CURRICULUM VITAE

David Alfred Eisner

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David Eisner

Born: 3 January, 1955, Manchester, England

1973-1976	King's College Cambridge
1976	B.A. (class II.1)
1976-1979	Balliol College Oxford
1979	D.Phil. (Supervisor Prof D. Noble)
1979-1980	Postdoctoral Research Assistant- Physiological Laboratory, Cambridge (with Prof I.M. Glynn).
1981-1986	University Lecturer, University College London.
1982-1988	Visiting Assistant Professor, Department of Physiology, University of Maryland
1986-1990	Wellcome Senior Lecturer, University College London
1988-	Research Associate Professor, Department of Physiology, University of Maryland
1989-1990	Reader in Physiology, University of London
1990-1999	Professor of Veterinary Biology, University of Liverpool
1992-1999	Head of Veterinary Preclinical Sciences Department
1999 -	Professor of Cardiac Physiology, Department of Medicine, The University of Manchester
2000-	British Heart Foundation Professor of Cardiac Physiology, The University of Manchester

Honours

1975	Powell Prize and Senior Scholarship: King's College Cambridge
1985	Awarded Pfizer Prize for research in biology
1986	Wellcome Senior Lectureship
1988	Awarded Wellcome Prize (Physiological Society)
1992	Q.R. Murphy Lecturer, University of Wisconsin.
1999	Fellow Academy of Medical Sciences
2000	Founder Fellow International Society for Heart Research
2001	Fellow American Heart Association
2007	Member Academia Europa
2008	Reimer Lecturer International Society for Heart Research
2008	Hon Dorothy Wedgwood Lecture for Young People

Editorial Boards *(items in italics are current)*

Journal of Physiology (1992-7)
Distributing Editor Journal of Physiology (1993-5)
Secretary to the Editorial Board Journal of Physiology (1995-7)
Cardiovascular Research (1996-1999)
Chairman Editorial Board of Journal of Physiology (1997-2000)
Journal of Molecular & Cellular Cardiology (1999-)
Associate Editor Journal of Molecular & Cellular Cardiology (2002-)
Circulation Research (1999-)
Senior Consulting Editor Circulations Research (2009-)
Cell Calcium (2000-)
Biophysical Journal (2005-)

Editor in Chief Journal of Molecular and Cellular Cardiology (2007-)
Basic Research in Cardiology (2009-)

Professional Appointments (since 1990)

Physiological Society Committee (1989-1993 & 1995-1998)
Programme Committee 1993 International Union of Physiological Sciences meeting
Scientific Advisory Committee, Animal Health Trust (1993 -6)
Wellcome Trust: Member Veterinary Interest Group (1990-5)
Wellcome Trust: Member of Physiology and Pharmacology panel (1990-5)
British Heart Foundation: Member of Project Grants Committee (1995-8)
Medical Research Council Grants Committee (1995-1998)
Member of Jury for Medinfar Prize (European Physiology prize 1996-)
Member Research Assessment Exercise Panel RAE 2001 Veterinary Science 1999-2001
Member Cardiovascular Sub Panel for RAE 2001
Council Member International Society for Heart Research (European Section) (2001-2007)
Committee Member British Society for Cardiovascular Research (2002-)
International Secretary, The Physiological Society (2003-7)
Member of International Council of ISHR (2004-)
Chair British Society for Cardiovascular Research (2006-8)
Member Cardiovascular Sub Panel for RAE 2008
Member of Council British Cardiovascular Society (2006-8)
British Heart Foundation Fellowships Committee (2006-)
Chair International Scientific Programme Committee for IUPS 2013
Vice-Chair European Working Group of Cellular Cardiac Electrophysiology (2006-2008)
co-Chair Gordon Research Conference on Cardiac Regulatory Mechanisms (2008)
Member of Evaluation Group Portuguese Foundation for Science & Technology
Chair European Working Group of Cellular Cardiac Electrophysiology (2008-)
Member Sectional Committee Academy of Medical Sciences
President Elect Federation of European Physiological Societies (2009-)

