parlance we attempt risk assessments of any human activity. Excessive consumption of alcohol is a risky business and, despite public education campaigns, advice continues to be ignored, particularly among the young with the inevitable consequence that incidence and mortality rates of alcohol-induced liver disease (ALD) continue to rise. The most dramatic rise in the figures has been most apparent in Scotland. The recent review by Breakwell *et al* not only identified the rising incidence of ALD throughout the UK, but showed that mortality rates in Glasgow due to alcohol-related causes had virtually doubled between 1998 and 2004 and demonstrated that 15 of the top 20 areas with the highest incidence of alcohol-related deaths were in Scotland.<sup>2</sup> Glasgow is at the top of the list with 83.7 deaths/100,000 population; Aberdeen is 20th with 30.7. A recent conference, Scots on the Rocks, held in Edinburgh attempted to identify some of the key risk factors, not least those encouraging the young to over imbibe. Attention was drawn to the recent study in north west England conducted by Bellis *et al* which showed that binge drinking was strongly related to expendable income and to individuals buying their own alcohol.<sup>3</sup> On the other hand, being bought alcohol by parents reduces the risk of bingeing. Parental supervision certainly seems to be lacking in several cases: this may similarly have impact in other areas.

In an attempt to develop a rational scale to assess the harm of potential drug misuse Nutt *et al* categorised three main factors: the physical harm to the individual user by the drug; the tendency of the drug to induce dependence; and the effect of drug use on families,



communities and society.<sup>4</sup> Overall, alcohol was fifth most prominent on the scale behind heroin and cocaine but ahead of tobacco, ecstasy and cannabis, to name but a few. In the category of social harm alcohol was second only to heroin.

The scientific evidence continues to be gathered and adds more to the concerns about alcohol. Much remains to be done to put in place necessary pathways to limit its damage.

Dr GE Ratcliffe, Editor

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The theme of this issue is alcohol and the muscle. I am much indebted to the respective authors for their most informative articles.

## Alcoholic cardiomyopathy

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### Epidemiology

Heart failure is a common chronic disorder with high-associated morbidity, mortality and cost, with left ventricular systolic dysfunction (LVSD) underlying most treatable cases. Approximately two-thirds of cases of LVSD result from ischaemic heart disease (IHD) the rest are referred to as non-ischaemic cardiomyopathies. Between 20–35% of all cases of non-ischaemic cardiomyopathy result from excessive ethanol intake, termed alcoholic cardiomyopathy (ACM). A recent epidemiological survey of subjects  $\geq 45$  years old found that 5.5% of the community have evidence of LVSD, with ACM underlying 13% of all cases and 30% of all non-ischaemic cases.<sup>1</sup>

Acute and chronic alcohol ingestion both depress myocardial function. Although in the long term very heavy alcohol intake is linked with ACM, the exact duration and level of alcohol consumption required to produce ACM has not been clearly established. Urbano-Márquez *et al* evaluated 48 asymptomatic chronic alcoholics, all drinking  $>70$  units of alcohol per week (mean intake 170 units/week).<sup>2</sup> They found a significantly lower mean left ventricular ejection fraction (LVEF) and greater mean end diastolic diameter and left ventricular mass index in very heavy drinkers compared with 20 healthy controls, all drinking less than 14 units per week. Furthermore, LVEF correlated negatively with lifetime alcohol intake. Importantly, none of the

alcoholic subjects had clinical or laboratory evidence of malnutrition, suggesting that these changes resulted from the toxic effects of chronic alcohol intake.

Similarly, Lazarević *et al* found evidence of left ventricular dysfunction in 89 asymptomatic alcoholics who had higher mean left ventricular mass index and left ventricular end systolic and end diastolic volume index compared with 30 control subjects.<sup>3</sup> A causal association between alcohol intake and LVSD is also suggested by McKenna *et al* who found that significantly more subjects with dilated cardiomyopathy drank more than the recommended weekly amount of alcohol (40% *v* 24%,  $p < 0.01$ ); were more likely to be alcohol abusers (27% *v* 16%,  $p < 0.05$ ), and had higher average total lifetime alcohol consumption (31,200 *v* 7,904 units,  $p < 0.01$ ) than controls.<sup>4</sup>

