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[Dr. Monro.]

Time brings about many changes in the principles and practice of medicine—in some instances slowly, and in others very quickly. We have an example of rapid changes in practice in the case of potassium bromide, which was introduced into the *London Pharmacopœia* in 1835, because of its supposed value in splenic enlargements; was removed from it in 1851, as inert and useless; and entered again into favour in 1857 and later years, on account of its value in epilepsy. It is now well known as the sovereign remedy for that disease, and the consumpt of this drug at the National Hospital for the Paralysed and Epileptic in one recent year was reported as amounting to more than a ton and a half.

The way in which drugs, both new and old, fall from positions of esteem is so well known to all that no example need be mentioned. An instance of change in our views as to the action of a medicine is seen in the case of digitalis, which not very many years ago was regarded as a narcotic and cardiac sedative. It was supposed to be of value in weakening the pulse when the heart was beating too strongly, and to be dangerous when the pulse was weak and irregular. I need scarcely remind you that these opinions are almost

[1] Introduction to a discussion at the Glasgow Southern Medical Society on 31st March, 1904. It is to be noted that the question of alcoholic drinks as beverages was entirely outwith the scope of the discussion, and was not considered by any of the speakers.

exactly the reverse of those which justly prevail at the present day.

A revolution just as striking as any of those I have mentioned can be witnessed in the views of medical men as to the therapeutic usefulness of alcohol. In one or more aspects, this revolution is already almost completed; in others it is incomplete, though making progress. Alison of Edinburgh, a teacher of Sir William Gairdner, urged in the twenties of last century the propriety of *supporting* fever patients instead of *depleting* them (*e.g.*, by blood-letting), and two of his pupils, Graves and Stokes of Dublin, went to considerable lengths in the use of alcohol as a stimulant. This practice spread, and received powerful support from the teaching of Liebig in 1858, that alcohol is a food. The well-known Dr. Robert Bentley Todd of King's College Hospital, pushed this mode of treatment to an extreme, and gave as much as 48 oz. of brandy per diem to a girl of 18 with pneumonia and enteric fever.[1] Although she became drowsy—in consequence, as Todd himself suspected, of "over-stimulation"—he kept up this dosage for twelve days (except on two days, when he reduced it temporarily to 36 oz., with, as he thought, unsatisfactory results). This practice is now obsolete, and the man who, more perhaps than any other, was influential in showing how undesirable the results were, was Sir William (then Dr.) Gairdner, formerly physician to the Glasgow Fever Hospital.

The following statistics, selected from Gairdner's writings,[2] show how fully justified he was in asserting that typhus, in young subjects at least, needed no stimulation by alcohol:—

		Mortality at all Ages.	In the Young.
Glasgow Fever Hospital—years immediately before Gairdner's appointment,	1861,	18·86 per cent.	3·2 per cent.
	1862,	16·92 ,,	3·6 ,,
Glasgow Fever Hospital—cases treated by Dr. Gairdner in 1862-63, .	...	11·93 ,,	less than 1·0 ,,
London Fever Hospital, .	...	20·89 ,,	6·2 ,,
Various hospitals, including Glasgow (statistics collected by Murchison), .	...	18·0 ,,	...
Todd's cases (quoted by Murchison), . . .	...	25·0 ,,	c. 17·0 ,,

Gairdner's cases included 189 in persons under 16 years of age, and of these only 1 died, viz., a child who was admitted

[1] *Clinical Lectures*, 1861, second edition, p. 122, Case 24.

[2] *On Alcoholic Stimulation*, Edinburgh, 1869; *The Physician as Naturalist, &c.*, Glasgow, 1889.

moribund, and was not seen during life by Dr. Gairdner. Alcohol was given in 1 case only of the 189, but this was long after the fever had run its course. The patient was a child with cancrum oris and great debility of the stomach.

