Cardiac output can also be measured by impedance plethysmography, an entirely different technique. Current is passed between circular electrodes round the neck and the lower thorax, and the electrical impedance of the thorax is measured between a second pair of electrodes adjacent to the first. As blood leaves the chest with each cardiac cycle the impedance of the system changes, and the cardiac output can be calculated. Reports vary about the correlation of results obtained in this way with those of standard techniques,[5,6] but impedance plethysmography is generally thought to be better at measuring changes in output in an individual than in making absolute output measurements.

A newer—and more expensive—technique is based on doppler echocardiography;[7] the reflection of ultrasound off moving red cells can be used to measure blood velocity, most conveniently in the ascending aorta. With each systole the velocity rises and falls, so the doppler record is a curve of velocity plotted against time. Integration of a velocity/time curve gives the "stroke distance", which can be thought of as the distance moved by a particle of blood, or alternatively as the length of a column of blood that passes a fixed point in a given time. Multiplying stroke distance by the cross-sectional area of the aorta gives the volume of that column, which can be corrected to ml/min—the stroke volume—and so the cardiac output can be derived. The results of this technique correlate well with others[8] but considerable experience is needed by the operator and measurements of the aortic cross-sectional area can be inaccurate. It has been suggested that stroke distance alone is an adequate measurement of cardiac function,[9] but claims that it correlates with stroke volume have not been substantiated.

The search for noninvasive techniques that measure something related to cardiac output led to various methods of measuring ventricular volume. In future, these measurements will probably be made most accurately by magnetic resonance imaging because of its ability to take pictures in multiple planes. Meanwhile, two-plane cineangiography is most precise, and echocardiography is a rather poor second. Assessment of ventricular volume depends on a formula that assumes the ventricular cavity to be a prolate ellipsoid,[10] but this approximation takes no account of cavity content such as papillary muscles, and none of abnormal wall movement. The difference between ventricular volumes at the end of diastole and the end of systole (the ejection fraction) can be assessed reasonably accurately, but not the absolute cardiac output. The ejection fraction is most easily determined by radionuclide labelling of the circulating blood; electrocardiographic gating is used to measure counts at the end of systole and diastole, which correlate with the volume of blood in the ventricle. Again, the measurements cannot be used accurately to assess actual cardiac output.

The enthusiasm with which new techniques are pursued disguises the fact that their results are not clinically very important. There is considerable evidence that the ejection fraction is an indicator of prognosis, and some workers seem to assume that if the ejection fraction is improved the patient will live longer. It is more likely that a low ejection fraction is simply a marker of bad heart disease, and that prognosis will depend on the nature of the underlying disorder. Measurement of cardiac output is not much more helpful: it can give normal results at rest in patients with severe heart failure, and resting outputs do not correlate with symptoms in chronically ill patients, although the response of cardiac output to exercise may provide a better measure of cardiac function. Cardiac output is greatly affected by factors other than cardiac performance, such as filling pressure, afterload, and myocardial contractility, which in turn is affected by neuroendocrine factors and by some drugs. Distribution of blood flow to brain, kidneys, and other vascular beds is probably more important than the output itself, so in the critically ill patient assessment of output adds little to haemodynamic monitoring and observations of renal output. However, it is probably true that therapeutic manoeuvres will only improve a patient's symptoms if there is an increase in cardiac output, so the development of a simple, safe, reliable, and cheap method of assessment remains a worthwhile objective.

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Kava

ATTENTION drawn to the plight of the Aboriginal people in Australia was an unhappy side of the otherwise vibrant bicentennial celebrations earlier this year. The problems caused by alcohol consumption in Aborigines are widely recognised; less well known are the effects of kava,[1] the traditional native drink of the Pacific islanders, which was introduced into Aboriginal communities in Arnhem Land in the "top-end" of Australia in the early 1980s.

