**Princeton Guide to Advanced Physics.** Alan C. Tribble. Princeton University Press, 41, William Street, Princeton, New Jersey 08540, USA. 1996. 397 pp. Price: $ 59.50, Clothbound.

It is widely acknowledged that the ability to solve problems is a most essential part of higher education in physics. Dissatisfaction with standard university curricula in our country in this regard is equally widespread. For a variety of reasons, our 'system' is simply unable to instil any notable degree of skill in problem-solving in students who are awarded B Sc and M Sc degrees in physics.

In this situation, any effort that helps propagate the problem-solving culture must be welcomed. Over the years, a number of collections of problems with solutions have appeared. Restricting oneself to the graduate level, and leaving out collections on specialized subjects (especially quantum mechanics), mention may be made of *University of Chicago Graduate Problems in Physics* by J. A. Cronin *et al.* (Addison-Wesley, 1967); *University of California, Berkeley Physics Problems* by M. Chen (Prentice Hall, 1974; Prentice Hall India, 1990); *Problems in Theoretical Physics* by L. G. Grechko *et al.* (Mir, 1977); and *Princeton Problems in Physics* by N. Newbury *et al.* (Princeton University Press, 1991). There are undoubtedly several other collections of this sort, covering all the standard subjects – classical and quantum mechanics, statistical physics, electromagnetism, etc. I opened the book under review with the expectation that it might be another such collection, but it is actually a 'comprehensive one-volume reference book' as proclaimed on the cover. The blurb on the back cover starts by saying, 'From classical mechanics to general relativity, the key principles in all areas of physics are surveyed in this one handy volume'. Indeed, the 12 chapters of the book do range over a wide pasture – mathematical methods, classical mechanics, electrodynamics, etc. Brief derivations are given of standard basic formulas and material in each subject, with some illustrative examples. The book reads like the set of note-books a graduate student preparing for a comprehensive exam is likely to build up – just the essentials, no deep insights, somewhat random in the detailed choice of topics, and hence somewhat disconnected; in short, no different from what we in this country would succinctly categorize as a guide rather than a textbook.

Now, a guide that is not too bad could actually be more effective than a combination of poor teaching and a textbook that is either bad, or is written at too inaccessible a level for the students concerned. The guide could then help build up the student's self-confidence and open the doors to more reliable and systematic sources of information. However, one must allow for the fact that guides are generally non-rigorous and superficial, and could be often misleading and occasionally downright wrong. The question, therefore, is: how reliable a guide is the one under review? It is, after all, published by Princeton University Press – an establishment whose output in physics and mathematics is known to be of the highest standards of excellence.

Unfortunately, I find that the book is not up to the mark. The number of incorrect statements scattered all over the book is simply too high to be acceptable, and often of a seriousness too great to be dismissed as arising out of carelessness or informality of presentation. Facile definitions and incomplete statements may be acceptable under the 'guide' framework, but totally erroneous ones cannot be condoned. Leaving aside even serious typos, let me list a few of the glaring errors I found on merely flipping through the pages of the book, to let readers judge for themselves:

p. 4: $\delta(x - x')$ is defined to be equal to 1 when $x = x'$.

p. 26: For $f(z)$ *analytic* in a neighbourhood of $z = a$, and $C$ a simple closed path surrounding $a$, it is claimed that $\int_C f(z)dz = 2\pi i$ Res $f(a)$. This is called the residue theorem! It is then *followed* by what happens when $f(z)$ has an isolated singularity at $z = a$, and it is claimed that

$$\text{Res } f(a) = \lim_{z \to a} (z - a) f(z)$$

for *any* isolated singularity at $z = a$ (!).

p. 30: In a section on tensors, the coordinates are introduced as covectors rather than vectors, and 'equations' such as

$g_{ij} = \partial x_j / \partial x_i'$, $A'^i = g_{ij} A^j$, etc. are written down, with absolutely no regard for the distinction between upper and lower indices, in a presumably arbitrary space!