Gairdner calculates that, if his 189 young patients had died at the same rate as in the

Glasgow Fever Hospital (1861-62),	there would have been	9	deaths	instead of	1.
London Fever Hospital,	,,	,, c.12	,,	,,	
King's College Hospital (under Todd)	,,	,, 30 to 35	,,	,,	

But nowadays, for one patient who gets alcohol as a food, there are probably a dozen or more who get it as a cardiac stimulant. Alcohol, in a pretty strong solution, by irritating the mucous membrane of the stomach and of the mouth, causes not only local dilatation of blood-vessels, but also a rise of the general blood-pressure, and therefore an increase in the velocity of the intracranial circulation. We can thus understand the utility of brandy and whisky in cases of fainting, particularly as one or other of these is generally at hand; but it has been found that 15 drops of tincture of capsicum will cause a rise of blood-pressure even less transient, and therefore more satisfactory, than that caused by $\frac{1}{2}$ or $\frac{3}{4}$ of an ounce of whisky. But it is obvious that reflex stimulation of the circulation, by the application of an irritant to the mucous membrane of the digestive tract, is not a procedure which it is desirable to keep up for days or weeks. The unsatisfactory and even risky nature of this treatment is emphasised by an experiment of Briggs,[1] which showed that the rise of blood-pressure in a temperate adult, after $\frac{1}{2}$ to 1 oz. of whisky, never lasted more than thirty minutes, and was followed by a slight but more lasting fall *below the previous level.* Clearly, this is not the kind of stimulation we want in acute or exhausting disease. We want something that will act directly upon the cardiac muscle or ganglia.

I fancy we were all trained as students to regard alcohol as a cardiac stimulant of the kind just indicated—a stimulant which, if it did not actually add to the energy of the cardiac muscle, would encourage it to use more freely such reserve of strength as it possessed, and would postpone the risk of final collapse until the natural decline of the fever, aided by feeding, nursing, and medicines, should set aside that risk, and allow the exhausted organ to recuperate. Of all aspects of the alcohol question—so far as therapeutics are concerned—this is by far the most important. Is alcohol a cardiac stimulant or not?

[1] *Bull. Johns Hopk. Hosp.*, 1903, p. 36.

I submit that the experimental evidence is strongly against alcohol, while the clinical evidence is, at the least, not very favourable.

Ringer and Sainsbury,[1] experimenting on the different alcohols by means of an isolated frog's heart, fed with bullock's blood, found that they all arrested the heart in diastole. I quote these words from their report:—"It is to be noted that, by their direct action on the cardiac tissues, these drugs are clearly paralysant, and that this appears to be the case from the outset, no stage of increased force of contraction preceding."

Experiments carried out by Martin and Stevens[2] in 1887, on the isolated dog's heart, also showed that diluted ethyl alcohol poisoned the cardiac muscle tissue, though it possibly acted, in addition, on the coronary arteries. The result was greater and greater stretching of the ventricular wall, with less and less complete systole, until the distended heart filled the pericardium and could stretch no more.

The most striking experiments, however, are those of Hemmeter,[3] who isolated the heart and lungs of dogs, supplied them with defibrinated calf's blood or dog's blood, and measured the quantities of blood pumped around by such hearts, under varying conditions, in periods of thirty seconds each. One observation may be quoted as illustrative of the series:—

Supplied with non-alcoholised blood, the heart propelled						196	c.c. in	30 seconds.
1 minute after alcoholised blood was turned on, it					,,	186	,,	,,
3 minutes	,,	,,	,,		,,	175	,,	,,
6 ,,	,,	,,	,,		,,	162	,,	,,
7 ,,	pure	,,	again turned on,		,,	194	,,	,,
3 ,,	alcoholised	,,	,,		,,	174	,,	,,

This experiment was made with 0·2 per cent methyl alcohol. Ethyl alcohol was not quite so hurtful, the loss of work in thirty seconds amounting to 17·45 c.c. with ethyl, as compared with 19·46 c.c. with methyl alcohol.

The diminution in the work done by the heart when supplied by alcoholised blood was accounted for by dilatation and imperfect systole.

There was no change in the pulse-rate which could be attributed to the alcohol employed.

These very important results of Ringer and Sainsbury, Martin and Stevens, and Hemmeter, are quite in accordance with the teaching of a more recent English writer, Leonard

[1] *Practitioner*, 1883, vol. i, p. 339.
[2] *Studies from Biol. Lab. Johns Hopk. Univ.*, 1887, vol. ii, p. 213.
[3] *Studies from Biol. Lab. Johns Hopk. Univ.*, 1889, vol. iv, No. 5.