Bibliometric Analysis

“h” index = 52

21 papers cited >=100 times

52 papers cited >=50 times

Research Synopsis

Overview: The majority of my research has involved studying the control of contraction of cardiac muscle and has been undertaken at the universities of Oxford, Cambridge, London and Liverpool & Manchester. Of particular interest have been the processes which control intracellular calcium concentration. The overall approach is to take an integrated view at a cellular level of how the various transporters and channels work together to produce stable control of Ca and how this is upset in disease. I currently head a research group of around 15 people. The work has been funded from a variety of sources including Research Councils and the Wellcome Trust. Major core support presently comes from the British Heart Foundation in the form of a Personal Chair.

Studies on sodium and sodium-calcium exchange. My initial work involved a study of the Na-K pump and its effects on contraction. We demonstrated that the increase of contraction and arrhythmias produced by decreasing extracellular potassium concentration were due to inhibition of the Na-K pump. In subsequent work we obtained the first direct measurements of the electrogenic Na-K pump current in cardiac muscle and used this to further study the mechanism of the relationship between the activity of the Na-K pump and contraction. This was followed by direct measurement of intracellular sodium concentration $[Na^+]_i$ using ion-selective microelectrodes. We showed that $[Na^+]_i$ was decreased by depolarization an effect which (see below) was later shown to result from the effects of membrane potential on the Na-Ca exchange. The direct measurements of $[Na^+]_i$ also gave the first information concerning the relationship between $[Na^+]_i$ and contraction. Specifically, we found that contraction was a steep power function of $[Na^+]_i$. This was true for both phasic and tonic contractions. This, and the dependence of tension on membrane potential were interpreted in terms of cellular calcium content being controlled by a surface membrane Na-Ca exchange.
As well as the above studies on the Na-K pump in cardiac muscle, I have also studied the basic properties of the pump. This included a kinetic analysis of the interactions between ATP, Pi and potassium. In subsequent work we examined the effects of membrane potential on both the electrogenic current and the sodium fluxes produced by the pump.

Calcium oscillations and waves in the heart. My next series of experiments and publications involved studies of the control of $[Ca^{2+}]_i$ using Ca-sensitive indicators. We investigated the factors that controlled resting $[Ca^{2+}]_i$. Depolarization was shown to produce a transient increase of resting $[Ca^{2+}]_i$ which decayed over a few minutes. The transient nature of this response was analyzed in terms of the effects expected from a Na-Ca exchange. At this time we also obtained the first measurements of Ca oscillations due to spontaneous sarcoplasmic reticulum (SR) Ca release. This spontaneous Ca release is responsible for initiating some ventricular arrhythmias associated with Ca overload.

Ischaemia and cardiac metabolites As well as studying normal aspects of cardiac function, I have also been interested in the effects of ischaemia and metabolic inhibition. Using nuclear magnetic resonance (nmr) we showed that the depressant effect of hypoxia on contraction was due to changes of intracellular phosphate rather than pH and also studied the relationship between metabolism, contraction and pH during ischaemia.

Control of intracellular Calcium. Since the mid 1980's my work has mainly used single cells as an experimental model. This was initially used to continue our studies of metabolic inhibition. Our early work with this technique produced the first use of "caged" calcium in intact cardiac

cells and demonstrated the importance of calcium-induced calcium release in cardiac muscle. In a series of subsequent papers we have used caffeine as a tool to investigate excitation-contraction coupling, in part making use of our observation that the Ca indicator Indo-1 could be used to measure caffeine as well as calcium concentration. We showed that the effects of low concentrations of caffeine could be attributed to a sensitization of calcium-induced Ca release. This and recent experiments using tetracaine to depress calcium-induced Ca release showed that manipulating calcium-induced Ca release only produces a *transient* effect on contraction. This shows that calcium-induced Ca release is not a useful locus for contractile regulation and suggests that, in order to produce a useful increase of contraction, the SR Ca content must be increased. These arguments have implications for many studies in which it has been suggested that calcium-inducd Ca release may be modulated.