### Pathophysiology

On a cellular level, the most likely mechanism underlying alcohol-induced myocardial damage is an alteration in myocardial redox status, with ethanol oxidation increasing the NADH/NAD redox potential within the cytosol and mitochondria and producing hydroxyethyl free radicals and reactive oxygen species. These changes increase lactate production and ketoacidosis and impair citric acid cycle activity, interfering with the transport and binding of calcium, mitochondrial respiration, myocardial lipid synthesis, myocardial protein synthesis and myofibrillar ATPase activity. Further changes include: apoptosis; inhibition of actin and myosin association; reducing muscle contractility; and stimulation of the sympathetic nervous system and renin-angiotensin-aldosterone system. These all potentially culminate in ventricular dysfunction. At a gross anatomical level, patients with ACM tend to have more myocardial hypertrophy and interstitial fibrosis than patients with idiopathic dilated cardiomyopathy (IDCM).

### Clinical characteristics

The age of onset and clinical characteristics of patients with ACM are similar to patients with IDCM, with half or more subjects with significant ventricular dysfunction being asymptomatic or minimally symptomatic at diagnosis, although at high risk of developing future symptoms. Symptoms include: breathlessness, fatigue, peripheral oedema, orthopnoea, paroxysmal nocturnal dyspnoea and palpitations. Signs include tachycardia or other arrhythmia, raised jugular venous pressure, gallop rhythm, displaced apex beat, mitral regurgitation and tricuspid regurgitation murmurs due to cardiac enlargement, hypotension, basal respiratory crepitations and peripheral oedema.

### Investigations

Investigations performed should include blood tests for renal and liver function and a full blood count to rule out anaemia. An assessment for diabetes mellitus should be made. Thyroid function tests should be performed as should a chest radiograph and electrocardiogram. Raised serum or plasma B-type natriuretic peptide levels (brain natriuretic peptide or N-terminal pro-brain natriuretic peptide) may help suggest a diagnosis of ventricular dysfunction in asymptomatic subjects or if the diagnosis is unclear, with echocardiography usually required to confirm the diagnosis and help assess aetiology.<sup>5</sup>

Further imaging to rule out underlying ischaemic heart disease should be performed, especially in the presence of risk factors, whether by myocardial perfusion imaging or direct coronary angiography, with exercise testing alone often unhelpful.

### Management

The mainstay of therapy for ACM is to treat the underlying cause, ie complete and continued abstinence from all alcohol consumption. Fauchier *et al* evaluated 50 patients with ACM and 84 patients with IDCM all on routine heart failure medication and all with normal coronary angiograms.<sup>6</sup> At four-year mean follow-up, patients with ACM who failed to abstain from alcohol had significantly worse

prognosis (cardiac death or heart transplantation) than patients with ACM and abstinence, with the only independent multivariate predictor of cardiac death in subjects with ACM being lack of abstinence at follow-up. The prognosis for IDCM and ACM with abstinence was no different. In a similar study of 338 men with non-ischaemic cardiomyopathy maintained on ACE-inhibitor therapy, 79 defined as alcohol abusers, at five-year mean follow-up, those who continued to abuse alcohol had a significantly worse prognosis than those with ACM who had stopped alcohol abuse, or subjects with IDCM.<sup>7</sup> Furthermore, those with ACM who had stopped abusing showed a significant increase in LVEF, suggesting reversibility of disease.

Medical therapy is identical to conventional therapy for other forms of heart failure, namely diuretics to improve symptoms and ACE inhibitors and/or angiotensin-II receptor antagonists, beta blockers such as carvedilol, metoprolol or bisoprolol, and aldosterone antagonists to improve symptoms and prognosis. Electrolyte abnormalities should be managed carefully to reduce the risk of arrhythmia. Thiamine (200 mg once daily) and multivitamins including vitamin B and C, and folic acid should be given to help treat any concomitant nutritional deficiencies. Cardiac resynchronisation therapy may be indicated in patients with significant heart failure symptoms (New York Heart Association class 3 or 4); evidence of cardiac dyssynchrony and significant left ventricular systolic dysfunction (LVEF <35%). Ultimately, in end-stage heart failure, cardiac transplantation may be required.

## Conclusions

Heart failure is common, with alcoholic cardiomyopathy underlying a significant proportion of cases, especially in patients free from ischaemic heart disease. Its presentation and management is similar to heart failure caused by other aetiologies. As well as standard heart failure medications, complete and continued abstinence from alcohol improves ventricular performance and prognosis and should be strongly encouraged.