Kava—the Tongan and Marquesan word for bitter—is the name for an Australasian shrubby

1. Gregory RJ, Gregory JE, Peck JG The relationship of kava to a cultural revitalization movement Report to the US Public Health Service, National Institute of Drug Abuse, 1977.

pepper (*Piper methysticum*) and the beverage prepared from it. The beverage that the natives prepare from the crushed rhizome and roots of the plant is pungent in the mouth, with an anaesthetic effect on the tongue. Kava is valued in island communities such as those of Fiji, Tonga, and Vanuatu, where it is ritually consumed by convened groups. Captain Cook described the native use in the account of his voyages in the South Seas in 1768. Consumption of small regulated quantities, accompanied by feasting, produces no obvious adverse effects, and kava is valued overall for its minor tranquillising and relaxant responses, and as a vehicle of social function.[2]

Germans became interested in the pharmacology of kava during their days of empire in the Pacific in the late 19th century and the first medical monograph on the subject was published in Germany in 1896.[3] German pharmaceutical companies marketed kava for its diuretic and genitourinary antiseptic qualities but this market was short-lived because kava in larger doses has unwanted effects. These effects have come to notice again in the Aboriginal consumers.[4,5]

The early medical anthropologist, W. H. R. Rivers,[6] attached such importance to the choice between kava root and betel nut that he believed the Melanesian people could be classified into two great divisions—the betel people and the kava people. His theory of Melanesian migration is based on this distinction. Anthropology is the most complex science known to man—Melanesians pay homage to this principle by wrapping their betel chew in leaves from the kava pepper plant.

Kava's chemistry and pharmacology, which is unique among psycholeptic substances, has lately been studied by Duffield and his team[7,8] in the biomedical mass spectrometry unit of the University of New South Wales. Kava is a suspension of lipid material in water. The major proportion of the lipid components consists of six chemically well-defined compounds containing the 4-methoxy-2-pyrone ring system. Duffield's group also identified another dozen trace constituents of urine, and with this information they began to study the metabolic transformation of these lipids in kava consumers. Investigations in volunteers showed that many of the lipid components are secreted in urine, together with several metabolites to which tentative chemical structures have been assigned. Tests in mice showed that ten minutes after injection of lipid extract, only one of the chemical constituents was found in the brain, but after forty-five minutes ten kava components were present. The aqueous fraction of kava, devoid of lipid material, had a sedative effect on the rodents. Dialysis experiments suggest that the active material has a molecular weight of less than 10 000; this material has not been identified by mass spectrometric techniques, which suggests that it could be a polar molecule of molecular weight 1000–10 000.

Matthews and his co-workers[4] at the Menzies School of Health Research in Darwin, Northern Territory, have now studied the clinical effects of the cult for kava in Arnhem Land. There is a clear preference for kava over alcohol in some Aboriginal communities because it does not cause the same violent behaviour. Nevertheless, preliminary results of a comparison between Aboriginal users and non-users show that kava drinkers are more likely to have general ill health, including shortness of breath and a characteristic rash; malnutrition, with 20% loss of body weight, 50% loss of body fat, and other biological changes; liver damage, with biochemical changes similar to those caused by large doses of alcohol; and other changes in red blood cells, white blood cells, and platelets. Although more research is needed, it is prudent to assume that heavy kava usage can be very harmful to health. Accordingly, Aboriginal people in Arnhem Land should be strongly advised to reduce consumption of kava; people who continue to drink it should be counselled to eat a more adequate diet. Moreover, kava should not be introduced into Aboriginal communities where it is not yet available.

These observations and related research findings are being used by the Commonwealth, State, and Northern Territory governments to guide the development of their policies on the availability of kava and on public information activities. Knowledge of the psychopharmacology of kava is still incomplete. Cawte[9,10] has alluded to the views and reactions of kava consumers and non-consumers in the same population. Kava users, according to the distinction popularised by David Reisman, are in general other-directed, while non-users are more inner-directed. Non-consumers are also of higher social status, a category threatened by modern camp living under foreign domination. Cawte noted that kava had a beneficial effect upon Aboriginal chronic schizophrenia, but, given the side-effects, this aspect is hardly worth pursuing.

The interested student of kava in all its modern aspects is probably best referred to the report by Dr Robert J. Gregory,[1] Department of Psychology, Massey University, Palmerston North, New Zealand, to the US National Institute of Drug Abuse in 1977.

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