What controls the Calcium store? Recent work has been aimed at investigating the factors which control the magnitude of contraction produced by the heart and, in particular, the question of the role of the sarcoplasmic reticulum. We developed a method to obtain a quantitative measurement of the SR Ca content and showed that this could be used to measure reproducibly even the small changes of SR Ca content which accompany changes of stimulation rate. Furthermore, we were able to show that the measured changes of SR Ca content could be *quantitatively* accounted for by the measured Ca influx into the cell (on the L-type Ca current) and efflux (on the Na-Ca exchange).We have also applied these techniques to studying SR Ca balance during spontaneous Ca release. We have shown that, as a cell is progressively overloaded with Ca, the SR Ca content increases until a maximum level is reached at which spontaneous Ca release occurs. Further increase of Ca entry simply increases the frequency at which spontaneous Ca release occurs. We have also been interested in the factors which determine whether or not an increase of $[Ca^{2+}]_i$ propagates. We showed, surprisingly at the time, that a locally-evoked increase of $[Ca^{2+}]_i$ does not propagate unless the preparation is Ca-overloaded. Subsequent work showed that propagation depended on an increase of the amount of Ca released.

Stability of control of SR content. The normal control of cardiac contraction requires that the SR Ca content be regulated. We have a major interest in aspects of this regulation. We discovered that a major mechanism is a process that we have termed “autoregulation” in which changes of SR Ca content modify the amplitude of the systolic Ca transient and indirectly modify the influx or Ca into the cell (on the L-type Ca current) and efflux (on Na-Ca exchange). Much of our current research is focused on the idea that this control system can become unstable and that such instabilities may contribute to conditions such as *pulsus alternans* where the amplitude of the cardiac contraction (and the underlying heart beat) alternate from beat to beat.

The origin of calcium-dependent arrhythmias including CPVT. In recent work we have investigated why mutations in the Ryanodine Receptor (RyR) result in Ca waves and arrhythmias such as CPVT (catecholaminergic polymorphic ventricular tachycardia). We have found that simply modifying the properties of the RyR does not by itself produce arrhythmogenic Ca waves. Waves only occur when SR Ca is elevated thus explaining why CPVT patients only have arrhythmias during beta adrenergic stimulation when SR Ca content is elevated.

Treatment of calcium-dependent cardiac arrhythmias. As mentioned above, spontaneous release of Ca from the SR contributes to the origin of cardiac arrhythmias. A major therapeutic challenge is therefore posed by the need to remove this unwanted Ca release while preserving the normal systolic release. This is made all the more important by the fact that such cardiac

arrhythmias are particularly prevalent in the context of heart failure where normal systolic Ca release is already depressed. As proof of principle we have recently demonstrated that the local anaesthetic tetracaine can abolish arrhythmogenic Ca release while *increasing* systolic release and we are investigating the mechanisms behind this.

Smooth muscle and other tissues In addition to the work described above, I have also studied the physiology of smooth muscle in particular the relationship between intracellular pH and Ca ions. These findings have shown the importance of the interactions between these ions on the process of contraction in smooth muscles, such as those in the uterus and vascular beds. Other cell types I have experience with include the carotid body, dorsal root ganglia, and squid axon.

Research grants (1990-)

1991 £201,735 from the Wellcome Trust: "The control of contraction in cardiac and smooth muscle."

1991 £78,451 from the British Heart Foundation: "The effects of metabolism on intracellular pH in mammalian cardiac muscle".

1991 £350,000 from the Wellcome Trust for building alterations (with Burgoyne, Dockray & Petersen).

1992 £24,970 from the Medical Research Council: "The role of membrane excitability in force depression during uterine hypoxia" (with Wray)

1992 £5,451 from the Wellcome Trust: "The effects of caffeine on agonist-evoked changes IP_3 and $[Ca^{2+}]_i$ in pancreatic acinar cells" (with Petersen).