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## Chronic alcoholic myopathy

### A common cause of muscle weakness and wasting in alcohol misusers

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## Ethanol misuse in the UK

Ethanol is one of the most commonly used recreational drugs in the UK. Nearly 40 million adults drink and the alcoholic drinks industry is worth more than £30 billion a year.<sup>1</sup> Most

people enjoy alcohol sensibly, though around 30% of men and 20% of women drink more than the recommended amounts.<sup>1</sup> Alcohol misuse, however, has been estimated to cost around a staggering £20 billion per year in the UK.<sup>1</sup> The cost of healthcare for alcohol-induced disease is estimated at £1.7 billion per year. In the UK, up to 70% of all attendances at accident and emergency departments and at least 20,000 deaths per annum result from alcohol misuse.<sup>1</sup>

The hepatic, cerebral and cardiovascular effects of alcohol are most widely publicised and researched. Yet, in alcohol misusers, the prevalence of chronic alcoholic myopathy is greater than that of cirrhosis, peripheral neuropathy and cardiomyopathy combined.

## The disease

For many years, doctors largely ignored the reports of physical weakness and difficulty walking from alcohol misusers. Then in the 1980s Professor Peters' group at Northwick Park Hospital examined skeletal muscle biopsies from over 150 consecutively admitted alcoholics.<sup>2</sup> Sixty per cent of these subjects were shown to have histological evidence of chronic alcoholic myopathy.<sup>2</sup>

Chronic alcoholic myopathy is arguably the most common disease of skeletal muscle in the UK and indeed the Western world, where it is estimated that between 40% and 60% of chronic alcohol misusers are affected.<sup>3</sup> Although chronic alcoholic myopathy is common, most textbooks on skeletal muscle diseases contain little information about it. Yet, the rare incurable inherited myopathies, such as Becker and Duchenne muscle dystrophy, receive considerable attention (Table 1).<sup>3</sup> Acute alcoholic myopathy is rare and only affects less than 5% of chronic alcoholics.

Chronic alcoholic myopathy results in muscle weakness and wasting. Affected patients suffer cramps and myalgia. When mild, it often goes unrecognised, but if severe, chronic alcoholic myopathy can cause significant morbidity with difficulties in gait and an inability to climb stairs or get out of a chair unaided. Essentially, chronic alcoholic

**Table 1.** Prevalence of muscle abnormalities. This table compares the prevalence of both acute and chronic alcohol-induced muscle diseases with the prevalence of a selection of the inherited myopathies. The prevalence of alcoholic myopathy is based on the assumption that the UK has two million ethanol abusers. Data derived from Reference 3.

	Prevalence of myopathies per 100,000 population
Chronic alcoholic myopathy	2,000
Acute alcoholic myopathy	20
Fasioscapulohumeral dystrophy	3
Malignant hyperthermia	2
Becker muscular dystrophy	1
Type II glycogen storage disease	1
Duchenne muscular dystrophy	0.5
Myotonic dystrophy	0.5
Myasthenia gravis	0.4

myopathy is akin to cardiac or cancer cachexia where patients may lose up to 30% of their muscle mass. Reduced muscle mass is a risk factor for impaired responses to immunological and metabolic stresses and worse outcomes when there is a concomitant disease.

It has been suggested that chronic alcoholic myopathy may be the visible features of a disease process affecting all muscle types.<sup>3</sup> Thus, ethanol is also cardiotoxic and can cause cardiomyopathy and impaired contractility. Furthermore, ethanol disrupts the functions of the smooth muscle in the gastrointestinal tract leading to motility disturbances and thus impaired nutrient absorption.<sup>4</sup> The resultant malnutrition compounds the toxic effects of alcohol.

Fortunately, alcoholic myopathy can often be cured with several months of abstinence.<sup>3</sup> Muscle mass usually returns to normal, however, recovery of muscle strength is sometimes incomplete after even five years abstinence, but the reasons for this are not known.

Studies on alcoholic myopathy Various aspects of chronic alcoholic myopathy have been examined. Its aetiology is complex and multifactorial. Some of the pathogenic mechanisms include inhibition of muscle protein synthesis and breakdown, membrane damage,

altered calcium regulation and the generation of free radicals.<sup>3,4</sup> Skeletal muscle protein adducts are also formed: both malondialdehyde (a product of lipid peroxidation) and acetaldehyde combine with skeletal muscle proteins to form aldehyde-protein adducts.<sup>3,4</sup> This has a number of biochemical implications, for example, protein adducts render the parent protein inactive and the protein-adducts are immunogenic.