p. 37: The discussion of the 2-body central force problem suggests that the author has simply equated $(d\vec{r}/dt)^2$ with $(dr/dt)^2$. It is also stated that a central force between two particles implies 'an interaction potential $V$, where $V$ is any function of the vector between the particles' and $V$ is subsequently written as $V(r, \dot{r} \ldots)$!

p. 39: The area swept out by the radius vector of a particle in the Kepler problem is claimed to be $A = (1/2)\ r\ (r\ \theta)$ !

p. 84: $\left| \vec{R} - \vec{r} \right|^{-1}$ is expanded as

$$(1/R) \sum_{n=0}^{\infty} (r/R)^n P_n(\cos\theta)$$

*for* arbitrary *(but finite)* $R$ and $r$ !

p. 194: Having used $|\psi\rangle$ to denote the *general* state of a system, the author goes on to say: 'If one measures the observable corresponding to the operator $A$ and finds the value $\lambda_n$ then immediately after the measurement the state vector satisfies $i\hbar\, \partial|\psi\rangle/\partial t = H|\psi\rangle$. We say that the measurement projects the original state vector onto eigenvectors (*sic*) of A.'

p. 198: '. . . quanta of matter or radiation must be represented by wave packets. For this reason the state function $\psi$ is also called a wave function. That is, $\psi$ must have properties of plane waves, $\psi \propto \exp i(kx - \omega t)$.'

p. 225: The eigenvalues of $L_z/\hbar$ are integers, it is claimed, because the wave function and its derivative 'must be continuous over the range 0–2π' of the azimuthal angle!

One could go on, but here's a good punchline:

p. 307: 'All mesons are unstable *and have no distinct antiparticles*.'

Need one say more? Book-writing is (or at least ought to be) a sobering exercise for both authors and publishers. It brings out so tellingly the need for painstaking attention to the smallest detail. This is all the more important in a society where students (and also administrators!) regard the printed word as sacred. In the spirit of these quotations, let me end with a memorable quote from a well-known elementary text on materials science. It is not my intention to add gratuitous insults, but to bring out the perils attendant upon attempting broad definitions without adequate care. In *Vol. I: Structure* by W. G. Moffatt, G. W. Pearsall and J. Wulff, of the 4-volume text *The Structure and Properties of Materials* (Wiley, 1964; Wiley Eastern, 1974), at the end of the very first chapter (on electrons and bonding), one

finds a set of 'definitions'. Among them are:

'Quantum mechanics: A branch of physics in which the systems studied can have only discrete values of energy, separated by forbidden regions; it is distinguished from continuum mechanics in which a continuum of energies is assumed possible.'

'Wave mechanics: A branch of mathematical physics involving the statements and solutions of differential equations describing wave behavior.'

Who says science is humourless?

V. BALAKRISHNAN
*Department of Physics,*
*Indian Institute of Technology,*
*Madras 600 036, India*

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**Myocardial Ischemia: Mechanisms, Reperfusion, Protection**. M. Karmazyn, ed. Birkhauser Verlag, P.O. Box 133, CH – 4010, Basel, Switzerland. 1996. 528pp. Price: SFR 198, DM 238.

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Myocardial infarction is one of the most common causes of hospital deaths. Thirty years ago it was generally accepted that insufficient blood flow due to coronary artery obstruction by atherosclerotic process is the cause of myocardial ischemia and that infarction is precipitated by occlusion of coronary artery by thrombus. Therapeutic approaches were aimed at thrombolysis and limiting the infarct size. Recent clinicopathological, angiographic and experimental studies indicate that additional structural and functional alterations are superimposed on chronic altherosclerosis and these are responsible for the development of various ischemic syndromes. Considerable clinical and experimental data have accumulated which make apparent that prognosis following infarction depends on the amount of residual viable normally functioning mycardium and that interventions which would decrease the extent of tissue death will reduce mortality in patients. It has also been recognized that restoring blood flow to the ischemic mycardium is not always associated with recovery but may lead to further damage, recognized as reperfusion injury.