1992 £54,632 from the British Heart Foundation: "Regulation of the sodium pump in cardiac muscle by ATP and its metabolites".

1992 £39,000 from the British Heart Foundation: "Propagation of cardiac calcium transients" (with O'Neill).

1993 £219,036 from the Wellcome Trust: "Spatiotemporal imaging of cytosolic Ca^{2+} by means of confocal laser microscopy" (with Gallacher et al)

1993 £141,201 from The Wellcome Trust: "A study of the interaction between membrane potential, pH, calcium and contraction in arterial smooth muscle" (with Wray).

1993 £341,150 from The Wellcome Trust for a new four year Ph.D. training programme in cellular and molecular physiology. This grant (awarded in conjunction with Professors Burgoyne, Dockray and Petersen) is the first tranche of a grant totalling £2,000,000.

1994 50,000 ecu from the European Community: "Molecular physiology of nerve, cardiac and vascular cells".

1994 13,131 ecu from the European Community: "Regulation of intracellular calcium in muscle"

1995 £353,840 from The Wellcome Trust for a new four year Ph.D. training programme in cellular and molecular physiology.

1995 £87,258 from The British Heart Foundation : "Regulation of diastolic intracellular calcium in the heart".

1995 £430,398 from The Wellcome Trust: "The control of cardiac contraction: measurement of sarcoplasmic reticulum Ca content and sarcolemmal fluxes" (with O'Neill).

1996 £359,180 from The Wellcome Trust for a new four year Ph.D. training programme in cellular and molecular physiology.

1996 £180,911 from the British Heart Foundation: "The control of systolic $[Ca^{2+}]_i$ in cardiac hypertrophy and failure" (with Trafford & Dimaline).

1996 £113124 from the Medical Research Council: "Control of uterine $[Ca^{2+}]_i$ by membrane potential: modulation by pH and metabolic inhibition" (with Wray).

1997 £129,859 from the British Heart Foundation: "Control of portal vascular smooth muscle: effects of intracellular pH" (with Wray).

1997 £129,208 from the British Heart Foundation: "Does regulation of the sarcoplasmic reticulum Ca release process produce a maintained effect on cardiac contraction? A study at physiological heart rate and temperature.

1997 £380,125 from the Wellcome Trust for a new four year Ph.D. training programme in cellular and molecular physiology.

1998 £149,072 from the British Heart Foundation. "The interdependence of s.r. Ca content, Ca sparks and cellular Ca balance: a study under normal and metabolically inhibited conditions" (with O'Neill).

1998 £144,782 from the British Heart Foundation. "Near membrane calcium gradients and the regulation of the Ca-activated chloride channel in cardiac muscle: a means to measure subsarcolemmal Ca" (with Trafford).

1998: £183,373 from the Wellcome Trust. "Mechanisms underlying the restoration of $[Ca^{2+}]$ following stimulation in uterine smooth muscle cells" (with Wray).

2000: £500,000 from the British Heart Foundation for a Personal Chair

2000: £112,758 from the British Heart Foundation. "Intracellular calcium and arrhythmogenesis", Ph.D. studentship – sum includes extension for a third year

2001: £128,410 from the British Heart Foundation. "What causes the heterogeneity of the systolic Ca release in isolated cardiac ventricular myocytes: the effects of inotropic manoeuvres" – Intermediate Fellowship for Dr M.E. Díaz.

2002: £390,000 from the British Heart Foundation. "Confocal studies of intracellular calcium concentration"

2002 £125,662 from the British Heart Foundation. "Role of SR Ca content in the inotropic effects of catecholamines"

2003: £135,010 from the British Heart Foundation. "What produces stability and alternans of the systolic calcium transient?"

2004: £126,474 from the British Heart Foundation. "Does increasing the open probability of the ryanodine receptor produce arrhythmias?"

2005: £82,473 from the British Heart Foundation. "Integrative analysis of Ca^{2+} cycling in cardiac myocytes in response to TNFα: the role of SERCA".

2006: £500,000 from the British Heart Foundation for renewal of Chair.