Hill,[1] who says that alcohol diminishes the systole and increases the diastole. When the dilatation of the heart has become so great that the organ fills the pericardium, cutting the latter gives temporary relief, but at last the movements cease in diastole.

How, then, did alcohol come to be so generally regarded as a cardiac stimulant? The question has been answered for us by Sir Samuel Wilks,[2] who says that "those who in times past gave it and found it useful were totally mistaken as to its action. They believed they gave a stimulant instead of a sedative, when they found it beneficial in such a case as fever, with high temperature, quick pulse, &c. This mistake led it to be so universally given in all cases of disease. If alcohol gave strength, there was no morbid state in which it was not useful." For my own part, I used to give alcohol freely in pneumonia, the principal fever with which we have to deal in a general hospital, but the results were disappointing. For the past two years or so I have scarcely given it at all in that disease, except occasionally as a narcotic or sedative, for instance at the crisis, and I am convinced that the patients have not suffered. I am disposed to think that they have benefited.

Another important group of cases consists of those in which the heart muscle is failing as a late result of chronic valvular disease. It is probably still the general practice to give alcohol to such patients, and yet Wilks[3] pointed out a dozen years ago that its influence in cardiac cases, with orthopnœa, dropsy, and engorgement of viscera is harmful. For such patients, mercurial purges, leeches, nux vomica, and often digitalis, are immeasurably superior to alcohol. It is noteworthy how many of these patients object to alcohol, not, apparently, because they do not like it, but because they recognise that they are none the better of it. Some patients, on the other hand, ask for it, but probably even these will be satisfied with a short course of the drug. I should say that patients of this group are at least as well without alcohol. If alcohol, taken in liberal quantities, weakens the heart of an otherwise healthy individual, it can scarcely be expected to strengthen a heart that is already weakened.

A phenomenon which gives us some concern in the later stages of fever is a great fall in the tension of the pulse, which becomes dicrotic, fully dicrotic, or even hyperdicrotic. When

[1] Schäfer's *Text-Book of Physiology*, 1900, vol. ii, p. 52.
[2] *British Medical Journal*, 1891, vol. ii, p. 462.
[3] *Loc. cit.*, p. 463.

the blood-pressure is so much reduced, there can be no great stability in the supply of blood to the heart itself, or to the nerve centres in the medulla. The question then is—Can alcohol help us here? Observation shows that in intoxicating and deadly doses alcohol causes a fall in blood-pressure. With regard to small doses, opinions differ so widely as to make it probable that these doses make very little difference one way or another. Nevertheless, the most recent researches point to the absence of any rise, and to the occurrence of a distinct fall, in pressure. If alcohol is given by the mouth, or by the rectum, or hypodermically, it causes a reflex rise owing to the tissue irritation it produces; but if this irritant effect is eliminated—*e.g.*, by giving a considerable amount of alcohol hypodermically, but diluted with plenty of normal saline solution—there is no rise; whereas true stimulants given in this manner cause a rise. We may take it, then, that absorbed alcohol cannot raise the blood-pressure, whether that pressure is normal or below normal to begin with.

The rapidity of the pulse is another point that has to be considered. Opinions differ widely as to the influence of alcohol on the pulse-rate. Some say that it causes (1) acceleration; others say (2) slowing; others (3) acceleration followed by slowing; others (4) slowing followed by acceleration; and others (5) little or no change. Parkes, as a result of his earlier researches, adopted the first of these five theories, while his later researches led him to adopt the third. The diversity of opinion seems to depend largely on the existence of remarkable differences between different individuals. Anstie, in his work on *Stimulants and Narcotics* (1864), showed that a great increase of rapidity after alcohol was much more marked in the lower mammals than in man. Dr. Findlay carried out many observations in connection with this subject on patients in my wards, and the net result of his and of the earlier recorded experiments appears to be that frequently, but not always, there is a slight and transient increase in the pulse-rate. The total for the whole twenty-four hours, however, is not increased; occasionally, indeed, it is diminished. Parkes found that exercise greatly increases the pulse-rate of those who have taken alcohol—an observation which is not in favour of that drug.