Investigations of the pathogenesis of alcoholic myopathy have traditionally focused on the effects of ethanol on specific aspects of metabolism. Our understanding of the disease process has been significantly increased by this time and resource intensive methodology. While there is clearly a need for hypothesis-driven research, significant pathways may be highlighted more holistic, or 'omic' techniques. Literally thousands of molecular and cellular factors involved in the development of alcoholic myopathy could be identified by genomics, proteomics and metabolomics. For example, recent studies in genomics, using gene arrays, show that the levels of messenger ribonucleic acid (mRNA) encoding over 400 skeletal muscle proteins are affected by alcohol dosage.

Remarkably, less than 1% of published alcohol-related research mentions chronic alcoholic myopathy. Although the causative agent is obvious, however, the mechanism by which alcohol induces a myopathy has not yet been elucidated. There is clearly a need for more work. We suggest that alcoholic myopathy should have greater public and clinical awareness and greater allocation of resources for research.

## Conclusion

Chronic alcoholic myopathy is common, preventable and reversible. Although the cause is clear, the pathogenesis is not yet well defined. Thus, we suggest that alcoholic myopathy should have greater public and clinical awareness and greater allocation of resources for research.

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## Alcohol and soft tissue compression

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Alcohol, humankind's 'favourite drug',<sup>1</sup> has a wide range of interrelated effects. Often these are conceptualised according to organ system and differentiated into being either chronic or acute. One danger in this approach is that we overlook alcohol's power as an anaesthetic, rendering the intoxicated user insensate. In this state, one is susceptible to the effects of prolonged immobilisation and maintained pressure on the soft tissues. 'Crush syndrome' is an under-emphasised emergency that potentially involves several different specialties – especially orthopaedics, anaesthesia, intensivist, and renal physicians.

Depending on anatomical site, prolonged compression of the soft tissues may result in neuropathy (as in 'Saturday night palsy'), compartment syndrome, rhabdomyolysis or a combination of these conditions.<sup>2</sup>

Compartment syndrome<sup>3</sup> is a clinical condition in which a rise in intracompartmental pressure (relative to the perfusion pressure) is sufficient to prevent perfusion of intracompartmental structures. It must be noted that the presence of a distal pulse in no way negates the existence of a proximal compartment syndrome. It is a surgical emergency mandating

## Case report

A 32-year-old man was brought to the emergency department with tense swollen calves, having lain supine with his legs over his bed footboard for approximately 12 hours. Prior to this he had been on a 12-hour drinking binge. Despite appropriate analgesia, he had great pain exacerbated by passive movements of the toes and cutaneous sensory loss consistent with bilateral tibial and peroneal neuropathy. The clinical diagnosis of compartment syndrome was confirmed with intracompartmental pressure measurements on both calves (pressure difference between diastolic pressure and compartmental pressure was <30 mmHg).

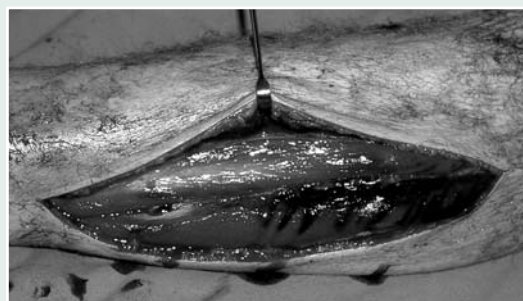
Admission blood tests were indicative of rhabdomyolysis with hyperkalaemic acidosis and renal compromise ( $\text{Na}^+$  142  $\text{mmol/l}^{-1}$ ,  $\text{K}^+$  5.9  $\text{mmol/l}^{-1}$ ,  $\text{H}^+$  56  $\text{nmol/l}^{-1}$ , urea 10.2  $\text{mmol/l}^{-1}$ , creatinine 135  $\mu\text{mol/l}^{-1}$ , and creatinine kinase (CK) 51,500  $\text{IU/l}^{-1}$ ). Per-urethral catheterisation yielded dark urine (Fig 1) that was strongly positive for protein and for which microscopy showed brown casts.