2006: £586,024 from the British Heart Foundation (Programme Grant). "The role of dyssynchronized Ca release in calcium alternans and its relation to electrical alternans"

2006: £175,593 from the British Heart Foundation. "Identifying How Cellular Calcium Buffers Modulate the Systolic Calcium Transient and Response to Beta-Adrenergic Stimulation in Isolated Cardio Myocytes"

2007: £53,891 from the British Heart Foundation. "Mechanisms underlying altered calcium homeostasis in the atria in heart failure"

2008: £190,327 from the British Heart Foundation. "An integrative approach to define the cellular mechanisms underlying the slow changes of QT interval following changes of heart rate"

Invited seminars and lectures (1990-)

1990: Cardiac Registrars Group.
University of Illinois.
American Heart Association, Dallas, Texas.
Royal Microscopical Society.
International Society for Heart Research.
American Heart Association.

1991: University College, London.
2nd International Conference on Sodium-Calcium Exchange, Baltimore, Maryland.
Physiological Society, Dundee.
Physiological Society, Oxford.
IUPS, Prague, Czechoslovakia.
Duke University, North Carolina.
Manchester University.
Cambridge University.

1992: University of Illinois, Chicago.
Loyola University, Chicago.
Cologne University, Germany.
St. Mary's Hospital Medical School, London.
Oxford University.
Edinburgh University, Scotland.
University of Wisconsin, U.S.A.
Physiological Society Symposium and Workshop.

1993: University of Edinburgh.
University of Bristol.
King's College London.
Association of Veterinary Teachers and Research Workers.
Bogomoletz Institute, Kiev, Ukraine.
University of Aberystwyth.
University of Tubingen, Germany.
International Society for Heart Research.
St. George's Hospital, London.

1994: International Meeting, Osaka, Japan
British Cardiac Society
Physiological Society Symposium
Invited lecture, Royal Society of Anaesthetists

1995: University of Berne, Switzerland
University of Pohang (Korea)
University of Massachusetts (USA)
University of Harare (Zimbabwe)
University of Edinburgh
British Cardiac Society
International Society for Heart Research (Prague)

1996: International Society for Heart Research, Chicago (USA)
University of Halle (Germany)
Joint Organizer of 3rd International Mammalian Myocardium Meeting
Invited Lecture Hungarian Physiological Society (Szeged)
University of Chicago
International Meeting on Cardiac Physiology (Banff, Canada)
University of Manchester

1997: Invited Speaker: International Physiology Meeting St Petersburg
University of Loyola, Chicago
University of Glasgow
Seminar: Royal Free Hospital

1998: Invited speaker International Society for Heart Research Rhodes
Invited speaker Gordon Research Conference
University of Bristol
University of Connecticut
British Cardiac Society Symposium

1999 University of Newcastle
University of Augusta, Georgia USA
University of Indiannapolis, USA
University of Calgary, Canada
Babraham Institute
Kings College London
Physiological Society Symposium, Glasgow
Symposium on Ion Channels, France
Symposium on Atrial Fibrillation, Nice, France
Hatter Institute University College London
Symposium on membrane transport, Columbia, Missouri, USA

2000 University of Leuven, Belgium
Loyola University Chicago, USA
Finch University Chicago, USA
University of Illinois, USA
Astrazeneca. Gothenborg, Sweden
Society for Hypertension, Helsinki, Finland
Gordon Conference, New Hampshire USA

2002 University of Halifax, Nova Scotia
International Society for Heart Research, Winnipeg, Canada
Queenstown, New Zealand
University of Lund, Sweden
Novartis Symposium on Smooth Muscle (*Chairman of meeting*)

2002: Gordon Research Conference, New Hampshire USA
International Society for Heart Research (Hungary)
Trinidad (Calcium symposium)
Landmark Lecture International Society for Heart Research, Wisconsin, USA
International Society for Heart Research, Yamagata, Japan
National Institute of Health, North Carolina USA
University of Oxford

Distinguished Lecturer of the Molecular Cardiology Institute, University of Maryland
University of Leicester.