One other matter has to be alluded to, viz., the influence of alcohol on temperature. It has been supposed by some that alcohol retards tissue metamorphosis to such a degree that it acts as an antipyretic. The most satisfactory evidence goes to show that any such action, if it occurs at all, is quite

insignificant. At the same time, through its power of causing vasomotor paralysis, and thus relaxing the cutaneous blood-vessels, large doses of alcohol are capable of exerting a very important influence on temperature, but the result depends upon the temperature of the external medium. The skin, when rendered hyperæmic by vasomotor relaxation, is a better conductor of heat than normal skin, and thus allows the temperature of the body to approximate to that of the surrounding atmosphere more nearly than in health. It thus happens, on the one hand, that very low temperatures are found in intoxicated persons who have lain out through a winter's night, and, on the other hand, that alcohol in hot climates predisposes to sunstroke. Apart, however, from its influence on the vasomotor system, we may discard any antipyretic effect of alcohol as insignificant, or non-existent.

After all this destructive criticism, I wish to point out that alcohol has properties which, under certain circumstances, may be very useful, although, as a rule, we can obtain similar results in disease by means of other agents which are free from the objections that may be urged against alcohol. For instance, whisky may with advantage be applied to the lips in syncope to cause a reflex rise in the blood-pressure. Sometimes it proves a suitable narcotic. It may also be given in cases where, after severe exposure, the surface remains persistently cold after the individual has been brought into a warm atmosphere; here, after absorption, it relaxes the cutaneous vessels by causing vasomotor paresis. It might be used to relieve the pain of neuralgia or dysmenorrhœa, but in such conditions it is a dangerous remedy, because the dose is likely to be often repeated; fortunately, better remedies are available.

To sum up, then, alcohol is a medicine with certain useful properties, but the limitations of its usefulness are far greater than is ordinarily supposed; and, even where it is useful, there are generally other remedies which are also useful, and at the same time more safe.

[Dr. Findlay.]

In studying the physiological action of alcohol on the cardio-vascular system, one is more impressed with the harmlessness than with the harmfulness of this agent; indeed, it is a matter of perpetual wonderment to see how large and even enormous quantities can be borne with comparative impunity. When, however, the circulatory system is in a state of disease,

alcohol, with few exceptions, is not only useless as a remedial agent, but is worse than useless, for it is frequently positively harmful. In a state of perfect health, the cardio-vascular system has a large reserve to draw on; in disease, this system is frequently working with the merest margin, or with no margin at all, and to give alcohol is to destroy that margin or to upset the balance altogether.[1] Thus, Dr. Monro has already alluded to the fact that alcohol frequently causes a distinct fall in blood-pressure; and further, Crile,[2] in experimenting on dogs reduced to a state of shock ("exhaustion of the vasomotor centre"), found that the most constant and the most marked effect of alcohol was a decline of the blood-pressure, the rapidity and the extent of which were proportional to the depth of the shock and the dosage of the alcohol employed. When considerable dosage had been given, the final breakdown of the circulation was more sudden than in the control animals.

Alcohol, even in small doses, is a vaso-dilator, and though it causes some loss of tone in the vessels of the body generally, it exerts its dilating influence principally on the vessels of the skin, and at the same time increases the rate of blood-flow within the cranium. This dilatation of blood-vessels is preceded by no vaso-constriction, unless alcohol be given in such a manner as will evoke reflex effects.[3] Alcohol is of therapeutic use in all conditions of arterio-capillary spasm, but the only advantage it has over other drugs of this type is that it is ubiquitous. It is insisted by most authorities that alcohol is never at any time a stimulant of the vaso-constrictors, but Lauder Brunton[4] suggests that in fevers alcohol may increase the power of the weakened vasomotor centre, either by acting upon it directly or by augmenting its blood-supply, and may thus give greater tone to the blood-vessels and raise the pressure within them. But it may be seriously questioned whether alcohol confers any benefit by increasing the cerebral blood-supply, for, as most authorities hold, and as Lauder Brunton himself elsewhere admits, alcohol has a paralysing influence on the nerve-cells themselves. Accordingly, the more quickly the alcoholised blood flows past the nerve cells, the

[1] J. Swientochowski, *Zeitschr. f. klin. Med.*, 1902, Bd. 46, SS. 284-310 (quoted in *Internat. Monatsschr. zur Erforschung des Alkoholismus und Bekämpfung der Trinksitten*, 1903, S. 33).

[2] *Boston Med. and Surg. Jour.*, 1903, p. 248.