After institution of forced alkaline diuresis, the patient was taken to theatre for emergency bilateral four compartment fasciotomies (Fig 2). The clinical findings were of grossly swollen posterior and deep posterior compartments with a small amount of necrotic muscle that was debrided. The wounds were left open for 72 hours and then closed, one incision directly and the second by split skin grafting (both sides). Subsequently, acute renal failure was treated by several courses of dialysis and normal renal function returned. The patient made good recovery with near normal motor function.



Fig 1. Preoperative photograph of patient with bilateral crush syndrome involving the posterior compartments of both lower limbs. Note the dark urine in the catheter bag indicative of myoglobinuria and rhabdomyolysis.

Fig 2. Lateral fasciotomy wound.



immediate orthopaedic referral. Delay in diagnosis and treatment is the predominant cause of subsequent morbidity.

Rhabdomyolysis<sup>4</sup> is a clinical condition involving muscle necrosis and leakage of intracellular contents to the circulation. The term covers a large range of clinical conditions – from the relatively minor (with a small elevation of creatinine kinase only) to life- and limb-threatening conditions with major metabolic disturbance, myoglobinuria and secondary renal failure.

### History

Sometimes the history is clear with a period of prolonged recumbency. Other times the history is confused or unreliable. With an obtunded patient, the diagnosis is still less clear and a high index of suspicion must be maintained (with immediate orthopaedic referral and compartment pressure monitoring).

### Examination

The clinical findings are of swelling with pain out of proportion to the apparent injury. Active movements are decreased, and the pain is exacerbated by passive stretch. Sensory changes also occur (both compressive and also secondary to compartment syndrome). If there is any doubt, pressure monitoring should be performed immediately including on patients with depressed conscious level (possibly still intoxicated/under the influence of other drugs).

### Investigations

The initial investigations are simple and can be performed expediently:

- 1 urine sample at catheterisation – dipstick and microscopy
- 2 urea and electrolytes – in particular for renal indices, especially urea, creatinine, and potassium
- 3 arterial blood gases
- 4 creatinine kinase (CK) – as an indicator of muscle damage

- 5 compartmental pressure monitoring (with a diagnosis of compartment syndrome if the diastolic pressure minus the compartmental pressure is less than 30 mmHg) which should be of a continuous, as opposed to one-off, nature.<sup>3</sup>

### Differential diagnosis

The differential diagnosis includes haematoma, deep vein thrombosis and necrotising fasciitis. Usually the clinical diagnosis should not be in doubt. A negative ultrasound scan may be of help in eliminating the first two diagnoses. The diagnosis of necrotising fasciitis is rarer, and is in any case managed similarly in terms of fluid resuscitation and surgical exploration with resection of dead/infected muscle.

### Treatment

Successful treatment depends on early diagnosis, referral, fluid resuscitation, electrolyte balance correction (and

renal replacement therapy if renal failure occurs), and surgical treatment by fasciotomy (or more rarely amputation). The single greatest threat to a decent outcome is delay in diagnosis and treatment. Alkalinisation is theoretically attractive, but its role remains unproven. Although the scars are extensive, fasciotomy is a limb-saving procedure with low functional morbidity.

### Late presentation

There is controversy about the surgical management of late compartment syndrome when complete muscle necrosis may have occurred,<sup>3</sup> with raised sepsis rates after exploration. If there is only partial muscle necrosis, however, fasciotomies are indicated to salvage remaining viable muscle, and thorough debridement of necrotic muscle is mandatory. If unsalvageable, amputation must be considered.

### Conclusions

Optimal management for crush syndrome has changed little in the last 20 years. Rapid diagnosis, appropriate referral and treatment are essential, including correction of fluid volume and electrolyte balance (possibly dialysis, if acute renal failure supervenes), and surgery – usually fasciotomies but possibly amputation.

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### Alcoholis publication dates

This bulletin will be published quarterly in March, June, September and December.

*Items for publication should be forwarded to the Editor.*

## News shorts

Three items which may contribute to reduced consumption of alcohol in the UK have recently made the media and medical press.

- 1 All bottles and cans containing alcoholic beverages are to carry details of units and recommended safe drinking levels by the end of 2008.
- 2 Alcohol branding on children's replica sports shirts is to end with new restrictions applying to all sponsorship contracts after 1 January 2008.
- 3 Tessa Richards, assistant editor of the *BMJ*, makes a plea for doctors to do more to tackle alcohol abuse (*BMJ* 2007;334:1142).

■ The MCA strongly supports these policies and proposals. Further comment from readers is encouraged.

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