2003: Plenary Lecture, joint meetings of the UK & Spanish Physiological Societies, Tenerife, Spain
University of Goettingen
International Workshop, Santiago, Chile
International Society for Heart Research, Strasbourg
Imperial College London

2004: Plenary Lecture, Norwegian Cardiological Society
University of Oxford
Cardiac Symposium, Physiological Society Glasgow
Rush University Chicago
University of Utah
North American Society of Pacing and Electrophysiology (NASPE)
British Cardiac Society
International Society for Heart Research (Dresden)
Gordon Conference USA
International Society for Heart Research (Brisbane)
Medtronic Inc USA

2005: Department of Physiology, UCLA
Chilean Physiological Society, Santiago, Chile
European Society of Cardiology, Stockholm
European Working Group in Cellular Electrophysiology, Antwerp
Black Symposium, British Pharmacological Society
International Society for Heart Research, Osaka, Japan

2006: University of Cleveland
University of Milan-Bicocca
German Cardiac Society
British Cardiac Society
University of Szeged
University of Nantes
Latin American Physiological Society

2007: University College London
University of Leicester
Academic Medical Centre, Amsterdam
United Arab Emirates University
Heart Rhythm Society, USA
International Society for Heart Research, Bologna Italy
Dresden University
American Heart Association

2008: French Cardiac Society
European Heart Failure Association (Basic Cardiovascular Science Council Lecture)
University of Newcastle
Meeting on Multiscale Modelling of the Heart – Auckland New Zealand
University of Oulu, Finland
University of Homburg Saar, Germany
Hospital Val d'Hebron, Barcelona, Spain
Yamagata University, Japan

International Society for Heart Research, Athens, Greece (Reimer Lecture)
British Cardiac Society
International Society for Heart Research, Cincinnati, USA
European Society of Cardiology, Munich, Germany
Chilean Physiological Society
University Medical Center Hamburg-Eppendorf
Netherlands Physiological Society

2009: Gordon Research Conference on Cardiac Arrhythmias, Italy
Japanese Circulation Society
Oulu, Finland
Heart Rhythm, Boston, USA
International Society for Heart Research, Baltimore, USA
International Union of Physiology Sciences (Kyoto)
Australian and New Zealand Cardiac Society (Sydney)
Society of General Physiologists, Woods Hole, USA

Publications (abstracts not included)

1. Cohen, I., Eisner, D. & Noble, D. (1978). The action of adrenaline on pace-maker activity in cardiac Purkinje fibres. *Journal of Physiology* 280, 155-168.

2. Attwell, D. & Eisner, D. (1978). Discrete membrane surface charge distributions, effects of fluctuations near individual channels. *Biophysical Journal* 24, 869-875.

3. Attwell, D., Cohen, I., Eisner, D., Ohba, M. & Ojeda, C. (1979). The steady-state TTX-sensitive ("window") sodium current in cardiac Purkinje fibres. *European Journal of Physiology* 379, 137-142.

4. Eisner, D.A. & Lederer, W.J. (1979). Inotropic and arrhythmogenic effect of potassium depleted solutions on mammalian cardiac muscle. *Journal of Physiology* 294, 255-277.

5. Eisner, D.A. & Lederer, W.J. (1979). The role of the sodium pump in the effects of potassium depleted solutions on mammalian cardiac muscle. *Journal of Physiology* 294, 279-3O1.

6. Attwell, D., Cohen, I. & Eisner, D. (1979). Membrane potential and ion concentration stability conditions for a cell with a restricted extracellular space. *Proceedings of the Royal Society (B)* 206, 145-161.

7. Attwell, D., Eisner, D. & Cohen, I. (1979). Voltage clamp and tracer flux data: effects of a restricted extracellular space. *Quarterly reviews of Biophysics* 12, 213-261.

8. Lederer, W.J., Spindler, A.J. & Eisner, D.A. (1979). Thick slurry bevelling. A new technique for bevelling extremely fine microelectrodes and micropipettes. *Pflugers Archiv* 381, 287-288.