[3] J. C. Hemmeter, *Trans. Med. and Chir. Fac. State of Maryland*, 1889, pp. 230, 231.

[4] *Practitioner*, 1876, xvi, 130.

more alcohol is brought in contact with them, and the sooner will they succumb to its narcotising influence. Besides, there is no longer any reason to imagine that alcohol may raise the blood-pressure when it has been indubitably shown that alcohol, if it affects blood-pressure at all, can only lower it.[1]

Binz[2] declares that the vessels of the skin are usually greatly contracted in high fever, hence the benefit of alcohol. Now, in the initial stage of fever, during what we term the rigor, there is vaso-constriction, and here, though there is no urgency about the condition, a dose or two of alcohol may contribute greatly to the patient's comfort by inducing vaso-dilation. But in the later stages and anxious periods of most fevers, there is already well-marked vaso-dilation, and, indeed, this of itself constitutes a distinct menace to life, for it would appear that the muscle of the vessels plays an important part in the onward propulsion of the blood, and, according to Pässler and Romberg,[3] death in the infections comes about from paresis of the constrictors of the small vessels, apart altogether from cardiac disability. Alcohol, however, cannot amend this state of matters; it is no more a vascular tonic than it is a cardiac tonic. At the same time, alcohol may not make matters worse in this respect, for frequently the fever has already dilated the vessels to a maximal degree, and no further vaso-dilation can be observed after alcohol. In cases of arterio-sclerosis, also, we find that alcohol can exert no influence, or but an insignificant dilating action on the rigid blood-vessels, just as we know that in these same cases digitalis can exert no considerable constrictor action.

Much diversity of opinion has been expressed regarding the effect of alcohol on the pulse-rate, but the most careful work of the last fifteen years shows beyond a doubt that, even in quantities sufficient to produce transient disturbance of the mental faculties, pure ethyl alcohol diluted with water has practically no influence on the pulse-rate of healthy men.[4] So

[1] H. W. Cook and J. B. Briggs, *Johns Hopkins Hospital Reports*, 1903, vol. xi, pp. 518-524; J. J. Abel, *A Critical Review of the Pharmacological Action of Ethyl Alcohol*, &c., Boston, 1903 (quoted by Cook and Briggs, *loc. cit.*).

[2] *Lectures on Pharmacology*, New Syd. Soc. *Transl.*, 1895.

[3] *Kongress f. innere Med.*, 1896 (quoted by Rosenfeld, *Der Einfluss des Alkohols auf den Organismus*, 1901, S. 52).

[4] Hemmeter, in 1889 (*Trans. Med. and Chir. Fac. State of Maryland*, 1889, p. 230) made such a statement, and further research has but confirmed the truth of it. Meyer (*Bericht über d. vii. internat. Congr. gegen d. Alkoholismus*, 1902, S. 43) and Rosenfeld (*Der Einfluss des Alkohols auf den Organismus*, 1901, SS. 50-52) express a like opinion.

certain is this that we may with confidence assert that, if half a glass of whisky accelerates the pulse to any appreciable extent, then the owner of that pulse is not in a condition of perfect health. The strength of the pulse in the healthy likewise remains unchanged, though the pulse may feel fuller to the examining finger. No support can be found for the view that there is ever any increase in the pulse tension after alcohol, either in health or in disease.[1] Alcohol cannot slow the abnormally rapid pulse of disease, unless that rapidity is due to some functional disturbance which the narcotising influence of alcohol can quiet. There is not a particle of evidence to prove that alcohol can stimulate the vagus mechanism, and, indeed, so far as we can find, absorbed alcohol is unable to influence any of the cardiac nerves.[2] Speaking somewhat generally, it may be said that the pulse-rate depends on the condition of the cardiac pump—the heart, and on the state of the blood-vessels. Dr. Monro has already shown that at no stage of alcoholisation is the cardiac muscle stimulated, and that in large doses alcohol acts on the muscle fibre of the heart as a deadly poison.[3] I have already mentioned that alcohol cannot help the circulation by tightening up the greatly relaxed blood-vessels in the severe infections, though it has also been pointed out that alcohol may be unable to make matters worse so far as the blood-vessels themselves are concerned. In these cases of high fever the heart beats very quickly, for, in order to deliver a sufficient quantity of blood to the tissues through dilated vessels, an enfeebled heart can only do so, or attempt to do so, by contracting more frequently, and it may be said that the frequency of the pulse bespeaks the urgency of the cardiac enfeeblement. If alcohol be given under these circumstances, it can only cause further embarrassment to the heart; the blood-pressure, unless the heart can put on a still quicker pace, or even in spite of a quicker pace, invariably falls. If the disease has already so seriously affected the cardiac musculature that the further disabling action of the alcohol on the muscle of the heart cannot be compensated for by an increase in the pulse-rate, then the heart gives in—throws up the sponge, so to speak—and beats irregularly and slowly.[4]