Karmazyn has edited a book which reflects the growth of understanding of pathophysiological mechanisms in acute myocardial ischemia and the current approaches to define rational therapeutic strategies to salvage ischemic myocardium. The book contains 31 articles grouped in 9 sections with distinctive titles. The contributors are all internationally recognized investigators.

The first, third and fifth articles are on nitric oxide. Zhao, Hintze and Kaley examine evidences for mediation of reflex coronary artery dilatation in ischemic conditions by nitric oxide produced by vascular endothelium. Several studies have earlier shown that activation of sympathetic or parasympathetic nervous system controls the resistance of coronary arteries. Bezold–Jarish reflex, carotid chemoreflex, arterial baroreceptors and ventricular receptors participate in this regulation. The observation that selective impairment of vagal control of coronary blood flow is due to the impairment of nitric oxide synthesis suggests that nitric oxide is a mediator of neural regulation of coronary vascular resistance.

Dusting provides a more extensive review on nitric oxide; its biosynthesis, role in inflammation and pathological conditions such as atherosclerosis, reperfusion injury and heart failure. Studies have demonstrated that atherosclerosis leads to disturbances in the activity of isoforms of nitric oxide synthase in the artery and can contribute to vasospasm, thrombosis and cell proliferation. These defects may be reversed, thus offering a promising target for therapy against some manifestations of vascular diseases. There is also evidence that derangement in nitric oxide function is associated with the development of atherosclerosis and vascular remodelling after angioplasty. Production and actions of nitric oxide are also abnormal in chronic heart failure. Nitrate, the stable end product of endogenous nitric oxide production is elevated and endothelium dependent vasodilatation impaired in patients with heart failure and the levels are proportionate to the severity of heart failure. The causes for enhanced release of nitric oxide from vascular endothelium and its functional consequences are still unclear.

Pabla and Curtis survey experimental studies in animals which imply a role for endogenous nitric oxide as a mediator for cardiac protection in atherosclerosis, myocardial infarction, reperfusion induced arrhythmias and coronary artery restenosis after angioplasty. Studies using inhibitors of nitric oxide synthase suggest that reduction of nitric oxide production exacerbates the disease state. Probably, to protect cardiac function in disease conditions, it may be of value to mimick nitric oxide function or induce its synthesis or block its degradation. Interestingly, in experimental animals short term exercise training increases the production of nitric oxide in the coronary vasculature and enhances nitric oxide synthase gene production in endothelial cells. Nitric oxide dependent coronary vasodilatation is also improved. These mechanisms may be responsible for improvement of cardiac function and reduction in incidence of coronary diseases, seen in patients who undergo exercise training.

Myocardial ischemia and reperfusion are associated with increased production of another endothelium derived peptide, endothelin. Reperfusion of ischemic myocardium is a potent stimulus for increased release of endothelin as is evident by elevated plasma levels of endothelin in patients with coronary artery disease who have had thrombolytic therapy or angioplasty. Elevated plasma levels are associated with diminished ventricular function and increased mortality. A number of investigations have been carried out in animals to assess the effects of endothelin receptor antagonists and other inhibitors in ischemia and reperfusion. Results imply that modulation of endothelin synthesis or its actions could represent novel therapeutic stategies. The topic is exhaustively dealt by Karmazyn.

Oxygen-free radicals have been extensively examined as potential mediators of injury in ischemia and during reperfusion. Free radicals can cause cellular dysfunction directly by oxidation of cell structure components. Another mechanism which has gained attention in recent years is alteration or destruction of second messenger pathways in the cell which affect cellular ionic homeostasis, contractile function and viability. Czubryt, Panagia and Pierce discuss the studies which address the deleterious effects of oxidants on various components of the second messenger system. The second messengers are important for regulation of normal cardiac function. Changes that occur in the signal transduction system during ischemia and reperfusion have possibly a role in arrhythmogenesis and other functional abnormalities. Recent evidences that reac-