9. Eisner, D.A. & Lederer, W.J. (1980). Characterization of the electrogenic sodium pump in cardiac Purkinje fibres. *Journal of Physiology* 303, 441-474.

10. Eisner, D.A. & Lederer, W.J. (1980). The relationship between sodium pump activity and twitch tension in cardiac Purkinje fibres. *Journal of Physiology* 303, 475-494.

11. Attwell, D., Cohen, I. & Eisner, D.A. (1981). The effects of heart rate on the action potential of guinea-pig and human ventricular muscle. *Journal of Physiology* 313, 439-461.

12. Eisner, D.A., Lederer, W.J. & Vaughan-Jones, R.D. (1981), The dependence of sodium pumping and tension on intracellular sodium activity in voltage-clamped sheep cardiac Purkinje fibres. *Journal of Physiology* 317, 163-187.

13. Eisner, D.A., Lederer, W.J. & Vaughan-Jones, R.D. (1981). The effects of rubidium ions and membrane potential on the intracellular sodium activity of sheep Purkinje fibres. *Journal of Physiology* 317, 189-205.

14. Eisner, D.A. & Richards, D.E. (1981). The interaction of potassium ions and ATP with the sodium pump of resealed red cell ghosts. *Journal of Physiology* 319, 403-418.

15. Lederer, W.J. & Eisner, D.A. (1982). The effects of sodium pump activity on the slow inward current in sheep cardiac Purkinje fibres. *Proceedings of the Royal Society of London (Series B).* 214, 249-262.

16. Eisner, D.A. & Richards, D.E. (1982). Inhibition of the sodium pump by inorganic phosphate in resealed red cell ghosts. *Journal of Physiology* 326, 1-10.

17. Arnold, L., Page, J., Attwell, D., Cannell, M.B. & Eisner, D.A. (1982). The dependence on heart rate of the human ventricular action potential duration. *Cardiovascular Research* 16, 547-551.

18. Eisner, D.A. & Richards, D.E. (1983). Stimulation and inhibition by ATP and orthophosphate of the potassium:potassium exchange in resealed red cell ghosts. *Journal of Physiology* 335, 495-506.

19. Vaughan-Jones, R.D., Lederer, W.J. & Eisner, D.A. (1983). Calcium ions can influence intracellular pH in mammalian cardiac muscle. *Nature* 301, 522-524.

20. Eisner, D.A., Lederer, W.J. & Vaughan-Jones, R.D. (1983). The control of tonic tension by membrane potential and intracellular Na activity in the sheep cardiac Purkinje fibre. *Journal of Physiology* 335, 723-743.

21. Eisner, D.A., Lederer, W.J. & Sheu, S.-S. (1983). The role of intracellular sodium activity in the antiarrhythmic action of local anaesthetics in sheep cardiac Purkinje fibres. *Journal of Physiology* 340 239-257.

22. Allen, D.G., Eisner, D.A., Lab, M.J. & Orchard, C.H. (1983). The effects of low Na solutions on intracellular Ca concentration and tension in ferret ventricular muscle. *Journal of Physiology* 345, 391-407.

23. Orchard, C.H., Eisner, D.A. & Allen, D.G. (1983). Oscillations of intracellular [Ca^{2+}] in mammalian cardiac muscle. *Nature* 304, 735-738.

24. Eisner, D.A., Vaughan-Jones, R.D. & Lederer, W.J. (1983). Comments on "active transport and inotropic state in guinea pig left atrium" which appeared in Circ. Res. 52: 411-422, 1983 *Circulation Research* 53, 834-835.

25. Eisner, D.A., Orchard, C.H. & Allen, D.G. (1984). Control of intracellular ionized calcium concentration by sarcolemmal and intracellular mechanisms. *Journal of molecular and cellular Cardiology* 16, 137-146.