Though all the newer research has given, or should have

[1] T. K. Monro and J. W. Findlay, "Alcohol and the Cardio-vascular System," *Med. Temp. Review*, 1904, p. 39.

[2] *Ibid.*, p. 78.

[3] *Ibid.*, 1903, pp. 331-335 and 361-374.

[4] J. Swientochowski, *Zeitschr. f. klin. Med.*, 1902, Bd. 46, SS. 284-310.

given, the death-blow to alcohol as a cardiac stimulant, we are by no means finished with the alcohol question. Not so very long ago, alcohol was extolled as almost a specific in sepsis and the infections, and at this moment many look on alcohol as a remedy capable of neutralising microbic toxines, while others hold that to give alcohol in these conditions is but adding fuel to the fire.[1] Clinical experience in this field is most contradictory, and even laboratory results do not give us the positive information we desire. Several observers have shown that alcohol renders animals more susceptible to microbes and their toxines; they show that alcoholised animals are immunised with difficulty or not at all, and that the continued administration of alcohol causes animals to quickly lose any immunity they may have acquired.[2] But these results teach us nothing that we can make use of for man, since we find that, had a man received as much alcohol in proportion to his weight as these unfortunate animals received, he would have been drinking not much less than half a bottle, and sometimes even more than three bottles of 50 per cent brandy in the twenty-four hours.[3] Now, these large quantities of alcohol caused digestive disorders, emaciation, hypothermia, and other disturbances in the animals experimented on, so that additional factors are introduced to prevent us transferring *in toto* these results to man. Nevertheless, it appears to be pretty well proved and accepted that alcoholism in man predisposes the body to infection by various microbes, and we might quite logically infer that, since this is so, it must be equally true that alcoholic therapeusis will predispose to an extension of the morbid process, which is merely a continued infection and invasion of other and surrounding tissues. Such reasoning, however, is not altogether justified by experimental results; still, support is lent to such a view by some of the most recent researches, though, with therapeutic doses—equal to from about 2½ to 4 oz. of 50 per cent brandy for a man—results are not so telling against alcohol.[4]

It is pretty well settled that alcohol diminishes the alkalinity of the blood, and it is known that diminished alkalinity of the blood goes hand in hand with increased susceptibility to infection; consequently, we must probably regard this diminished alkalinity, that alcohol can cause, as a factor likely to

[1] Pearce Gould, "Alcohol in Surgery," *Practitioner*, 1902, vol. lxix, p. 573.

[2] Sims Woodhead, "The Pathology of Alcoholism," *Med. Temp. Review*, 1903, p. 105 *et seq.*

[3] *Glasgow Med. Jour.*, 1904, vol. lxi, pp. 237, 238.

[4] *Glasgow Med. Jour.*, 1904, vol. lxi, p. 238.

interfere with the acquirement of immunity. Woodhead and others consider it proved that alcohol interferes with phagocytosis, and diminishes and may even abolish the bacteria-destroying (bacteriolytic) power of the blood, though only the other month the well-known American pharmacologist, Hobart Amory Hare,[1] brought forward experimental evidence showing that alcohol enhances this power. This whole question is still to a great extent *sub judice*. Alcohol has been wrested from many a proud position by the hardest of hard and painstaking work, but its vitality is tremendous, and as soon as it is dislodged from one high place it turns up in another. Indeed, so tremendous is this vitality of alcohol, that one feels almost compelled to believe that it must be more than a narcotic, vaso-dilator, and reflex stimulant; but it has yet to be proved that alcohol can play the *rôle* of an antitoxine or a generator of antitoxines.

[1] *Therapeutic Gazette*, 15th May, 1903.

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