

No part of this website may be reproduced, stored in a retrieval system or transmitted in any form by any means electronic, mechanical, photocopying, recording or otherwise without written permission of the copyright owner. Building on: higher education at the local level. Explicitly integrating disability into higher education is essential to achieve inclusive learning environments. Inclusive practice must be an integral part of undergraduate and graduate teaching and learning. This paper discusses the rationale for integrating disability in higher education and the significant challenges that need to be addressed to do so. The paper considers examples from disability sport, employment, and higher education, and presents strategies and principles that can be adopted to create inclusive learning environments in academic institutions. The paper concludes with a series of recommendations for universities and others to reflect on their practices in relation to disability. Ischemia-induced alteration of calcium-induced calcium release in guinea pig left ventricular myocardium. The effect of myocardial ischemia on the sarcoplasmic reticulum (SR) Ca²⁺-ATPase and its regulation by Ca²⁺ and calmodulin (CM) was studied in guinea pig left ventricular papillary muscles. Ischemic conditions were obtained by clamping the aorta for 15 min. This treatment produced a significant (p less than 0.01) decrease in the muscle mechanical behavior and in the SR Ca²⁺-ATPase activity, determined by fluorimetry and by measurements of the Ca²⁺-uptake and Ca²⁺-release rates, respectively. The SR Ca²⁺-ATPase activity was related to a decreased apparent affinity for Ca²⁺ (pCa-histogram) as well as an increase in the catalytic rates. In addition, the Ca²⁺-induced Ca²⁺-release was significantly increased by ischemia. The effect of Ca²⁺ on Ca²⁺-release was proportional to [Ca²⁺]⁰ but was greater in ischemia than in controls, suggesting that the enhancement of Ca²⁺-induced Ca²⁺-release is related to an increase in the sensitivity of the SR Ca²⁺-release channel to Ca²⁺. The CM-dependent stimulation of Ca²⁺-release was unaffected by ischemia. These results suggest that Ca²⁺-induced Ca²⁺-release is an important determinant of cytosolic Ca²⁺ during ischemia. The alterations of the SR Ca²⁺-ATPase activity during

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