26. Eisner, D.A. & Vaughan-Jones, R.D. (1983). Do calcium-activated potassium channels exist in the heart? *Cell Calcium* 4, 371-386.

27. Allen, D.G., Eisner, D.A. & Orchard, C.H. (1984). Factors influencing free intracellular calcium concentration in quiescent ferret ventricular muscle. *Journal of Physiology* 35O, 615-630.

28. Allen, D.G., Eisner, D.A. & Orchard, C.H. (1984). Characterization of oscillations of intracellular calcium concentration in ferret ventricular muscle. *Journal of Physiology* 352, 113-128.

29. Eisner, D.A., Lederer, W.J. & Vaughan-Jones, R.D. (1984). The quantitative relationship between intracellular Na activity and tension in sheep cardiac Purkinje fibres. *Journal of Physiology* 355, 251-266.

30. Requena, J., Whittembury, J., Tiffert, T., Eisner, D.A. & Mullins, L.J. (1984). A comparison of measurements of intracellular Ca by Ca electrode and optical indicators. *Biochimica et Biophysica Acta* 805, 393-404.

31. Eisner, D.A. & Lederer, W.J. (1985). Na-Ca exchange: stoichiometry and electrogenicity. *American Journal of Physiology* 248, C189-C902.

32. Valdeolmillos, M. & Eisner, D.A. (1985). The effects of ryanodine on calcium-overloaded sheep cardiac Purkinje fibers. *Circulation Research* 56,452-456.

33. Allen, D.G., Eisner, D.A., Pirolo, J.S. & Smith, G.L. (1985). The relationship between intracellular calcium and contraction in calcium overloaded ferret papillary muscles. *Journal of Physiology* 364, 169-182.

34. Requena, J., Whittembury, J., Tiffert, T., Eisner, D.A. & Mullins, L.J. (1985). The influence of chemical agents on the level of ionized [Ca^{++}] in squid axons. *Journal of general Physiology* 85, 789-804.

35. Eisner, D.A. & Valdeolmillos, M. (1985). The mechanism of the increase of tonic tension produced by caffeine in sheep cardiac Purkinje fibres. *Journal of Physiology* 364, 313-326.

36. Nieman, C.J. & Eisner, D.A. (1985). Effects of caffeine, tetracaine, and ryanodine on calcium-dependent oscillations in sheep cardiac Purkinje fibres. *Journal of general Physiology* 86, 877-889.

37. Eisner, D.A. & Valdeolmillos, M. (1986). A study of intracellular calcium oscillations in sheep cardiac Purkinje fibres measured at the single cell level. *Journal of Physiology* 372, 539-556.

38. Eisner, D.A. & Valdeolmillos, M. (1986). Measurement of intracellular calcium during the development and spontaneous relaxation of tonic tension in sheep cardiac Purkinje fibres. *Journal of Physiology* 375, 269-281.

39. Allen, D.G., Eisner, D.A., Morris, P.G., Pirolo, J.S. & Smith, G.L. (1986). Metabolic consequences of increasing intracellular calcium and force production in perfused ferret hearts. *Journal of Physiology* 376, 121-141.

40. Cannell, M.B., Eisner, D.A., Lederer, W.J. & Valdeolmillos, M. (1986). Effects of membrane potential on intracellular calcium concentration in sheep Purkinje fibres in sodium-free solutions. *Journal of Physiology* 381, 193-203.

41. Eisner, D.A., Valdeolmillos, M. & Wray, S.C. (1987). The effects of membrane potential on active and passive sodium transport in xenopus oocytes. *Journal of Physiology* 385, 643-659.

42. Vaughan-Jones, R.D., Eisner, D.A. & Lederer, W.J. (1987). Effects of changes of intracellular pH on contraction in sheep cardiac Purkinje fibers. *Journal of general Physiology* 89, 1015-1032.

43. Eisner, D.A., Elliott, A.C. & Smith, G.L. (1987). The contribution of intracellular acidosis to the decline of developed pressure in ferret hearts exposed to cyanide. *Journal of Physiology* 391, 99-108.

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